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25 March 1996

Mr. Eric Newman
USEPA
General Remedial Section (3HW23)
841 Chestnut Street
Philadelphia, PA 19107

Re: Halby Chemical Superfund Site
Risk Evaluation

Dear Mr. Newman:

By this letter and the enclosed reports, Witco Corporation is providing a response to EPA's letter of 5 February, 1996, and a response to Item 8.3g of the 20 July 1995 USEPA CERCLA §106 Order (Order) issued to Witco for the referenced site. This item requires Witco to "Develop and submit for approval, soil clean-up level(s) sufficient to protect human health and the environment."

There are several components to this response as follows:

- This letter responds to each of EPA's comments in it's 5 February, 1996 letter which reviewed the "Review of Constituent of Concern Toxicity" (Primary Constituent of Concern (COC) Risk Assessment (RA);
- Enclosure 1 - "Review of Arsenic Carcinogenicity by Oral Exposure" (Arsenic RA) provides a more complete rationale for the arsenic evaluation;
- Enclosure 2 - "Review of Ground Water Migration Exposure" (Soil/Ground Water RA) discusses the appropriate screening levels for soil to ground water cross-media transfer;
- Enclosure 3 - "Development of Human Health Screening Levels for Additional Constituents" (Additional COC RA) screens for remaining site constituents not considered in the Primary COC RA.

RESPONSES TO COMMENTS IN NEWMAN TO VYAS LETTER

In the text of the letter, there were several items which required some further discussion. These subjects are discussed below.

Selection of Target Hazard Quotient

EPA's discussion on target hazard indices is unclear. The cleanup level calculation tables state a target hazard quotient (HQ) of 0.33 with a note that "Hazard Quotient for each contaminant is apportioned amongst all of the noncarcinogens", but also contains a note that "each contaminant affects a different target organ and the HQ was set at 1." Nonetheless, the Draft Soil Screening Guidance (USEPA 1994) allows that soil screening levels can be set at an HQ of 1, if they have different endpoints, but should be divided for common endpoints. The Primary COC RA and the Additional COC RA provides screening of all site contaminants of concern and the endpoints are provided in the table below.

	Target Organ	Critical Effect
carbon disulfide	peripheral nervous system	neurotoxicity
manganese	nervous system	neurologic disturbances
thiocyanate	thyroid	thyroid toxicity
bis(2-ethylhexyl) phthalate	liver	increased weight
aluminum	(a)	(a)
antimony	blood	altered chemistry
beryllium	(a)	(a)
mercury	nervous system	neurotoxicity
vanadium	(a)	(a)

(a) - Reference dose based on No Observable Adverse Effects Level (NOAEL), no adverse effects reported from selected study.

As shown, only three constituents have similar endpoints - carbon disulfide, mercury and manganese, which all affect the nervous system. Therefore, the HQ for these toxicants should be set at 0.33. All remaining toxicants can be screened at an HQ of 1.

Action Levels

We also recognize the inefficiency of remediating multiple times to different action levels. However, it also important not to lose sight of the intent for the EPA removal order which is to address what EPA believes is an imminent and substantial endangerment. If action levels were calculated to establish what represents an imminent and substantial endangerment, those levels would undoubtedly be higher than what has been presented so far, since they would have significantly shorten exposure duration, etc. Nevertheless, the risk levels we have been discussing to date for site workers have incorporated long-term exposure at the site and to the extent possible, we would attempt to remediate to these final levels.

It is also important to note that for carcinogens, EPA guidance clearly allows an acceptable risk range from 10^{-4} to 10^{-6}. It is not appropriate to automatically assume that this site should be remediated to a 10^{-6} risk level. In assessing the carcinogenic risk, we have attempted to be reasonable by assuming a mid-point risk of 10^{-5}. At this risk level for an individual consituent, it would be possible to have up to 10 carcinogens and still not exceed the upper bounds (10^{-4}) of acceptable risk.

In particular, in the area of the water supply line, the completion of all sampling indicates that none of the site constituents exceed the action levels in your letter for a site worker (i.e. a worker exposed on a regular basis to surficial soils). EPA has not calculated a construction worker scenario to predict subsurface exposures, but our calculations on this issue also show no action level exceedance for a construction worker scenario along the water supply line.

Trip Report

In your letter, you provided a copy of analysis collected from the marsh area which indicated some levels of herbicides. Witco has reviewed this matter and strongly believes Witco is not responsible for these constituents. Witco is presently unaware of any Witco use, manufacture, unintended manufacture, storage, transportation, or disposal, of these materials at the site. These materials are produced in dramatically different ways than the products Halby produced during Witco's ownership.

Witco has information that the railroad routinely sprayed herbicide along the right-of-way to suppress vegetation, and that U.S. Borax, the parent company of Pyrites, operated a herbicide packaging operation on the Pyrites property. These two facts may explain the presence of herbicides in the area.

RESPONSE TO SPECIFIC COMMENTS IN MEMO FROM JAFOLLA TO NEWMAN

The comments in the referenced memo are duplicated herein in bold followed by our responses in regular type.

1. **It is not clear whether the document (page 1) is presenting soil cleanup levels or soil screening levels. Soil cleanup levels are generally more site-specific, albeit in some cases similar to the soil screening levels.**

   The intent of the Primary COC RA was to provide a screening of the major COCs from the CH_2MHILL risk assessment, with the addition of carbon disulfide which was not noted previously. The screening levels are intended to determine what constituents are COCs. In order to calculate cleanup levels, the accumulated risk from the respective carcinogenic COCs and non-carcinogenic COCs should be calculated, then cleanup levels back-calculated to provide an acceptable risk. What has become obvious is that only a handful of constituents are COCs, and they tend to have different endpoints. This means that there is little accumulation of risk, and the screening levels default to cleanup levels. This is especially true for this site since based on the CH_2MHILL risk assessment, the constituents we have focused on to-date represent over 90% of the site risk (with the addition of carbon disulfide).

   In fact, analysis of all noncarcinogens detected in soil indicates that carbon disulfide, thiocyanate, and manganese are the only constituents which exceed the screening levels. Consistent with the Draft Soil Screening Guidance (USEPA), the total Hazard Quotient was set to 1.0 and one-third (0.33) was attributed to each of these constituents, since they have the same endpoint. Since these are the only constituents which exceed their screening levels, the screening levels for these constituents should be equivalent to the cleanup levels.

Likewise, the cleanup levels for carcinogens do need to be calculated to account for cumulative carcinogenic effects. However, analysis of all OU-2 samples shows that only arsenic exceeds soil screening levels, except for one sample location where benzo(a)pyrene was observed at 8.1 mg/kg versus a screening level of 7.84 mg/kg. All other samples were considerably lower, and we consider this 8.1 mg/kg data point to be an outlier, and therefore the only carcinogen exceeding screening levels is arsenic. Based on this, the action level defaults to the screening level.

2. **The Report states that soil-to-ground water cleanup levels were not calculated because "no imminent threat appears to exist for ground water." Please note that a remedial action for the ground water has not been determined at this time. In-house soil-to-ground water cleanup levels will be calculated at a later date after a remedial decision is made.**

   To clarify, the Primary COC RA stated "For example, considering that no one is using site ground water, and the ground water contamination is well understood, no imminent threat appears to exist for ground water. Nonetheless, additional information to evaluate the soil to ground water pathway will be collected in the future" This statement was intended to indicate that while no imminent and substantial endangerment existed for ground water, we would provide screening information so that the remedial action would consider all impacts. This analysis is provided in the Soil/Ground Water RA.

3. **The constituents of concern (COCs) which appear to drive the risk at the site according to the document are arsenic, carbon disulfide, manganese, and thiocyanate. Please note that while this may be the case now, deletion or addition of COCs may be necessary after the risk assessment is completed.**

   All detected constituents were screened and that analysis is presented in the Additional COC RA. The results show that no additional constituents exceed cleanup levels on OU-2.

4. **Estimates for arsenic in soil (not water) may be lower downwards as much as an order of magnitude at the discretion of the manager. However, correction for bioavailability and methylation reactions is not acceptable. Correction for bioavailability will be acceptable only if site specific studies are**

conducted, since bioavailability is matrix dependent. Methylation of arsenic is already taken into consideration in the toxicity criteria. Therefore, correction of the intake for methylation is not appropriate.

A complete discussion of recent information and previous EPA evaluations is provided in the Arsenic RA. The clear conclusion from this information is that it is entirely appropriate to consider both arsenic methylation reactions and bioavailability in arsenic carcinogenic evaluations. We believe that methylation has not been taken into account since the studies EPA relied upon included a dose which exceeded the methylation reaction range. Therefore, the screening levels previously developed in the Primary COC RA should be applied.

5. **The reference dose (RfD) for manganese in soil is 0.024 mg/kg/d, not 0.14 mg/kg/d. This RfD applies to manganese in soil and water.**

We rechecked IRIS and found the most current manganese Reference Dose (RfD) of 0.14 mg/kg-day. Please inform us if your information is different. Correcting to this RFD will change the level EPA calculated. As noted later in this document, we have applied a modifying factor of 3 when calculating acceptable soils levels.

6. **The provisional RfD for thiocyanate is 2E-02 mg/kg/d. There is no basis for use of the alternative RfD of 1E-01 mg/kg/d recommended by the Respondents.**

ERM has recently completed a more thorough review of thiocyanate toxicological information and RFD development for the Lemoyne Superfund Site in Alabama. This review shows that an RFD of 1.0-1.3 is fully supported by the scientific data available. We have provided a copy of that review as Enclosure 4.

7. **The appropriate receptors for the Halby site were indicated in the Report to be construction workers and trespassers. The EPA determined that residents live on-site and therefore, the appropriate receptors are residents and workers. It is assumed that construction workers will be adequately protected and that subsurface soil will be cleaned to levels protective of either a resident or a worker depending on future site use.**

   Witco acknowledges that residents are present and we are evaluating the best approach to address their presence. Furthermore, we agree that for long-term use of the site, the site worker scenario is the most appropriate receptor to analyze. However, we believe that a separate construction worker scenario is appropriate to reflect short-term exposure to subsurface soils. Remedial action options should consider this fact and also incorporate appropriate deed restrictions which limit site disturbance in certain circumstances.

8. **The volatilization factor for carbon disulfide is 1010 as calculated in the soil-screening guidance, not 3340 indicated in the Report.**

   EPA is correct in that the volatilization factor for carbon disulfide is 1010.

RESPONSE TO OTHER NOTES ON ATTACHMENT

In the memo from Jafolla to Newman, there were additional comments provided regarding attachments. Responses to those comments are provided below.

3. **While the attached tables do not consider the dermal route, interim calculations (not attached) indicate that the dermal route may drive the cleanup for manganese for the residential exposure scenario. ECAO (The Environmental Criteria Assessment Office) will be contacted to determine the appropriateness of using the oral adjusted toxicity criterion for assessing the dermal route for manganese (e.g., to determine if a similar mechanism of action is expected).**

   ERM has evaluated the dermal exposure route for manganese, setting the hazard quotient equal to 0.33 consistent with EPA's

approach. The results of that analysis for a site worker are as presented below:

The EPA algorithm for calculating human health risk for dermal contact from soils was used (EPA Region III, 1995). The algorithm is as follows:

$$RBC\ (mg/kg) = \frac{THI \times BW \times AT \times 365\ days/year}{EF \times ED \times (1/RfD) \times 1.0E\text{-}06 \times SA \times AF \times ABS}$$

RBC = risk-based concentration (set to equal a hazard index of 1.0)
THI = Hazard Index (0.33)
BW = Body weight (70 kg)
AT = Averaging time (ED x 365 days/years)
EF = Exposure frequency (250 events/yr)
ED = Exposure duration (25 yr)
SA = Skin surface area available for contact (5300 cm2/event)
AF = Soil-to-skin adherence factor (1 mg/cm2)
ABS = Absorption factor (1% for manganese)
RfD = Reference dose (4.70E-02 mg/kg-day for manganese) *

* EPA has recently consolidated the RfD for manganese in soil and water to 0.14 mg/kg-day. The EPA recommends applying a modifying factor of 3 to this RfD when assessing exposures to manganese in nondietary exposures, such as with soil. Therefore, an RfD of 4.70E-02 was used in the above calculation.

