UNITED STATES DISTRICT COURT
DISTRICT OF UTAH, CENTRAL DIVISION

UNITED STATES OF AMERICA

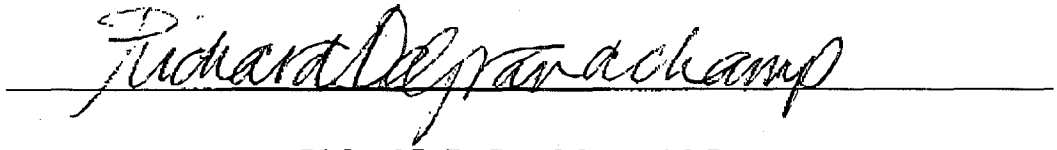
V.

MAGNESIUM CORPORATION OF AMERICA, *et al.*

EXPERT REPORT
OF
Dr. RICHARD L. DEGRANDCHAMP, Ph.D.

Regarding the Magnesium Corporation of America,
Rowley, Utah

Prepared For
U.S. Department of Justice
Environment and Natural Resources Division
Washington, D.C.



Richard L. DeGrandchamp, Ph.D.
University of Colorado/Scientia Veritas, LLP
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1 SUMMARY OF OPINIONS

This report presents my toxicological evaluation and risk assessment for exposures to uncontrolled releases of toxic contaminants at the Rowley, Utah, Facility (Facility). The U.S. Department of Justice (DOJ) requested that I evaluate the toxicity of all chemicals used or produced as byproducts at the Facility and determine whether any potential exposures could pose a threat to human health for any individual.

After careful review of all pertinent sampling data, I have concluded that dioxins and hexachlorobenzene (HCB) are the two primary contaminants of concern that pose health threats following *chronic* exposures. I have concluded the following four groups are exposed to these toxic contaminants:

- Past and current Facility employees who have worked in either the electrolytic or melt reactor buildings;
- Facility workers' families;
- The commercial laundry service employees who unknowingly wash contaminated coveralls; and
- Future workers exposed to remote areas of the Facility.

My toxicological evaluation and assessment of potential threats to human health lead to the conclusion that past, current, and future exposures could pose a range of health threats to some or all of the above targeted populations. My conclusions are based on the following findings:

- Total dioxin TEQ body burden levels measured in Facility workers are well above U.S background levels (see Section 7.1.1);
- The maximum dioxin TEQ body burden measured in Facility workers is about eight times higher than the average background body burden, posing an exceptionally high cancer risk of between 6×10^{-3} and 2×10^{-2} (or between 6 in 1,000 and 2 in 100) (see Section 7.1.1);
- In addition to cancer risk, the noncancer health threat posed by the measured dioxin body burdens is very high, and the estimated dioxin exposure for the Facility worker with the highest dioxin burden is 16 times higher than a recommended safe exposure level (see Section 7.1.2);
- Like dioxin, HCB body burdens in Facility workers are very high (they should be zero)—and the estimated cancer risk for the maximum HCB body burden is approximately 2×10^{-4} (or 2 in 10,000) (see Section 7.1.3);

- HCB poses a noncancer health hazard to Facility workers—the maximum Facility receptor body burden was 6788 ppb lipid-adjusted, which is approximately 38 times the safe level of 180 ppb lipid-adjusted (see Section 7.1.4);

- Facility coveralls are highly contaminated with dioxin and HCB, and it is probable that workers have been contaminating their vehicles and homes (see Section 8.0);

- Families of Facility workers may inadvertently be exposed to dioxins and HCB as a result of take-home contamination (see Section 8.0);

- Workers for the contract off-site laundry service that cleans Facility workers' coveralls are unknowingly being exposed to toxic contamination (see Section 8.0); and

- Contamination in some remote areas could pose a cancer risk greater than 1×10^{-3} if future workers are exposed to the areas (see Section 9.4).