The parameters used in the calculation, listed above, represent those typically used in an industrial scenario for exposures to affected soil. Using these assumptions, the performance standard for dermal contact to manganese in soil is 30,700 mg/kg. This level is considerably higher than the level EPA calculated for ingestion and therefore dermal contact will not be the controlling exposure route.

CONCLUSIONS

After reviewing the information EPA has presented, we believe it would be helpful to summarize the screening levels derived from the EPA and ERM risk analysis completed to date. The table on the next page presents a consolidation of the of the EPA Region III developed levels for surficial soil, and the ERM levels for surficial soils, subsurface soils, or migration

to ground water. The most stringent levels presented assume existing conditions. Obviously, if a cap or other containment is placed on the impacted areas, or if ground water use is not a reasonable scenario, the soil to ground water levels could be considerably higher.

If you have any questions on this matter, please call me at 203-552-2476, or Richard Dulcey at 610-524-3610.

Sincerely,

Raj Vyas

Raj Vyas
Witco Corporation
Corporate Manager
Environmental Remediation

cc: Jane Biggs-Sanger (DNREC)
Patricia Miller, Esq (3RC22)
Michael Towle (3HW31)
Robert Root
James A. Nortz, Esq.
William F. Mercurio
Richard J. Dulcey

enclosure

Summary of Screening Levels (mg/kg)
Surface Soil - Site Worker

	EPA Surface Soil	ERM Surface Soil	ERM Soil to Ground Water Impact	Most Stringent Level based on Existing Conditions
aluminum	1,000,000[b]	2,040,000	NA	1,000,000
carbon disulfide	516[a]	3,450	106	106
manganese	24,808[a*]	42,200	5,000,000	24,808
mercury	610[b]	202	290	202
thiocyanate	40,880[a]	204,000	240	240
vanadium	14,000[b]	14,300	NA	14,000
arsenic	4[a]	1,000	1,460	c
benzo(a)pyrene	0.78[b]	7.84	168	7.84[d]
benzo(b)fluoranthene	7.8[b]	78.4	1,012	78.4[d]
benzo(k)fluoranthene	78[b]	784	80,224	784[d]
chrysene	780[b]	7,840	45,168	7840[d]
indeno (1,2,3-cd) pyrene	7.8[b]	78.4	56,856	78.4[d]

Notes:
- a - From EPA 2/5/96 Letter
- b - From Region III Risk Based Concentration list
- c - TBD pending EPA review of Arsenic RA
- d - Based on midpoint 10^{-5} cancer risk
- * RFD needs to be confirmed by EPA

The most stringent levels presented assume existing conditions. Obviously, if a cap or other containment is placed on the impacted areas, or if ground water use is not a reasonable scenario, the soil to ground water levels could be considerably higher.

Summary of Screening Levels (mg/kg)
Subsurface Soil - Construction Worker

	ERM Subsurface Soil	ERM Soil to Ground Water Impact	Most Stringent Level based on Existing Conditions
aluminum	5,320,000	NA	5,320,000
antimony	2,130	NA	2,130
beryllium	867	18,400	867
carbon disulfide	74,000	106	106
manganese	465,000	5,000,000	465,000
mercury	5,270	290	290
thiocyanate	532,000	240	240
vanadium	37,300	NA	37,300
arsenic	70,000	1,460	a
benzo(a)pyrene	511	168	168[b]
benzo(b)fluoranthene	5,100	1,012	1,012[b]
benzo(k)fluoranthene	51,000	80,224	51,000[b]
bis(2-ethylhexyl) phthalate	106,000	12,001	12,001[b]
carbazole	186,000	166	166[b]
chrysene	511,000	45,168	45,168[b]
indeno (1,2,3-cd) pyrene	5,110	56,856	5,110[b]

Notes: a - TBD pending EPA review of Arsenic RA

b - Based on midpoint 10^{-5} cancer risk

The most stringent levels presented assume existing conditions. Obviously, if a cap or other containment is placed on the impacted areas, or if ground water use is not a reasonable scenario, the soil to ground water levels could be considerably higher.

REPORT

Witco Corporation

Halby Chemical Site Review of Arsenic Carcinogenicity By Oral Exposure

25 March 1996

Environmental Resources Management, Inc.
855 Springdale Drive
Exton, Pennsylvania 19341

On November 14, 1995, Witco Corporation provided the report entitled "Review of Constituent of Concern Toxicity" (Primary COC RA) as a response to EPA's response to Item 8.3g of the 20 July 1995 USEPA CERCLA §106 Order (Order) issued to Witco for the Halby Chemical Site, New Castle, Delaware. This item requires Witco to "Develop and submit for approval, soil clean-up level(s) sufficient to protect human health and the environment." EPA's provided comments in it's 5 February, 1996 letter, and stated that certain assumptions which were included in the assessment of arsenic were not appropriate. This document is intended to provide additional scientific information for the appropriateness of the assumptions in the Primary COC RA.

INTRODUCTION AND HISTORICAL

The current USEPA oral cancer slope factor (i.e., 1.5 mg/kg-day) was developed from studies of a Taiwanese population exposed to high naturally-occurring arsenic levels in their drinking water that developed skin cancers (IRIS, 1995). To calculate the oral slope factor, USEPA used the median value of arsenic in drinking water wells and assumed water consumption of 3.5 L/day for males and 2 L/day for females. They did not consider other exposures (i.e., dietary) of arsenic. Interestingly, arsenic is somewhat unique in that there is essentially no evidence of carcinogenicity in experimental animals (Klaassen, 1996). Obviously, there are a number of issues relating to the use of these epidemiological/ecological studies (i.e., the Taiwanese Studies) to develop a cancer slope factor applicable to the American population. This paper discusses many of these issues and their application to oral exposure to arsenic in soil and water.

Like many metals and metalloids, arsenic is difficult to characterize because it's chemistry is so complex. There are many forms of arsenic in the environment, including both inorganic and organic forms, yet the analytical methodology is not readily available to distinguish the forms, thus the regulatory approach is to consider it as a single element. Ambient levels of arsenic in soil range from 1 to 40 ppm (ATSDR), with mean values of about 5 ppm. Drinking water usually contains a few micrograms of arsenic per liter, with most US drinking water levels reported at lower than 5 µg/L. Diet provides a variable source of arsenic exposure averaging about 0.40 mg/day of intake, with a significant increase if seafood is a part of the diet.

Exposure to arsenic results in deposition in specific tissues, including the skin, nails and hair. The *in vivo* metabolism of arsenic is complicated by the form of arsenic ingested, but appears to include conversion to trivalent arsenic followed by methylation. The methylation reaction first forms monomethylarsenic and then dimethylarsenic. Dimethylarsenic is the principal transformation product in humans, and is rapidly excreted in the urine. Dimethylarsenic is much less toxic than the inorganic trivalent form and is considered a detoxication reaction. However, exposure to inorganic arsenic may exceed the rate of its transformation, resulting in toxicity from the inorganic form. Klaassen (1996) states that ..."consideration of toxic dose-response to inorganic arsenic must be assessed in the light of what is known about metabolic transformation".

METHYLATION REACTIONS

USEPA scientists published an article in 1988 stating that arsenic is an example of a threshold carcinogen, where tumors are produced only if the dose exceeded the threshold (Marcus & Rispin, 1988). They reported a series of skin lesions that may occur following oral exposure to arsenic: hyperpigmentation, keratosis, Bowen's disease and squamous cell skin cancer. These lesions do not represent different stages in the evolution of a single lesion, but of a different cellular lineage. Further, the appearance of each of these lesions is dose-related, and the higher the dose the more likely skin cancers will occur. The justification for the threshold classification was primarily based on the ability of the human body to methylate trivalent arsenic. They also stated that pentavalent arsenic is readily converted to the trivalent form in the blood. Further Valentine et al., (1979) measured arsenic levels in blood, urine and hair in five US communities with high arsenic level in drinking water (ranging form 6 to 393 µg/L). The results showed that arsenic levels in urine and hair were in proportion to the concentration in drinking water, but that levels in blood only increased if the water exceeded 100 µg/L. Marcus and Rispin summarized the methylation issue with the following statements:

- The major site of methylation is the liver.
- Trivalent arsenic (As III) is the substrate for methylation, and pentavalent arsenic (As V) must be reduced to As III before methylation can occur.
- Dimethylarsenic acid (DMA) is the major metabolite found in animals and man and it appears mainly in the urine. It is formed from Monomethylarsenic acid (MMA) enzymatically.
- Monomethylarsenic acid (MMA) is a minor metabolite and its formulation does not appear to be enzymatic.

- Methylation results in a detoxication of inorganic arsenic (about one order of magnitude per methyl group), and increases the rate of arsenic excretion.
- Methylation of As III is dependent on dose level. Excess As III inhibits the enzymatic processes leading to its own methylation.

This review article also states that the presence of excess trivalent arsenic inhibits the enzymatic conversion of MMA to DMA, because of the propensity of trivalent arsenic to bind to sulfhydryl groups in essential enzymes and cofactors. This leads to tissue deposition of trivalent arsenic and can interfere with deoxyribonucleic acid (DNA) methylation and hence, the DNA repair processes. Also, the rate of conversion of methylated forms of arsenic diminishes beginning at around 250 µg/day, with saturation of methylating capacity at above 500 µg/day in healthy adult males. In addition, when nutrition is poor or when inorganic arsenic is present in excess, then inhibition of methylation occurs and the tissues can be bathed in trivalent arsenic. The authors state that "At chronic doses above about 600 µg/day of arsenic exposure, arsenic then can be deposited and bound to tissues such as skin, lung, and hair, which are particularly rich in sulfhydryl groups." For comparison, the major studies performed with the Taiwanese population (Tseng, 1968, 1977), based on the assumption that males consumed 3.5 L/day, resulted in levels of arsenic exposure of 600, 1,400, and 2,800 µg/day, depending on the concentration of arsenic in different areas. In addition, the calculation of the Taiwanese population did not account for dietary exposure to arsenic, which would have further elevated their exposures. All of these exposures were above the estimated threshold for saturation of methylation reaction.

The conclusions of Marcus and Rispin of why arsenic should be considered a threshold carcinogen were:

- Arsenic intake must exceed the methylating capacity of the body before any lesions (hyperkeratosis or skin cancer) are produced.
- The level of daily excretion of inorganic arsenic in the urine rises only after the methylating capacity of the liver is exceeded.
- The percent of DMA in the urine parallels the rise of inorganic arsenic but plateaus, while the level of inorganic arsenic does not.
- The production of hyperkeratosis is dose-related and has a threshold of 350-400 µg/day of arsenic.
- The biochemical mechanism of action of arsenic has been elucidated: arsenic in excess is capable of binding to sulfhydryl groups, and MMA in excess binds to the dithiol cofactor, blocking enzymatic conversion of itself to DMA and by mass action spills inorganic

arsenic into the blood, which is then deposited in skin and other tissues with high levels of sulfhydryl groups.

- Arsenic binds irreversibly to the lung and lung cancer has been observed to occur where air concentrations exceed 500 $\mu g/m^3$.
- Valentine demonstrated that it is necessary to consume at least 200 µg of arsenic daily before blood arsenic levels rises, demonstrating a physiological threshold for increases in blood level of arsenic.

Lastly, they concluded that the chronic adverse effects of arsenic require the daily ingestion of more than 200-250 micrograms of arsenic, and that by definition is a threshold phenomenon and applies equally well to skin cancer.

A recent review of biological mechanisms of methylation of arsenic (Styblo, et al., 1995) indicated that the capacity for methylation of inorganic arsenic is probably determined by four factors: 1) the extent to which individual steps in the methylation pathway are saturable processes, 2) the availability of cofactors and substrates needed for methylation, 3) the range of genetically determined capacity for arsenic methylation and 4) competition between arsenic and other substrates at rate-limiting steps in the methylation pathway. They indicated that the existence of interindividual variation in capacity for methylation of arsenic is consistent with the concept that arsenic is a threshold carcinogen.

ARSENIC BIOAVAILABILITY

The human studies that serve as the basis for oral toxicity values for arsenic all involve arsenic in drinking water (Tseng, 1977; IRIS, 1995), and therefore reflect the toxicity of soluble forms of arsenic. It is quite obvious that these soluble forms are likely to be more easily absorbed from the gastrointestinal tract than arsenic associated with other media. Freeman et al., (1993) studied the bioavailability of arsenic in several different media, including, intravenous administration, oral soluble forms and oral administration of arsenic associated with soil. This study involved rabbits and the administration of arsenic in solution (1.95 mg/kg), arsenic by intravenous administration (1.95 mg/kg) and three doses of arsenic in soil (0.78, 1.95 and 3.9 mg/kg). Urine and feces were collected from all animals and analyzed for arsenic. The results indicated that absorption of arsenic from solution was approximately 50 to 65%, and 24% in soil compared to the intravenous administration. It is recognized that urinary excretion of total arsenic is a good indicator of the absorbed dose of arsenic, and the amount of arsenic in the urine following 3.9 mg/kg of arsenic in soils is still less than the amount following 1.95 mg/kg in

solution. Approximately 80% of the administered dose of arsenic in soil (for all three dose levels) was recovered from the feces, indicating that the major portion of arsenic was not absorbed into the body.

A recent abstract from California EPA (Salocks, et al., 1996) indicated that less than half of the arsenic in soil associated with mine tailings was soluble in aqueous solution and therefore potentially bioavailable. Vaessen, et al. (1994) studied the absorption of arsenic from soil and compared the results to intravenous administration of arsenic. They concluded that the bioavailability of inorganic arsenic from soil was 8.3 $\pm$ 2.0%.

Accordingly, bioavailability is an important consideration when evaluating the risk associated with arsenic in soil, and it is inappropriate to assume that arsenic in a soil matrix would be absorbed at the same rate as arsenic dissolved in water. While it appears that less than 25% of the administered arsenic in soil is adsorbed into the body, it also appears that the difference in absorption between orally administered arsenic in solution and in soil is between 8 and 48%; therefore, a bioavailability factor for arsenic in soil of 0.28 is recommended (i.e., the midpoint of the two studies).

MECHANISM OF ARSENIC CARCINOGENICITY

Unlike many organic carcinogens, metal compounds (e.g., arsenic, chromium, nickel, beryllium) do not produce direct mutagenic DNA damage or adducts. The proposed mechanism for the carcinogenesis of arsenic is consistent with the threshold effect of arsenic. A recent review on metal carcinogens by Snow (1992) stated that ..."arsenic carcinogenesis shows clear evidence for a threshold below which there is no response." The proposed mode of action is that arsenic has the ability to inhibit DNA repair and induce gene amplification. Arsenic compounds are not mutagenic in either bacterial or mammalian systems, however, arsenite is a co-mutagen, whereby the genotoxic and mutagenic response to other mutagens is enhanced. Li and Rossman (1989) reported that arsenite inhibits DNA ligase II, an enzyme required in the final stages of DNA excision repair. This effect could account for both the co-mutagenic and clastogenic activities of arsenite in mammalian cell systems. Snow (1992) stated that "It is likely that inorganic arsenic compounds also act as cocarcinogens rather than primary carcinogens *in vivo*". Thus, these effects are threshold events, requiring a sufficient concentration of arsenic to initiate these effects.

CONCLUSIONS

It is clear from a through review of the scientific literature that arsenic is a threshold carcinogen because of it's mechanism of action. Further, it clear that arsenic associated with soil is not as bioavailable as arsenic dissolved in water. Lastly, methylation reactions that occur *in vivo* are detoxication reactions, and toxicity only occurs when these enzyme pathways are saturated, leading to a cellular increase in arsenic. However, since the current USEPA oral cancer slope factor is based on the Taiwanese studies of arsenic in drinking water at levels that exceed every estimate of the threshold for saturation of the methylation reactions, it is appropriate to recognize that for low doses, that methylation will be complete or near complete. Accordingly, since the levels found in soil will not exceed the estimated saturation levels, we have made appropriate assumptions in our 14 November 1995 submittal. For purposes of calculations, it is appropriate to assume that 90% of the exposed dose of arsenic will be methylated and not available for participation in a carcinogenic response. In addition, since the bioavailability of arsenic in soil ranges from 8 to 48%, it is appropriate to assume arsenic bioavailability of 28% (the mid point).

In reality, since arsenic is a threshold carcinogen, it should not be evaluated as an oral carcinogen, unless the exposure is greater than 600 µg/day. It is apparent that this approach is becoming recognized, since both Delaware and California no longer evaluate arsenic as oral carcinogens.

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REPORT

Witco Corporation

Halby Chemical Site Review of Ground Water Migration Exposure

3 January 1996

Environmental Resources Management, Inc.
855 Springdale Drive
Exton, Pennsylvania 19341

1.0 INTRODUCTION

This report presents the methodology and results of the calculation of soil criteria based on the protection of ground water for the constituents of interest at the Halby Chemical Site in Wilmington, Delaware. These criteria were developed according to the United States Environmental Protection Agency's (USEPA) *Soil Screening Guidance* (DRAFT) issued in December, 1994. Site specific considerations were incorporated into calculations where appropriate and where site-specific data were available.

2.0 METHODOLOGY

The methodology used for addressing migration of chemicals from soil to ground water reflects the complex nature of fate and transport in subsurface soils. The methodology employed herein back-calculates an acceptable concentration in soil from an acceptable ground water concentration. Acceptable ground water concentrations are typically set at federal drinking water standards, known as Maximum Contaminant Level Goals (MCLGs) or Maximum Contaminant Levels (MCLs). If a chemical constituent does not have a nonzero MCLG or MCL, an acceptable health-based level (calculated from toxicological data) is substituted for the MCLG/MCL. Health-based levels (HBLs) used in this evaluation were developed by Roy L. Smith, Senior Toxicologist at USEPA Region III. These HBLs were considered appropriate because the Halby site is within the jurisdiction of USEPA Region III.

As stated above, MCLs/MCLGs are federal drinking water standards. Likewise, health-based levels are also calculated based on the ingestion of ground water as drinking water. The Halby Chemical Site is located in a heavily industrialized area in Wilmington, Delaware and is zoned commercial as are the surrounding properties. In addition, the shallow and intermediate ground water on this site contains very high levels of naturally occurring total dissolved solids (TDS), rendering the ground water non-potable (TDS levels > 2,500 mg/l). Based on this information, it may be concluded that the ingestion of ground water as drinking water from the Halby Chemical site will not occur under present or future use conditions. Therefore, setting target ground water concentrations at federal drinking water standards was considered overly conservative and inappropriate for the Halby Chemical site. The Federal TDS Secondary MCL is 500 mg/l, which further suggests the non-potability of this water for drinking.

The Public Health Evaluation for OU-1 reinforces this fact when it states that "The results of the field investigation also suggested that the lower Potomac was isolated from the Columbia aquifer and upper Potomac in the area of the site, while the Columbia and the upper Potomac appeared to communicate. Neither of the two shallow aquifers is currently used as a drinking water source at the site, and the water quality in the Columbia is such (high sulfates, sodium, and TDS) that it would be unfit to use for consumptive purposes without extensive pretreatment." In fact, Delaware has designated this portion of the Christina River basin for industrial water use protection only.

In this evaluation, target ground water concentrations were set at 100 times the nonzero MCL/MCLG or, where MCLs/MCLGs were not available, at 100 times health-based concentrations. This protocol was developed by the Pennsylvania Department of Environmental Protection under Pennsylvania's Act 2 Land Recycling Program (PADEP, 1995) for aquifers with TDS levels greater than 2,500 mg/l, and is considered an appropriate approach for the Halby Chemical site.

For estimating the acceptable soil concentration for migration to ground water, the following equilibrium soil/water partition equation that describes the ability of chemicals to sorb to organic carbon in soil was used. The following equation is taken from USEPA (1994), with the terms defined on Table 1.

$$SSL = C_W \times DAF \times (K_d + ((O_W + O_a H)/Pb)) \quad (3)$$

As chemicals move from soil into ground water, the concentration in ground water for each chemical may be reduced by a variety of physical, chemical, and biological processes. This reduction in the chemical concentration is expressed as the dilution attenuation factor (DAF), which is defined as the ratio of the soil leachate concentration to the receptor exposure point concentration (USEPA 1994). The acceptable ground water limit MCL/MCLG/HBL * 100 is multiplied by the DAF to obtain a target soil leachate concentration for the partition equation.

Insufficient site-specific data were available to calculate a site-specific DAF. Therefore, a DAF of 10 was used according to USEPA, 1994 for the calculation of generic soil screening levels. Values for Henry's law constant and partitioning coefficients were taken from the scientific literature. References for each chemical-specific parameter are noted on Table 1.

The partitioning of inorganic constituents in soil is more complex than for organics. A variety of soil conditions, in addition to organic carbon content, will affect the derivation of the partitioning coefficient for

inorganics. In USEPA, 1994, MINTEQ2, an equilibrium geochemical speciation model was used to estimate Kd values for metals, which incorporates a range of pH conditions into Kd calculations. Where available, Kd values for inorganics were taken from the *Soil Screening Guidance.* (USEPA, 1994).

3.0 *RESULTS*

Calculated soil levels protective of ground water, incorporating a DAF of 10, are presented on Table 2. A review of all data for OU-1 and OU-2 shows that only a limited number of locations on site exceed the SSLs including portions of the lagoon, the ditch, and the process area. Table 3 provides a list of those locations.

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Table 1 *Soil Screening Level Definition of Terms*

Parameter	Site-Specific or Default Value
SSL = Soil screening level (mg/kg)	--
C_w = Target soil leachate concentration (mg/L)	(nonzero MCLG, MCL, or HBL (if no MCLG/MCL)) * 100
MCLG = Maximum Contaminant Level Goal (mg/L)	
MCL = Maximum Contaminant Level (mg/L)	
HBL = Health-based Level (mg/L)	
DAF = Dilution Attenuation Factor	10 (default)
K_d = Soil-water partition coefficient (L/kg)	chemical-specific (for inorganics); Koc x foc (for organics)
K_{oc} = Soil organic carbon/water partition coefficient (L/kg)	chemical-specific
f_{oc} = Fraction organic carbon in soil (g/g)	0.02 (default)*
O_w = Water-filled soil porosity (L/L)	0.3 (default)
O_a = Air-filled soil porosity (L/L)	0.13 (default)
H = Henry's law constant (unitless) atm-m3/mol	chemical-specific
P_b = Dry soil bulk density (kg/L)	1.5 (default)

Table Notes:

All default values were taken from the DRAFT Soil Screening Guidance (USEPA, 1994) except * which was taken from USEPA Risk Assessment Guidance for Superfund, Human Health Evaluation Manual Part B: Development of Risk-Based Preliminary Remediation Goals (USEPA, 1991).