It is worth noting that that while workers are acutely aware of the frequent releases of chlorine gas because of its characteristic pungent odor and its greenish cloud, the presence of dioxins and HCB cannot be detected with their senses, so exposures to these toxic compounds occur without warning. Dioxins and HCB are widespread in some areas of the Facility, and workers unknowingly have come in contact with them on a daily basis for up to 25 to 30 years. Whereas workers can quickly don their respirators when they detect a chlorine gas release, exposures to dioxins and HCB are insidious and go completely unnoticed. Furthermore, while physical symptoms of chlorine gas exposure appear immediately, exposure to dioxin and HCB can go completely unnoticed until cancer develops, which can be more than 10 years after the initial exposure. From a toxicological perspective, chronic dioxin and HCB exposures are a thousand times more toxic than chlorine exposures for Facility workers.

Lastly, I have disregarded the toxic effects of PCBs, which are also produced as byproducts and released during production. Based on the relative concentrations that have been measured at the Facility, I have concluded that PCB exposures do not merit the same attention that is necessary for dioxin and HCB exposures.

2 PRIMARY SOURCES OF INFORMATION AND DATA

I relied upon the following sources of information to form my opinion:

- Observations, and photographs taken during a personal tour of the Facility 26 and 27 July 2006;

- Observations and photographs taken during a personal tour of the Facility 20 September 2006;

- Sampling notes and photographs taken by Emilio Llamozas during coverall sampling event 8 and 9 November 2006 (US-SP-080196-367);

- 20 September 2006 URS surface wipe sampling results;

- *Baseline Risk Assessment*, Draft, MWH 2003 (SET00459);

- *Health Hazard Evaluation Report*, NIOSH HETA, #2004-169-2982, U.S. Magnesium, Rowley, Utah, October 2005 (US-SP-077528-618);

- *Occupational Risk Assessment Report For Hexachlorobenzene*, MWH, 2003. (SET00459-762);

- NEIC Report 2006 (US-SP-078427);

- URS Validated Coverall Data (US-SP-080146-95);

- 24 January 2007 telephone conference with Drs. Don Patterson and Wayman Turner, CDC laboratories, regarding HCD data units;

- *Current Waste Pond Sediment Sampling Report*, MWH 2005 (US-SP-011589-671);

- Depositions taken from Facility Management; Mr. Gines, Mr. Francom, Mr. Tripp, Mr. Silva;

- *Report to Congress on Workers' Home Contamination Conducted Under The Workers' Family Protection* (Act 29 U.S.C 671a), U.S. Department of Health and Human Services, Public Health Service, Center For Disease Control and Prevention, National Institutes of Occupational Safety and Health (1995);

- CDC's National Center for Health Statistics (NCHS) database of the *National Health and Nutrition Examination Survey (NHANES), Second Report* conducted during 2001-2002;

- *Health Risks from Dioxin and Related Compounds Evaluation of the EPA Reassessment*, National Research Council, National Academy of Sciences (2006);

- *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A),* EPA/540/1-89/002, U.S. EPA. 1989 (Office of Emergency and Remedial Response: Washington, DC).

- Integrated Risk Information System (IRIS) Chemical Files 2006, online at http://www.epa.gov/iris/index.html;

- *Toxicological Profile for Hexachlorobenzene,* U.S. Department of Health and Human Services Public Health Service, Agency for Toxic Substances and Disease Registry, September 2002;

- *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*, NAS Review Draft, EPA/600/P-00/001Cb, December 2003; and

- *Assessment of the Health Risk of Dioxins: Re-Evaluation of the Tolerable Daily Intake (TDI),* World Health Organization Consultation, 25-29 May 1998, Geneva, Switzerland.