Table 2 *Calculation of Soil Concentrations Protective of Ground Water*
Halby Chemical Site
Wilmington, Delaware

	Cw x 100 mg/l		Koc l/kg		Kd l/kg	H atm-m3/mol		SL mg/kg
Volatile Organic Compounds								
Acetone	370	b	0.37	a	0.0074	3.97E-05	a	767.90
Benzene	0.5	m	83	a	1.66	5.59E-03	a	9.40
2-Butanone (MEK)	190	b	1.23	a	0.0246	4.66E-05	a	427.05
Carbon disulfide	2.1	b	240	a	4.8	1.70E-02	c	106.27
2-Hexanone (BMK)	190	s	135	a	2.7	1.75E-03	a	5521.81
Methylene chloride	0.41	b	8.8	a	0.176	2.03E-03	a	1.57
4-Methyl-2-pentanone (MIBK)	290	b	6.2	a	0.124	1.49E-05	a	939.75
Toluene	75	b	150	a	3	6.37E-03	a	2416.98
Trichloroethene	0.16	b	65	a	1.3	9.10E-03	a	2.45
Semivolatile Organic Compounds								
Acenaphthene	220	b	4848	c	96.96	1.50E-04	a	213753.17
Anthracene	1100	b	18620	a	372.4	6.51E-05	a	4098602.54
Benzo(a)anthracene	0.0092	b	1.30E+06	a	26000	6.60E-07	a	2392.02
Benzo(b)fluoranthene	0.0092	b	5.50E+05	a	11000	1.20E-05	a	1012.02
Benzo(k)fluoranthene	0.092	b	4.36E+06	a	87200	1.04E-03	a	80224.19
Benzo(g,h,i)perylene^	150	^	7.70E+06	a	154000	1.40E-07	a	2.31E+08
Benzo(a)pyrene *	0.00092	b	915911	c	18318	8.36E-07	c	168.53
Bis(2-ethylhexyl)phthalate	0.6	m	100000	a	2000	1.10E-05	a	12001.20
Carbazole	0.34	e	2441	c	48.82	8.12E-05	c	166.67
Chrysene	0.92	b	245470	a	4909.4	7.26E-20	a	45168.32
Dibenzofuran	15	b	12590	a	251.8	1.69E-02	^	37809.01
Di-n-butylphthalate	370	b	1380	a	27.6	6.30E-05	a	102860.83
2,4-Dichlorophenol	11	b	871	a	17.42	6.66E-06	a	1938.20
Fluoranthene	150	b	41687	a	833.74	1.69E-02	a	1251000.08
Fluorene	150	b	5011	a	100.22	2.10E-04	a	150631.12
Indeno(1,2,3-cd)pyrene	0.0092	b	3.09E+07		618000	2.92E-20	a	56856.02
2-Methylnaphthalene^	150	^	8511	a	170.22	1.69E-02	^	255720.08
2-Methylphenol (o-cresol)	18	b	54	c	1.08	1.64E-06	c	230.40
Naphthalene	150	b	1122	a	22.44	4.60E-04	a	33962.45
Phenol	2200	b	27	a	0.54	2.70E-07	a	16280.02
Phenanthrene^	150	^	38904	a	778.08	4.00E-05	a	1167420.21
Pyrene	110	b	45709	a	914.18	1.09E-05	a	1005818.04
Pesticides								
Aldrin	0.0004	b	48394	c	967.88	1.03E-04	c	3.87
4,4'-DDT	0.02	b	239883	a	4797.7	3.80E-05	a	959.57
Dieldrin	0.00042	b	35481	a	709.62	2.00E-07	a	2.98
Endosulfan I	22	b	2041	a	40.82	1.01E-04	a	9024.48
Endosulfan II	22	b	2344	a	46.88	1.91E-05	a	10357.61
alpha-Chlordane	0.2	m	1.00E+06	a	20000	8.60E-04	h	40000.41
gamma-Chlordane	0.2	m	1.00E+06	a	20000	1.30E-03	h	40000.41
Heptachlor	0.04	m	21877	a	437.54	2.30E-03	a	175.10
Metals								
Aluminum	20	sm	N/A		0	0.00E+00		N/A
Antimony	0.6	m	N/A		0	0.00E+00		N/A

ORIGINAL (Red)

Table 2 *Calculation of Soil Concentrations Protective of Ground Water*
Halby Chemical Site
Wilmington, Delaware

	Cw x 100 mg/l		Koc l/kg	Kd l/kg		H atm-m3/mol	SL mg/kg
Arsenic	5	m	N/A	29	c	0.00E+00	1460.00
Barium	200	m	N/A	1.4	c	0.00E+00	3200.00
Beryllium	0.4	m	N/A	4600	c	0.00E+00	18400.80
Cadmium	0.5	m	N/A	120	c	0.00E+00	601.00
Chromium	10	m	N/A	19	c	0.00E+00	1920.00
Cobalt	220	b	N/A	10000	d	0.00E+00	22000440.00
Copper	130	m	N/A	10000	c	0.00E+00	13000260.00
Cyanide (total)	20	m	N/A	1		0.00E+00	240.00
Lead	1.5	m	N/A	39810	d	0.00E+00	597153.00
Manganese	5	sm	N/A	100000	d	0.00E+00	5000010.00
Mercury	0.2	m	N/A	145	c	0.00E+00	290.40
Nickel	10	m	N/A	21	c	0.00E+00	2120.00
Selenium	5	m	N/A	5	c	0.00E+00	260.00
Thallium	0.05	m	N/A	71	c	0.00E+00	35.60
Thiocyanate	20	+	N/A	1		0.00E+00	240.00
Vanadium	26	b	N/A	0		0.00E+00	N/A
Zinc	500	sm	N/A	420	c	0.00E+00	2101000.00

Notes:
a=Montgomery and Welkom, 1990 and 1991.
b=USEPA, 1995.
c=USEPA, 1994.
d=Dragun, 1988.
e=USEPA, 1995a.
h=Howard, 1989.
s=MEK used as surrogate
^ = fluoranthene used as surrogate
m=MCL
sm=secondary MCL
+=cyanide used as a surrogate
N/A=not applicable
NA=Insufficient data to calculate.

Table 3 - Soils Exceeding Site Screening Levels for Ground Water Migration

Volatile Organic Compounds	Sample Location	Concentration (mg/kg)
CS_2	SED-05	110
CS_2	SED-02	160
CS_2	SED-03	9,400
CS_2	SED-24	222
CS_2	HCS-2	2,100
CS_2	HCS-3	5,900
CS_2	HAS-2	8,600
CS_2	HCS-4	120
CS_2	HCS-5	8,100
CS_2	SSS-04	1,800
CS_2	HAS-3	41,000
CS_2	SSS-16	320
CS_2	HAS-4	16,000
CS_2	HAS-6	730
CS_2	HCS-6	6,400
CS_2	HAS-5	98,000
CS_2	HCS-11	140
CS_2	HCS-12	7,300
CS_2	HCS-13	28,000
CS_2	HCS-14	1,300
CS_2	HCS-15	107,000
CS_2	HCS-16	39,000
CS_2	HCS-8	110,000
CS_2	SB-02	432
CS_2	HCS-17	11,000
CS_2	HCS-18	430
Methylene chloride	SED-08	4.6
Methylene chloride	HAS-6	110
TCE	SED-02	5.2

Table 3 (cont) - Soils Exceeding Site Screening Levels for Ground Water Migration

BNAs	Sample Location	Concentration (mg/kg)
Benzo (a) pyrene	TG-1-02	300
Benzo (a) pyrene	TG-1-04	400
Benzo (a) pyrene	TG-1-01	200

Metals	Sample Location	Concentration (mg/kg)
Arsenic	SED-8	3,110
Arsenic	SED-5	2,980
Arsenic	SED-2	2,520
Arsenic	SSS-25	1,700
Arsenic	SB-13	2,350
Arsenic	HAS-6	3,280
Arsenic	HAS-5	4,260
Arsenic	HAS-4	4,470
Arsenic	HAS-3	3,590
Arsenic	SSS-4	4,430
Arsenic	SB-02	1,670

Table 1 - Phase II Field Treatability Study Pretreatment Carbon Disulfide Concentrations (mg/kg)

	TSB-2		TSB-9		TSB-11		TSB-12	
Treatment Status:	Pre-treat	Pre-treat	Pre-treat	Pre-treat	Pre-treat	Pre-treat	Pre-treat	Pre-treat
Sample Location:	Middle	Perimeter	Middle	Perimeter	Middle	Perimeter	Middle	Perimeter
Sampling Date:	3/6/97	3/6/97	3/6/97	3/6/97	3/7/97	3/7/97	3/6/97	3/7/97
Sample Depth								
0-1'	0.690	0.002 J	0.022	0.003 (0.074 dup)	0.80 J	0.006	ND	0.003
1'-2'			0.003		0.120	0.005 J (dup)		
2'-3'	0.006 J	0.018	0.004	0.001	0.080	0.006 J	0.021	0.008
3'-4'		0.003				0.009 J (dup)	0.010	
4'-5'	100	130	2.30	0.73	460	220	1.60	0.620 J
5'-6'		31					5.40	
6'-7'		190	7.20		10,000	2,200	34	10
7'-8'			1,800		7,400		3,900	5,000
8'-9'	120,000	1,500	820	ND	610		2,800	160
9'-10'		13,000	7,200	7,000	6,700		7,500	12,000
10'-11'	4,500	8,500	10,000	280	18,000	8,600	8,000	4,800
11'-12'		1,500	5,400		1,900	2,100	2,800	1,500

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Notes:
J - Estimated Value
ND - Not Detected
Blank cells indicate no recovery interval

Table 2 - Phase II Field Treatability Study Tr...ment Protocols

	TSB-2					
Treatment Status: Sample Location: Sampling Date: Sample Depth	Pre-treat Middle 3/6/97	Pre-treat Perimeter 3/6/97	Average Conc. per Interval (mg/kg)	Lbs of CS2 per yard of soil	Lbs of Percarb. per yard of soil (11:1 molar ratio)	Estimated Lbs Percarb. per Interval
0-1'	0.690	0.002	0.346	0.00	0.02	0
1'-2'						
2'-3'	0.006	0.018	0.008	0.00	0.00	0
3'-4'		0.003				
4'-5'	100	130	90	0.24	4	9
5'-6'		31				
6'-7'		190	190	0.51	9	20
7'-8'						
8'-9'	120,000	1,500	63,625	172	3,130	6,542
9'-10'		13,000				
10'-11'	4,500	8,500	4,750	13	234	488
11'-12'		1,500				

TOTAL PERCARB. 7,059 lbs

Notes

1) Estimated pounds of sodium percarbonate per interval for TSB-2R and TSB-9R is based on Terra's 6-foot diameter auger or a 28.3 sq. ft. test plot area.

2) Estimated pounds of percarbonate per interval for TSB-11 and TSB-12 is based on 3'x10' test plot area (which is suitable for CBA's equipment) or a 30 sq. ft. test plot area. In addition, due to the nature of CBA's equipment the total dose of sodium percarbonate will be added at the surface of the test plot and then mixed to depth.

Table (Cont.) - Phase II Field Treatability Study Treatment Protocols

	TSB-9					
Treatment Status: Sample Location: Sampling Date: Sample Depth	Pre-treat Middle 3/6/97	Pre-treat Perimeter 3/6/97	Average Conc. per Interval (mg/kg)	Lbs of CS2 per yard of soil	Lbs of Percarb. per yard of soil (11:1 molar ratio)	Estimated Lbs Percarb. per Interval
0-1'	0.022	0.003	0.032	0.00	0.00	0
1'-2'	0.003	0.074				
2'-3'	0.004	0.001	0.003	0.00	0.00	0
3'-4'						
4'-5'	2.30	0.73	1.515	0.00	0.07	0
5'-6'						
6'-7'	7.20		904	2	44	93
7'-8'	1,800					
8'-9'	820	ND	5,760	16	283	592
9'-10'	7,200	7,000				
10'-11'	10,000	280	3,990	11	196	410
11'-12'	5,400					

TOTAL PERCARB. 1,096 lbs .

Notes

1) Estimated pounds of sodium percarbonate per interval for TSB-2R and TSB-9R is based on Terra's 6-foot diameter auger or a 28.3 sq. ft. test plot area.

2) Estimated pounds of percarbonate per interval for TSB-11 and TSB-12 is based on 3'x10' test plot area (which is suitable for CBA's equipment) or a 30 sq. ft. test plot area. In addition, due to the nature of CBA's equipment the total dose of sodium percarbonate will be added at the surface of the test plot and then mixed to depth.

Table 2 (cont.) - Phase II Field Treatability Study Treatment Protocols

	TSB-11					
Treatment Status: Sample Location: Sampling Date: Sample Depth	Pre-treat Middle 3/7/97	Pre-treat Perimeter 3/7/97	Average Conc. per Interval (mg/kg)	Lbs of CS2 per yard of soil	Lbs of Percarb. per yard of soil (11:1 molar ratio)	Estimated Lbs Percarb. per Interval
0-1'	0.800	0.006	0.463	0.00	0.02	0
1'-2'	0.120	0.005				
2'-3'	0.080	0.006	0.044	0.00	0.00	0
3'-4'		0.009				
4'-5'	460	220	340	0.92	16.73	37
5'-6'						
6'-7'	10,000	2,200	5,450	15	268	595
7'-8'	7,400					
8'-9'	610		3,655	10	180	399
9'-10'	6,700					
10'-11'	18,000	8,600	7,650	21	376	835
11'-12'	1,900	2,100				

TOTAL PERCARB. 1,867 lbs .

Notes

1) Estimated pounds of sodium percarbonate per interval for TSB-2R and TSB-9R is based on Terra's 6-foot diameter auger or a 28.3 sq. ft. test plot area.

2) Estimated pounds of percarbonate per interval for TSB-11 and TSB-12 is based on 3'x10' test plot area (which is suitable for CBA's equipment) or a 30 sq. ft. test plot area. In addition, due to the nature of CBA's equipment the total dose of sodium percarbonate will be added at the surface of the test plot and then mixed to depth.

	TSB-12					
Treatment Status: **Sample Location:** **Sampling Date:** **Sample Depth**	**Pre-treat Middle 3/6/97**	**Pre-treat Perimeter 3/7/97**	**Average Conc. per Interval (mg/kg)**	**Lbs of CS2 per yard of soil**	**Lbs of Percarb. per yard of soil (11:1 molar ratio)**	**Estimated Lbs Percarb. per Interval**
0-1'	ND	0.003	0.002	0.00	0.00	0
1'-2'						
2'-3'	0.021	0.008	0.012	0.00	0.00	0
3'-4'	0.010					
4'-5'	1.60	0.62	2	0.01	0.10	0
5'-6'	5.40					
6'-7'	34	10	2,236	6	110	244
7'-8'	3,900	5,000				
8'-9'	2,800	160	5,615	15	276	613
9'-10'	7,500	12,000				
10'-11'	8,000	4,800	4,275	12	210	467
11'-12'	2,800	1,500				

TOTAL PERCARB. 1,325 lbs

Notes

1) Estimated pounds of sodium percarbonate per interval for TSB-2R and TSB-9R is based on Terra's 6-foot diameter auger or a 28.3 sq. ft. test plot area.

2) Estimated pounds of percarbonate per interval for TSB-11 and TSB-12 is based on 3'x10' test plot area (which is suitable for CBA's equipment) or a 30 sq. ft. test plot area. In addition, due to the nature of CBA's equipment the total dose of sodium percarbonate will be added at the surface of the test plot and then mixed to depth.

Table 3 - Analytical Parameter Sample Matrix

No.	Analytical Parameter	Sample Matrix	Method Description
1	Total Cyanide	Soil	SW-846-9010A
2	Leachable Cyanide	Soil	ASTM D-3987-85/SW-846 9010A
3	CS2	Soil	DNREC HSCA/CLP SOW 3.1
4	TCLP As	Soil	SW-846 1311/6010A
5	Total As	Soil	CLP SOW 3.1
6	Sulfide	Soil	SW-846-9030A
7	Sulfate	Soil	ASTM D-3987-85/EPA 375.4
8	pH	Soil	SW-846-9045
9	Hydrogen Sulfide	Off-gas	EPA Method 16
10	Hydrogen Cyanide	Off-gas	OSHA ID-120
11	Sulfur Dioxide	Off-gas	EPA Method 16M
12	Carbon Disulfide Carbonyl Sulfide	Off-gas	EPA Method 16M
13	Ammonia	Off-gas	NIOSH 6015

REPORT

Witco Corporation

Halby Chemical Site Development of Human Health Screening Levels for Additional Constituents

25 March 1996

Environmental Resources Management, Inc.
855 Springdale Drive
Exton, Pennsylvania 19341

1.0 INTRODUCTION

This document presents risk-based concentration screening levels (RBCs) in response to Item 8.3g of the CERCLA §106 Order (Order) issued by USEPA Region III to Witco Corporation at the Halby Chemical Site, Wilmington, Delaware (Site). Order Item 8.3g specifically requires Witco to "develop and submit for approval, soil clean-up level(s) sufficient to protect human health and the environment."

This document focuses on those constituents identified in the CH_2MHill RA as constituents of concern (COCs) for the surficial and subsurface soil that failed the initial screening. In accordance with USEPA Region III guidance (EPA Region III, 1993), COCs were selected during the initial screening by comparing constituent concentrations to RBCs based on conservative endpoints (e.g., cancer risk set to equal 1.0E-06 and hazard quotient set to equal 0.1). This approach is intended to identify and focus on dominant contaminants of concern and exposure routes early in the risk assessment process. Constituents that failed the screening process were retained as COCs and carried through the CH_2MHill quantitative risk assessment. To address the identified COCs that posed the greatest risk, this document provides alternative RBCs to those that were used for risk-based screening of constituents presented in the CH_2MHill RA. Similar to the CH_2MHill RA, the approach used herein is to develop RBCs to gauge the risk posed by the site constituents using realistic assumptions. Further, the technical approach used is consistent with the methods CH_2MHill applied for the Operable Unit 2 (OU2) Risk Assessment. However, this evaluation should not be used to represent a risk assessment comparable to that required for an RI/FS. Such an evaluation will either be addressed in EPA's final RI/FS for OU2, or as a future activity under the Order.

Based on the previous investigation of the site by USEPA, investigative work to date conducted by Witco and the results of the CH_2MHill RA, the constituents that appear to drive the risk are arsenic, carbon disulfide, manganese, and thiocyanate. These constituents have been addressed separately in a report issued on 15 November 1995. However, to complete the requirements outlined in Item 8.3g, as described above, alternative RBC screening levels have been developed for the remaining COCs detected in the surficial and subsurface soil of OU2 that contributed to the potential risk. Alternative RBCs for exposure scenarios deemed appropriate for OU2 are presented for each constituent.

There are no current users of the ground water and future use of the regional ground water is not expected. Migration of ground water has been addressed in "Halby Chemical Site - Review of Ground Water Migration Exposure" submitted to EPA on March XX, 1996.

2.0 *DEVELOPMENT OF ALTERNATIVE RISK-BASED CONCENTRATION SCREENING LEVELS*

Calculation of alternative risk-based concentrations (RBCs) for noncarcinogens and carcinogens, provided herein, follow methodology provided in the Draft Soil Screening Guidance (USEPA 1994) and methodology used by CH_2MHill in the March 1995 Risk Assessment (RA) for the Halby Chemical site. However, in accordance with USEPA guidelines, certain parameters used in the alternative RBC development have been redefined to provide more realistic concentration levels.

RBCs developed in the CH_2MHill RA considered potential exposures by residential and industrial receptor populations using typical USEPA default parameters. Further, acceptable risk and toxicity endpoints were set by CH_2MHill at 1.0.E-06 for potential exposures to carcinogens and a Hazard Quotient of 0.1 for potential exposure to systemic toxicants. Future residential land use of OU2 is not expected, hence, residential users are not included in the alternative RBC development. For purposes of this assessment, the development of alternative RBCs for OU2 focuses on using realistic exposure scenarios, thus appropriate receptor populations will consider only industrial exposures. Site workers are expected to be exposed only to constituents detected in surficial soils, whereas construction workers may be exposed to constituents detected both in surficial and subsurface soils.

According to the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), the acceptable risk range for carcinogens is 1.0E-04 to 1.0E-06. Therefore, the alternative RBCs for carcinogenic constituents at OU2 were set to equal the midrange cancer endpoint of 1.0E-05. The 1.0E-05 risk is considered an acceptable risk, particularly for industrial sites. Further, it should be noted that according to the Draft Soil Screening Guidance (USEPA 1994), soil screening levels (SSLs) can be set at a hazard quotient (HQ) of 1.0 for chemicals with different endpoints. The HI is the standard measure of risk for noncarcinogenic constituents. Considering that the SSLs are based on Reference Doses that are typically set to be protective of the most sensitive populations, using an HI of 1.0 in such a manner is reasonable and conservative. Therefore, we have applied this HI value for each constituent since they each have different

target organs for toxicity, except for mercury which may cause an neurotoxic effects similar to that of manganese and carbon disulfide. Because of similar effects posed by these constituents, the HQ for each is set at 0.33. The principal target organs and principal critical effects for each noncarcinogenic constituent are present in Table 2-1.

Table 2-1 Target Organ and Critical Effects for Noncarcinogens

	Target Organ	Critical Effect
bis(2-ethylhexyl) phthalate	liver	increased weight
aluminum	(a)	(a)
antimony	blood	altered chemistry
beryllium	(a)	(a)
mercury	nervous system	neurotoxicity
vanadium	(a)	(a)

(a) - Reference dose based on No Observable Adverse Effects Level (NOAEL), no adverse effects reported from selected study.

Calculation of risk-based concentrations for noncarcinogens and carcinogens follow the methodology as presented in the CH_2MHill risk assessment and are presented in the following equations.

NONCARCINOGENS

$$RBC\ (mg/kg) = \frac{THI \cdot BW \cdot AT_{nc} \cdot 365\ days/year}{EF \cdot ED \cdot ((1/I_{ng}RfD) \cdot 1E^{-6}kg/mg \cdot I_{ng}R) + ((1/I_{nh}RfD) \cdot I_{nh}R \cdot ET \cdot (1/VF + 1/PEF))}$$

CARCINOGENS

$$RBC\ (mg/kg) = \frac{RISK \cdot BW \cdot AT_{c} \cdot 365\ days/year}{EF \cdot ED \cdot (OSF \cdot 1E\text{-}6mg/kg \cdot I_{ng}R) + (ISF \cdot I_{nh}R \cdot ET \cdot (1/VF + 1/PEF))}$$

The input parameters used to calculate RBCs are defined in the following table.