3 QUALIFICATIONS

3.1 Education and Scientific Research

I received a B.S. in biochemistry from Eastern Michigan University in 1978 and a Ph.D. in toxicology from the University of Michigan in 1986. After receiving my Ph.D., I received further training in toxicology/pharmacology as a postdoctoral fellow at Rutgers University, Department of Pharmacology and Toxicology, and also held a joint appointment as a research associate at Cornell University Department of Pharmacology in the School of Medicine, from 1986 to 1988. I was awarded a National Institutes of Health Fellowship in Physiology at the University of Colorado School of Medicine Department of Physiology from 1988 to 1991, where I conducted toxicology experiments and directed the scientific training of numerous medical and graduate students in medical, environmental, and industrial toxicology.

3.2 Faculty Positions and Teaching Experience

I am currently an Adjoint Assistant Professor, University of Colorado Health Sciences Center, School of Pharmacy, Department of Molecular Toxicology and Environmental Health, Denver, Colorado, where I teach toxicology, risk assessment, and statistics to physicians and doctoral candidates in toxicology.

I also have a faculty appointment in the graduate program in the Department of Environmental Sciences in the University of Colorado Health Sciences Center, where I am the instructor for the Risk Assessment course, which is a required course for a graduate degree. I also lecture on basic toxicology topics and give a special lecture on the toxicity of dioxins and polychlorinated biphenyls in the Toxicology course.

I was also on the teaching faculty at the Naval Civil Engineer Corps Officers School (CECOS), Port Hueneme, California, where I was responsible for developing a three-day course in risk assessment and risk management for risk assessors/managers in the Department of Defense.

I have also taught specialty courses in toxicology and statistics for the U.S. Navy Bureau of Medicine, Environmental Health Center in Norfolk, Virginia.

Over my career, I have taught more than 600 students in my toxicology and risk assessment courses.

3.3 Professional Experience

I am President and Principal Toxicologist of Scientia Veritas, L.L.P., a consulting company that specializes in toxicology, risk assessment and management, industrial hygiene, and occupational medicine. I have over 27 years of professional experience as a toxicologist and have conducted or reviewed over 300 human health risk assessments and toxicological evaluations. My *curriculum vitae* is presented in Appendix A.

3.4 Compensation and Other Testimony

My billing rate is $150.00 per hour for litigation support and $170.00 per hour for court testimony and depositions.

4 INTRODUCTION

At the request of the United States Department of Justice, I have prepared this expert report based on my toxicological evaluation/risk assessment for the Rowley Facility (Facility; previously known as MagCorp and Magnesium Corporation of America) in Rowley, Tooele County, Utah. The goal of my investigation was two-fold. First, I identified all those individuals or groups of individuals who could be exposed to the toxic contaminants released by the Facility. Second, I evaluated whether any of the Facility's contaminants could pose a threat to the health of individuals exposed to those contaminants.

The Facility produces magnesium using an anhydrous electrolytic production process (National Enforcement Investigative Center; NEIC 2006). The primary raw material used in the production process

is concentrated brine from the Great Salt Lake. The Facility is located approximately 40 miles west of Salt Lake City, Utah, about 15 miles north of Interstate 80, exit 77 (Rowley). The Facility occupies 4,525 acres in a rural desert area in Tooele County, west of the Great Salt Lake. Photograph 1 (Appendix B) shows a 3 August 2002 satellite image of the Facility (adapted from NEIC 2006). The Facility comprises numerous buildings and structures that fall within the orange rectangle in Photograph 1 (Appendix B). For purposes of my toxicological investigation/risk assessment, I refer to this area as the plant operations/management area, where my main focus is worker health. My primary focus in the plant areas are the workers who labor in or are otherwise exposed to contaminants within the electrolytic and melt reactor buildings. The contaminants in these two buildings are in the form of dusts and I will refer to the dusts as either melt reactor dust (in the melt reactor building) or anode dust (in the electrolytic building).

I refer to all remaining property outside the designated plant area as "the remote areas." The remote areas include the sewage pond; a landfill; and other areas used for waste materials, such as smut piles, barium sulfate pile, and gypsum pile. An open channel system consisting of five earthen, open-air ditches conveys Facility liquid wastes from the processing area to an earthen, open-air, 400-acre surface impoundment and the old waste pond. Four of the five ditches are oriented in the south-to-north direction where they intersect the main ditch. The main ditch is oriented from west to east and extends to the surface impoundment.