Table 2-2 Input Parameters for RBC Calculations

	Parameter	Site Worker	Construction Worker
BW	Body Weight (kg)	70	70
AT_{nc}	Non-carcinogen Averaging Time (years)	25	1
AT_c	Carcinogen Averaging Time (years)	70	70
EF	Exposure Frequency (days/year)	250	10
ED	Exposure Duration (years)	25	1
$I_{ng}R$	Ingestion Rate (mg/day)	50	480
$I_{nh}R$	Inhalation Rate (M^3/hr)	2.5	2.5
ET	Exposure Time (hours/day)	8	8
PEF	Particulate Emission Factor (M^3/kg)	$6.79x10^8$	$6.79x10^8$
RISK	Target Risk	1.0E-05	1.0E-05
THI	Target Hazard Index	1	1

These values are very conservative and may need to be re-evaluated, using sound technical judgment, for use in any future risk assessment activities. In addition, the calculations have been completed with an assumption that the chemical substances are 100% absorbed from a soil matrix. Likewise, this assumption may also need to be modified in any future activities.

Using the above defined equations and parameters, the alternative RBCs for the site worker and construction worker are provided in Tables 2-3 and 2-4. In addition, RBCs developed by CH_2MHill are also provided in each

table for comparative use. Supporting information used to calculate the RBCs are provided in Appendix A.

Table 2-3 ***COMPARISON OF ALTERNATIVE RBCS FOR SITE WORKERS (mg/kg in surficial soil)***

	Halby RA Site Worker	Alternative RBC Site Worker	Maximum Concentration
benzo(a)pyrene	0.78	7.84[a]	0.37 (8.1) [c]
benzo(b)fluoranthene	7.8	78.4[a]	9
benzo(k)fluoranthene	2.5	784[a]	5.7
chrysene	2.4	7840[a]	8.1
indeno(1,2,3-cd) pyrene	5.1	78.4[a]	5.8
aluminum	21,000	2,040,000[b]	32,000
mercury	6.4	202[b]	12
vanadium	150	14,300[b]	290

[a] Application of cancer risk of 1.0E-05

[b] Application of hazard index of 1.0 (except mercury which is set at 0.33)

[c] One sample location (SB-18) reported concentration of 8.1. All other samples reported at significantly lower levels.

Table 2-4 COMPARISON OF ALTERNATIVE RBCS FOR CONSTRUCTION WORKERS
(mg/kg in surficial and subsurface soil)

	Halby RA Construction Worker[a]	Alternative RBC Construction Worker[a]	Maximum Concentration (surficial/subsurface soil) *
benzo(a)pyrene	2	511[a]	0.37 / 3.6 *
benzo(b)fluoranthene	7.8	5,100[a]	9
benzo(k)fluoranthene	2.5	51,000[a]	7
chyrsene	2.4	511,000[a]	8.1
indeno (1,2,3-cd) pyrene	5.1	5,110[a]	5.8
carbazole	1.2	186,000[a]	1.4
bis(2-ethylhexyl) phthalate	0.032	106,000[a]	0.77
aluminum	21,000	5,320,000[b]	32,000
antimony	8.5	2,130[b]	23.4 (3,800)[c]
beryllium	3.5	867[a]	7.1
mercury	6.4	5,270[b]	12
vanadium	150	37,300[b]	290

[a] Application of cancer risk of 1.0E-05.

[b] Application of hazard index of 1.0 (except mercury which is set at 0.33).

[c] One sample location (SB-20) reported a concentration of 3,800. All other samples reported at significantly lower levels.

3.0 *COMPARISON OF OU2 SOIL DATA TO ALTERNATIVE RBCS*

The appropriate receptors for the Halby site at the present time are the construction worker. For longer term evaluation, the site worker is also included. Incorporating the current toxicological information discussed herein, but retaining an individual Hazard Quotient of 1.0 (except for mercury which is set at 0.33) and a 1×10^{-5} carcinogenic risk, the above listed RBCs would apply.

The application of these RBCs should consider the likely exposure scenarios. For the construction worker, exposure could occur to either surface or subsurface soils. Therefore, the maximum concentration of all soils should be compared to the RBCs for the construction worker. For the site workers, the only probable exposure would be to surficial soils.

A review of the data shows that no surficial and subsurface soil concentrations exceeded the RBCs for the site worker or the construction worker, except for one detection of benzo(a)pyrene in surficial soil (SB-18) and antimony detected in one subsurface soil sample (SB-20 at 4 to 6 feet). Sample SB-18 is located behind the truck repair facility. Sample location SB-20 is located in the lagoon area.

4.0 *EVALUATION OF FATE AND TRANSPORT*

Anthropogenic combustion of fossil fuels including wood, coal, and oil are the major sources of PAHs in the environment (Sims and Overcash, 1983). Likewise, the presence of PAHs in the soil of OU2 is probably due to the proximity of the coal and coke piles, located adjacent to the Witco property boundary and not related to any historic use of PAHs in Witco plant processes. However, because benzo(a)pyrene was detected at a concentration slightly above the RBC, environmental fate processes may be used to show that the presence of benzo(a)pyrene poses not significant risk to human health.

Several studies have been recently conducted that show that polycyclic aromatic hydrocarbons (PAHs) readily biodegrade under certain environmental conditions. PAHs may undergo a variety of fate processes such as chemical oxidation, photolysis, and volatilization; however, microbial degradation is the major process affecting the persistence of PAHs in the environment. Several studies have been conducted to determine the rates of degradation of aromatic compounds in soil. Half-life values reported for benzo(a)pyrene in soil ranged from 30 to 694 days at a temperature range of 15 - 25 C (Sims and Overcash, 1983). A recent

study conducted by Park et al. (1990), reported 95% confidence limit half-life values for benzo(a)pyrene ranging from 178 to 315 days in McLaurin sandy loam soil. Considering this information, it may be assumed that microbiological degradation of benzo(a)pyrene will continue and will eventually reduce the levels of benzo(a)pyrene found in the soil of OU2 to nondetectable levels. Considering the largest half-life values of 315 days, one can expect that within 0.86 years the level of benzo(a)pyrene reported in soil (based on sampling data reported in May 1993) will decrease by one half. Therefore, the level of benzo(a)pyrene in sample SB-20 has likely biodegraded below the RBC to approximately one-half of the concentration previously reported

Antimony was also detected above the RBC in one subsurface soil sample (SB-20 at 4-6 feet). This concentration was not consistent with the remaining soils samples with concentrations ranging from 4.5 to 23.4 mg/kg. Because the elevated sample appears to be an analytical anomaly, using the average concentration of antimony, which is well below the alternative RBC, would provide a more representative level for comparison. Furthermore, construction worker activities would not likely place an individual in the area of SB-20 for any period of time, thus further reducing any potential risk.

5.0 *CONCLUSIONS*

Based on the development of alternative RBCs and subsequent comparison to surficial and subsurface soils at OU2, further removal actions are not warranted for the additonal COCs evaluated herein. As described above, the single detection of benzo(a)pyrene above the RBC will likely naturally biodegrade over time and will not pose a significant health risk to the site worker or construction worker. Likewise, the single detection of antimony above the alternative RBC is also considered an insignificant potential health risk to on-site workers. No further removal actions are necessary for the OU2 soils.

6.0 *REFERENCES*

EPA Region III. 1993. Selecting Exposure Routes and Contaminants of Concern by Risk Based Screening. EPA/903/R-93-001.

EPA Region III. 1995. Risk-Based Concentration Table, July - December 1995.

EPA. 1994. Soil Screening Guidance. EPA/540/R-94-101.

Park, K.S., R.C. Sims, R.R. DuPont, W.J. Doucette, and J.E. Matthews. 1990. Fate of PAH Compounds in Two Soil Types: Influence of Volatilization, Abiotic Loss and Biological Activity. Environmental Toxicol & Chem. 9:187-195.

Sims, R.C. and M.R. Overcash. 1983. Fate of Polynuclear Aromatic Compounds (PNAs) in Soil-Plant Systems. Residue Reviews 88: 1-68.

Table 1
Soil Risk-Based Concentrations - Potential Site Worker Exposure
Surface and Subsurface Soils
Halby Chemical Site
Wilmington, Delaware

Constituent of Concern	Inhalation RfD (mg/kg/day)		Oral RfD (mg/kg/day)		Inhalation CPF (1/mg/kg/day)		Oral CPF (1/mg/kg/day)		US EPA Carcinogenic Classification	Noncarcinogenic Screening Level (mg/kg)	Carcinogenic Screening Level (mg/kg)	Selected Screening Level (mg/kg)
benzo(a)pyrene	NA		NA		6.10E-02	w	7.30E+00	i	B2	NA	7.84E+00	7.84E+00
benzo(b)fluoranthene	NA		NA		6.10E+00	e	7.30E-01	e	B2	NA	7.80E+01	7.80E+01
benzo(k)fluoranthene	NA		NA		6.10E-01	e	7.30E-02	e	B2	NA	7.80E+02	7.80E+02
chyrsene	NA		NA		6.10E-03	e	7.30E-03	e	B2	NA	7.84E+03	7.84E+03
indeno(1,2,3-cd)pyrene	NA		NA		6.10E-01	e	7.30E-01	e	B2	NA	7.84E+01	7.84E+01
aluminum	NA		1.00E+00	e	NA		NA		-	2.04E+06	NA	2.04E+06
mercury	8.57E-05	i	3.00E-04	i	NA		NA		B2	2.02E+02	NA	2.02E+02
vanadium	NA		7.00E-03	h	NA		NA		C	1.43E+04	NA	1.43E+04

Notes:
RBCs calculated following guidance provided in USEPA Soil Screening Guidance, 1994. Target risk level set at 1.0E-05 and target hazard level set at 1.0 (except mercury which is set at 0.33).
NA= Information not available from IRIS or HEAST.
i=USEPA Integrated Risk Information System (IRIS), accessed December, 1995.
h= USEPA Health Effects Assessment Summary Tables (HEAST), FY 1995.
w= Value withdrawn.
e=EPA-NCEA Regional Support provisional value.

Table 2
Soil Risk-Based Concentrations - Potential Construction Worker Exposure
Surface and Subsurface Soils
Halby Chemical Site
Wilmington, Delaware

Constituent of Concern	Inhalation RfD (mg/kg/day)		Oral RfD (mg/kg/day)		Inhalation CPF (1/mg/kg/day)		Oral CPF (1/mg/kg/day)		US EPA Carcinogenic Classification	Noncarcinogenic Screening Level (mg/kg)	Carcinogenic Screening Level (mg/kg)	Selected Screening Level (mg/kg)
benzo(a)pyrene	NA		NA		6.10E-02	w	7.30E+00	i	B2	NA	5.11E+02	5.11E+02
benzo(b)fluoranthene	NA		NA		6.10E+00	e	7.30E-01	e	B2	NA	5.10E+03	5.10E+03
benzo(k)fluoranthene	NA		NA		6.10E-01	e	7.30E-02	e	B2	NA	5.10E+04	5.10E+04
chrysene	NA		NA		6.10E-03	e	7.30E-03	e	B2	NA	5.11E+05	5.11E+05
indeno(1,2,3-cd)pyrene	NA		NA		6.10E-01	e	7.30E-01	e	B2	NA	5.11E+03	5.11E+03
carbazole	NA		NA		NA		2.00E-02	h	B2	NA	1.86E+05	1.86E+05
bis(2-ethylhexyl)phthalate	NA		2.00E-02	i	NA		1.40E-02	i	B2	1.06E+05	2.66E+05	1.06E+05
aluminum	NA		1.00E+00	e	NA		NA		-	5.32E+06	NA	5.32E+06
antimony	NA		4.00E-04	i	NA		NA		-	2.13E+03	NA	2.13E+03
beryllium	NA		5.00E-03	i	8.40E+00	i	4.30E+00	i	B2	2.66E+04	8.67E+02	8.67E+02
mercury	8.57E-05	i	3.00E-04	i	NA		NA		D	5.27E+03	NA	5.27E+03
vanadium	NA		7.00E-03	h	NA		NA		-	3.73E+04	NA	3.73E+04

Notes:
RBCs calculated following guidance provided in USEPA Soil Screening Guidance, 1994. Target risk level set at 1.0E-05 and target hazard level set at 1.0 (except for mercury which is set at 0.33).
NA= Information not available from IRIS or HEAST.
i=USEPA Integrated Risk Information System (IRIS), accessed December, 1995.
h= USEPA Health Effects Assessment Summary Tables (HEAST), FY 1995.
w= Value withdrawn.
e=EPA-NCEA Regional Support provisional value.

Witco Corporation

Review of USEPA'S Derivation of Provisional Thiocyanate RfD Stauffer/LeMoyne Site

28 February 1996

Environmental Resources Management, Inc.
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Witco submitted comments on the Stauffer/LeMoyne Site Draft Feasibility Study to USEPA Region 4 on 16 December 1994, including an alternative toxicity value for thiocyanate. In the Risk Assessment completed for the site, thiocyanate was the largest risk contributor. On 16 December 1995, J. Benante, USEPA Region 4, submitted a letter to Mr. James Baller, which rejected Witco's alternative toxicity value. After consideration of the input from EPA's Superfund Technical Support Center, which proposed a more stringent toxicity value, EPA decided to retain the value reported in the Record of Decision for Operable Unit #1 - Groundwater.

Witco is providing this document to reach a conclusion with EPA regarding thiocyanate toxicity. We believe that the experimental studies which EPA relied upon are flawed in such a manner that they do not meet EPA's methodology for developing toxicity values. This is supported by an earlier EPA toxicity review that considered the same studies and which rejected these same studies which EPA's 16 December letter relied upon. Moreover, most of these studies were never designed to evaluate the toxicity of thiocyanate; in fact, a number (i.e., 14) of these publications are over 50 years old.

We have herein provided:

1) A review of standard EPA methodology in developing toxicity values;

2) A review of the problems with the studies that EPA relied upon; and

3) A presentation of data supporting an alternative toxicity value.

Our conclusion is a recommended alternative toxicity value that is still totally protective of human health. We request that EPA consider the technical merit of this alternative value as being based on the best available information.

1.0 INTRODUCTION AND BACKGROUND

Witco submitted comments on the Stauffer/LeMoyne Site Draft Feasibility Study to USEPA Region 4 on 16 December 1994, and included an alternative Reference Dose (RfD) for thiocyanate of 1.0 mg/kg-day. On 16 December 1995, J. Benante, USEPA Region 4, submitted a letter to Mr. James Baller, which rejected Witco's alternative RfD and reported that EPA's Superfund Technical Support Center (STSC) had developed a provisional RfD for thiocyanate of 0.0004 mg/kg-day (note: this value is 2,500 times lower than the value recommended by Witco in the December 1994 comments). Appended to J. Benante's letter of 14 December 1995 was an attachment with background information on which the provisional RfD was based (95-23b/07-5-95). This 15-page document (referred to as STSC #2 in this document) reviewed a number of medical/scientific articles on thiocyanate that had been published in the last 67 years. Most of these studies were never designed to evaluate the toxicity of thiocyanate; in fact, a number (i.e., 14) of these publications are over 50 years old. For example, the four studies selected as "co-principal" studies were all designed for other purposes such as hypertension evaluation, and three of the four are over 50 years old.

It is important to note that, some time earlier, the STSC, Environmental Criteria and Assessment Office (ECAO) had developed a provisional RfD for thiocyanate in a document with the same title as the one provided by J. Benante, but with a heading of "CMT1/12-01-93." The conclusion from STSC #1 was a provisional RfD of 0.02 mg/kg-day.

The purpose of this report is to critically review the documents submitted by STSC and to recommend an alternative RfD that applies appropriate conservatism, consistent with EPA policy, and is totally protective of human health.

1.1 REVIEW OF USEPA RFD METHODOLOGY

The USEPA has developed a methodology for noncarcinogenic endpoints that basically assumes that a threshold or dose level exists below which no adverse effects occur. In order to apply this threshold concept to risk assessment, the EPA has developed a methodology to develop Reference Doses (RfD) for noncarcinogenic endpoints. The RfD is developed from either human or animal studies by dividing the NOAEL by a combination of uncertainty factors (UFs) and a modifying factor (MF). Prior to the development of the RfD methodology, noncarcinogenic effects of chronic

exposure were evaluated using values called Acceptable Daily Intakes (ADIs), which were developed by dividing the no observed adverse effect level (NOAEL) by safety factors. The terminology has changed, but the methodology is essentially the same. The following are relevant definitions of the terms and UFs used in the RfD approach (USEPA):

Reference Dose: a provisional estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (for chronic RfDs).

Adverse Effect: A biochemical change, functional impairment, or pathological lesion that either singly or in combination adversely affects the performance of the whole organism, or reduces an organism's ability to respond to an additional environmental challenge.

Lowest-observed-adverse-effect-level (LOAEL): The lowest exposure level at which there are statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group.

Lowest-observed-effect-level (LOEL): The lowest exposure level at which there are statistically or biologically significant increases in frequency or severity of any effects between the exposed population and its appropriate control group. The effects that are seen at this level may or may not be considered adverse.

No-observed-adverse-effect-level (NOAEL): An exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered to be adverse. In an experiment with more than one NOAEL, the regulatory focus is primarily on the highest one, leading to the common usage of the term NOAEL to mean the highest exposure level without adverse effects.

No-observed-effect-level (NOEL): An exposure level at which there are no statistically or biologically significant increases in the frequency or severity of any effect between the exposed population and its appropriate control.

According to USEPA's Risk Assessment Guidance for Superfund (RAGS), the selection of studies regarding the potential for a contaminant to cause adverse health effects may include controlled epidemiologic investigations, clinical studies, and experimental animal studies. Further,

RAGS states "If adequate human studies (confirmed for validity and applicability) exist, these studies are given first priority in the dose response assessment and animal studies are used as supportive evidence." RAGS also states "At present, however, human data adequate to serve as the sole basis of a dose-response assessment are available for only a few chemicals." This latter point is critical to an evaluation of thiocyanate, since the studies which STSC #2 relies upon have some critical faults.

The RFD is developed in the following fashion:

$$RfD = \frac{NOAEL}{UF \times MF}$$

Uncertainty Factors:

- Intraspecies - A UF of 10 is used to account for variation in the general population and is intended to protect sensitive subpopulations (e.g., elderly, children).
- Interspecies - A UF of 10 is used when extrapolating from animals to humans. This factor is intended to account for the interspecies variability between human and other mammals.
- A UF of 10 is used when an NOAEL derived from a subchronic instead of a chronic study is used as the basis for a chronic RfD.
- A UF of 10 is used when an LOAEL is used instead of an NOAEL. This factor is intended to account for the uncertainty associated with extrapolating from LOAELs to NOAELs.

In addition to the UFs listed above, a modifying factor (MF) is applied:

- An MF ranging from >0 to 10 is included to reflect a qualitative professional assessment of additional uncertainties in the critical study and in the entire data base for the chemical not explicitly addressed by the preceding uncertainty factors. The default value for the MF is 1.

1.2 *STSC #2 PROVISIONAL RFD STUDIES*

EPA developed its provisional RfD based on four co-principal studies selected by STSC. They are:

- Dahlberg, et al., 1984, "Intake of Thiocyanate by Way of Milk and Its Possible Effect on Thyroid Function," Am. J. Clin. Nutr. 39(3): 416-20;

- Palmer, et. al., 1929a, "Clinical use of Potassium Sulfocyanate in Hypertension: a Preliminary Report of 59 Cases," New Engl., J. Med. 201: 709-714;
- Barker, et. al., 1941, "Further Experiences with Thiocyanates," J. Am. Med. Assoc., 117(19): 1591-1594; and
- Smith and Rudolf, 1928, "The Use of Sulphocyanate of Soda in High Blood Pressure," Canadian Med. Assoc. J. 19: 288-292.

A discussion of each is provided below.

1.2.1 *Review of Dalhberg*

The 1984 Dahlberg study was designed as a nutritional study, not a toxicology study. The specific explanation was that thiocyanate added to milk can help in preserving milk when refrigeration is not available. Thiocyanate has an antibacterial activity and thus increases the shelf-life of milk; however, the authors were concerned about possible thyroid effects of thiocyanate, so they designed this study. The stated reason for the concern over possible thyroid effects was a study by Barker (1936), where patients with severe hypertension were treated with 300 to 1,000 mg/day of thiocyanate, and some patients developed goiter. This study by Barker indicated that the blood level of thiocyanate should be 80 to 120 mg/l for effective hypertension treatment, and that toxic effects begin to appear at blood levels of 150 to 300 mg/l of thiocyanate. In the Dahlberg study, only one concentration of thiocyanate in milk was tested (i.e., 20 mg/L). Thirty-seven adult volunteers were involved in the study, in which each individual consumed 200 ml of milk twice a day for 3 months. This resulted in a daily dose of 8 mg of thiocyanate to each volunteer, with blood levels of thiocyanate elevated to a maximum of 7.8 mg/l in non-smokers and 10.7 mg/l in smokers. The control (i.e., background) level of thiocyanate in these two groups was 4.0 and 8.4 mg/l in non-smokers and smokers, respectively. There were no reported adverse health effects on any of the 37 volunteers and no alteration in serum concentration of any thyroid hormones.

1.2.2 *Review of Palmer*

The 1929 Palmer study involved the treatment of hypertensive patients with potassium thiocyanate with doses between 100 mg and 300 mg per day for three to four weeks. Approximately one-half of patients experienced a beneficial reduction of systolic blood pressure of 30 mm of Hg. Also, no toxic effects were reported from the treatment regime used in this study. All of the patients were suffering from essential hypertension. The daily dose of their treatment would be 195 mg/day or approximately 2.8-3.5 mg/kg-day (note that this is different than the

"LOAEL" reported by STSC of 1.5 -1.7 mg/kg-day). The STSC apparently determined that, since these hypertensive patients experienced a significant drop in blood pressure, if that same drop occurred in normotensive individuals it would be an adverse effect, and therefore STSC determined this dose to be an LOAEL. As reported, no normotensive patients were tested, and no adverse effects were reported; therefore, this dose could logically be declared an NOAEL.

1.2.3 *Review of Barker*

The 1941 Barker study was a follow-up to their 1936 study and focused on 246 patients that had been on thiocyanate therapy for periods of 2 to 10 years. The goal of the therapy was to produce blood levels of greater than 60 mg/l of thiocyanate after several weeks of therapy and thereby reduce hypertension. In order to achieve that blood level of thiocyanate, patients initially received 300 mg of potassium thiocyanate, which if necessary was doubled in the second or third week of therapy. The authors state that none of the patients experienced severe intoxication unless the blood thiocyanate level was above 200 mg/l, and only 18 of the total of 246 patients experienced adverse toxic effects. Since the therapeutic decisions were based on blood thiocyanate levels, and since the administered doses were modified to give blood levels in the 80 to 120 mg/l range, it is impossible to accurately calculate an administered dose for any patient. The STSC report states that the LOAEL for this study was 2.6 mg/kg-day, yet this is calculated from only the initial dose and cannot be documented as the actually administered dose. The Barker study states that "Throughout the whole course of cyanate administration the dosage may be constantly varied - either increased or decreased - depending on the patients' symptoms, blood pressure and blood level concentration." Furthermore, the STSC report states that the adverse effects in this study were thyroid toxicity, yet the study only says that these effects occur occasionally, and only at high blood levels of thiocyanate.

1.2.4 *Review of Smith and Rudolf*

The 1928 Smith and Rudolf study indicated that six non-hypertensive persons were given 1,000 mg/day of sodium thiocyanate (i.e., 716 mg/day thiocyanate ion), and all had a fall in systolic blood pressure of from 15 to 30 mm of Hg in one week. Note: this is a relatively small reduction in blood pressure, and there was no control group (i.e., placebo) to determine the effect of the study alone. The STSC study incorrectly states that the thiocyanate dose level in this study was 3.3 mg/kg-day, when it is actually 10 mg/kg-day (716 mg/kg /70 kg body weight = 10 mg/kg-day).

1.2.5 *Summary Review of STSC #2 Principal Studies*

After an evaluation of these and the other studies, the STSC selected the Dahlberg study to develop the provisional RfD for thiocyanate. The STSC stated that the NOAEL for that study was 0.11 mg/kg-day (the NOAEL was calculated by dividing the 8 mg/day of thiocyanate by 70 kg of body weight; however, it should be pointed out that 28 of the 36 volunteers were females and would be expected to have a lower body weight), and dividing by a combined uncertainty factor of 300 (10 to account for extrapolation from a subchronic study, 10 for human variability and 3 for database deficiencies). Since this study only considered one dose and no toxic effects were reported, it fails to meet the criteria for development of a true NOAEL. This fact is even more obvious when reviewing the thiocyanate blood levels examined. In the Dahlberg study, the maximum blood level was 10 mg/L, the remaining studies seem to agree that toxicity does not develop unless the blood level exceeds 100 to 200 mg/L. The earlier STSC #1 also reviewed the Dahlberg study and concluded, "The report by Dahlberg et al. (1984), in which humans were exposed to low levels of sodium thiocyanate in milk was also not used for derivation of the provisional RfD, because the dose level used was 500-fold lower than the LOAELs observed in animal studies and use of such low freestanding NOAEL may have been overprotective."

1.3 *SUMMARY OF HUMAN STUDIES FOR RFD DEVELOPMENT*

Of the four studies listed above, the Dahlberg study is totally inappropriate for RfD development, because the dose is too low, higher doses were not used, and the study was not a chronic study. The Palmer study is also inappropriate, since the only "toxic effect" is reduction of blood pressure in hypertensive patients; however, the study may indicate a possible NOAEL of 2.8 - 3.5 mg/kg-day. The 1941 Barker study is also totally inappropriate for development of an RfD, because the dose used is impossible to determine. The Smith and Rudolf study is also inappropriate, since only six normortensive patients were used; however it may indicate an LOAEL of 10 mg/kg-day. A logical conclusion is that none of the human studies should be used to develop an RfD, because they do not meet the requirements EPA has developed for studies as referenced on Page 2. The earlier STSC document (STSC #1) referred to the clinical studies with thiocyanate and included the following conclusion that exact dose administration was unmonitored: "However, because of variability in the rate of excretion of thiocyanate between patients (Domzalski et al., 1953), blood levels, rather than dose, were typically used in clinical practice to monitor drug levels. Doses were titrated as necessary to maintain therapeutic blood levels and avoid toxic

ORIGINAL (Red)

effects." This fact was ignored in the current STSC (#2) document. Even if one assumes that any of these studies contains usable information, the studies fail the criterion for a well-controlled human study and so should only be used as supporting information.

2.0 ALTERNATIVE RFD CONSIDERATIONS

Both STSC #1 and #2 reviewed animal studies but concluded that the available human studies were useful. As discussed above, and in EPA's earlier review (STSC #1), the available human studies have serious flaws. ERM has reviewed the available animal data as well as other EPA studies, toxicology, and policies. This section presents those findings which show that a different RfD is indicated.

2.1 REVIEW OF THE ANIMAL STUDIES

The only chronic study identified was published in 1989 by Lijinsky and Kovatch. Male and female rats received sodium thiocyanate in their drinking water (i.e., 20 ml/day of 0.32 % sodium thiocyanate) 5 days per week for their lifetimes. The NOAEL for thiocyanate has been estimated at approximately 75 mg/kg-day for males and 128 mg/kg-day for females. The authors report no noticeable adverse effects or tumor development for the duration of the study. Since only one dose was used, this study also fails to meet the criteria of true NOAEL; however, the authors indicate that the daily dose received by the female animals was about one-third of the reported LD_{50} for sodium thiocyanate, which is generally as high a dose as that which can be administered chronically without mortality. There was only one dose tested, so no LOAEL was determined. While this study used only one dose level, it is the only available chronic study and can ideally be used to develop an RfD. The study reported that no adverse effects were noticeable following the lifetime administration of thiocyanate, and the authors have indicated that both gross pathology and complete histopathology were conducted (personal communication, W. Lijinsky). Therefore, the dose of 128 mg/kg-day would be the NOAEL for development of an RfD.

Several subchronic studies were identified by the STSC, and the following table summarizes the results of these studies:

Authors	NOAEL	LOAEL
Philbrick, et al.	NA	67 mg/kg-day
Kanno et al.	NA	418 mg/kg-day
Nagasawa et al.	NA	120 mg/kg-day[a]
Nagasawa et al.	405 mg/kg-day[a]	NA
Pyska	NA	73 mg/kg-day[b]
Pyska	NA	65 mg/kg-day[b]
Pyska	65 mg/kg-day[b]	194 mg/kg-day[b]
Wolff, et al.	NA	170 mg/kg-day[a]
Rawson, et al.	NA	457 mg/kg-day[a]
Haydens, et al.	NA	NA[c]
Lindberg, et al.	NA	NA

a Differs from LOAEL developed by STSC by using actual animal weights.

b Differs from NOAEL developed by STSC by using midpoint of water consumption (i.e., 25 ml).

c Differs from STSC since there is insufficient information available in this study to determine exposure, let alone an LOAEL.

EPA methodology prefers the use of the chronic study in animals to develop an RfD. There is no reason to use a subchronic study if a quality chronic study exists and no reason to use a study with only an LOAEL, if a study with an NOAEL exists. Therefore, deriving an RfD from the chronic animal study data would indicate an RfD of 1.3 mg/kg-day as follows:

$$RfD = \frac{128 \text{ mg/kg-day}}{(10 \times 10 \times 1)^a} = 1.3 \text{ mg/kg-day}$$

a (10 for intrahuman, 10 for interhuman)

2.2 *REVIEW OF THIOCYANATE THYROID EFFECTS*

Thiocyanate has been very well studied in terms of its effects on the thyroid. It is classified as an ionic inhibitor, because it interferes with the concentration of iodine by the thyroid gland, and appears to inhibit the organification of iodine. The thyroid effects of thiocyanate are based on

the amount of thiocyanate in the blood and the amount of iodine in the diet. Certain foods (i.e., cabbage) containing certain plant glycosides may result in elevated thiocyanate blood levels, as does cigarette smoking. In portions of the world where the iodine intake is low and the intake of these plant glycosides is high, conversion to thiocyanate is thought to be involved in the incidence of endemic goiter. Current therapy with sodium nitroprusside may also result in elevated thiocyanate blood levels. The most recent pharmacology text book (Goodman and Gilman's, 1996) indicates that plasma concentrations of thiocyanate should be monitored and not allowed to exceed 100 mg/l. This reference also states that "Rarely, excessive concentrations of thiocyanate may cause hypothyroidism by inhibiting iodine uptake by the thyroid gland." Nevertheless, the thyroid effects of thiocyanate are more blood-level-related than dose-related, since the clearance by kidney is so important to the resulting blood level. It is obvious from the studies reviewed that the only legitimate toxic effect of thiocyanate exposure is its effect on the thyroid gland, by inhibiting the uptake of iodine; however, the studies clearly indicate that this effect is rapidly reversible with discontinuance of thiocyanate exposure or iodine administration. Also, the effect of any thiocyanate exposure would be reduced in the presence of an adequate iodine intake, which is the case with a normal American diet.

2.3 *REVIEW OF BLOOD LEVELS OF THIOCYANATE*

As indicated in the Barker (1941) study, background levels of thiocyanate in the blood are around 4 mg/l (and over 8 mg/L in smokers). However, as also indicated, toxic effects do not occur until the blood level is above 100 to 200 mg/l. Obviously this is the level where thiocyanate can interfere with the concentration of iodine in the thyroid gland.

2.4 *REGULATORY STATUS OF THIOCYANATE*

The only regulatory listing located for any thiocyanate salt was for ammonium thiocyanate. Ammonium thiocyanate is regulated under CERCLA as having a reportable quantity and under the Clean Water Act Program. The reportable quantity (RQ) for ammonium thiocyanate is 5,000 pounds. No chemical listed has a greater reportable quantity. Also, many other ammonium compounds have the same RQ (i.e., ammonium oxalate, ammonium chloride, ammonium carbonate, and ammonium acetate). For comparison, the RQ for soluble cyanide compounds is 10 pounds.

There is no occupational exposure level for thiocyanates in either OSHA or ACGIH. Thiocyanates are naturally produced from plant glycosides and cigarette smoke, and saliva can naturally contain up to 250 ppm of thiocyanate ion.

In man the major pathway for metabolism of cyanide ion is to convert to thiocyanate, which is readily excreted in the urine. Once the reaction forming thiocyanate occurs, it is essentially irreversible (ATSDR, 1995). One of the main treatments of cyanide intoxication is to provide additional sulfur donors (i.e., sodium thiosulfate) to enhance the conversion to thiocyanate. The most recent ATSDR Toxicology Profile for Cyanide includes a reference to ammonium thiocyanate, but the only study referenced for toxicity is the Philbrick, et. al. (1979) study on potassium thiocyanate, for which ATSDR lists 67 mg/kg-day as the LOAEL from that study.

2.5 *COMPARISON TO OTHER RFD'S*

A review of the IRIS database provides an instructive comparison to the RfD for other chemical substances. For example, the RfDs for all cyanide compounds range from 0.005 to 0.2 mg/kg-day. These values are 12 to 50 times greater than the RfD developed in STSC #2 for thiocyanate. The most recent edition of Region III's Risk-Based Concentration Table (20 October 1995) lists a new RfD for thiocyanate of 0.02 mg/kg-day, with an EPA-NCEA reference. This value is 50 times greater than the value listed in the STSC #2 document. The source of this RfD is also STSC, as provided in STSC #1. This Region III document lists risk-based concentrations for thiocyanate of 730 µg/l for tap water, 1,600 ppm for residential soil (ingestion), and 41,000 ppm for industrial soil (ingestion). Further, investigation of all of the RfDs listed in IRIS or HEAST only reveal about 50 chemicals with lower RfDs than the one developed by STSC #2 for thiocyanate.

3.0 SUMMARY AND RECOMMENDATIONS

While thiocyanate has been extensively studied in humans and animals, the studies have mostly focused on pharmacological activity and mechanisms of thyroid action. None of the human studies appear to have the necessary scientific validity to serve as the basis for an RfD. The one chronic study with thiocyanate appears to suffer from an incomplete description of the investigation for non-tumor endpoints; however, recent conversations with the author indicate that both gross observations and complete histopathologic evaluations were conducted and no adverse effects identified. The various sub-chronic animal studies all suffer from some major flaws in experimental design for a sound toxicological study, and it is doubtful if any of the studies were conducted under current Good Laboratory Practices (GLP). Accordingly, we have developed an RfD using the chronic animal study, resulting in an RfD of 1.3 mg/kg-day. This RfD is almost identical to the value identified in the Baller Hammett Report of 15 December 1994 (i.e., 1.0 mg/kg-day). This value is totally consistent with EPA methodology and protective human health.

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