The Facility specializes in the manufacture and supply of magnesium ingots; magnesium recycling services; and chemical byproducts chlorine, ferric chloride, ferrous chloride, calcium chloride, and hydrochloric acid (NIOSH 2005, NEIC 2006). Approximately 400 workers are employed by the Facility (NIOSH 2005), laboring in a variety of millwork, chemical processing, and foundry operations where magnesium has been produced since 1972.

There are two points in the manufacturing process where toxic chlorinated hydrocarbons (CHCs), including dioxins, furans, PCBs (collectively called "dioxin-like compounds" or simply "dioxins") and HCB are formed *de novo* as byproducts in high concentrations (NEIC 2003). The first is in the melt reactor, where a coal coke mixture (used to scavenge oxygen) is added to the spray dry powder feedstock of magnesium chloride. Two feed systems and four melt/reactor trains constitute the primary production process. Initial chlorination using chlorine gas occurs in the refractory-lined, electrically heated melt cells. Product from the melt cells flows through a covered, refractory-lined laundering system and associated reactor cells for final chlorination. Engineering controls for off-gas products from the melt/reactor cells include local exhaust ventilation in the form of a dedicated off-gas extraction system.

Since there are many uncontrolled releases of chlorinated hydrocarbons (CHCs) within the melt reactor, any Facility employees who perform work in this building were a focus of my investigation.

The second point at which dioxins and HCB are formed is in the electrolytic cells (NEIC 2003). $MgCl_2$ salt is transported in vacuum trucks to the electrolytic cells for final separation of magnesium from chloride. Two electrolytic cell lines are currently in operation. The cells are refractory-lined steel wells that contain the molten $MgCl_2$. Two electrodes—a positively charged graphite anode and a negatively charged steel cathode—separate the magnesium and chlorine. The cell bath, or electrolyte, consists of 10% to 20% $MgCl_2$ and 25% to 40% sodium chloride. When direct current passes through the molten electrolyte, magnesium ions move toward the negatively charged cathode and deposit magnesium metal, while chloride ions move toward the positively charged anode and form chlorine gas that bubbles at the anode surface. To replenish the $MgCl_2$ within the reactor building, melted $MgCl_2$ is added to the cells four times per day. A sample from each cell is analyzed each day to determine the amount of $MgCl_2$ each cell will require over 24 hours. Molten magnesium metal is removed from cells twice per day by vacuum suction into a mobile pressure vessel, which is then transported and discharged into the cast house crucibles. The source of carbon, which is necessary to form dioxins and HCB, is the graphite anode. As dioxins and HCB are formed, the anode degrades, giving rise to the anode dust. Releases of this dust into the working environment can occur at many points from the anode header to the ultimate destination at the grizzly boxes, where it is physically removed with a shovel; the distance from the anode header to the grizzly boxes is approximately 200 to 300 feet. Because the composition of the anode dust does not change appreciably along this traverse, any worker exposed at any point will come into direct contact with concentrated dioxins and furans. I present my toxicological findings on the Facility Workers in Section 6.

After evaluating the Facility workers, I shifted my focus to individuals who could be exposed to contaminants outside the Facility. This would involve anyone who comes in contact with either the contaminated Facility workers themselves or with their contaminated work clothes. Take-home contamination is a major concern because children and women are more sensitive to the toxic effects of dioxins and HCB. Take-home contamination is inadvertently carried to workers' vehicles and homes, where their families are exposed. The vast majority of workers do not shower or bathe before they leave the Facility plant, and some may still take their work clothes home to be laundered by their wives. In addition to the Facility workers' families, the Facility has retained G&K Services to clean the workers' coveralls. The high levels of contaminants on workers' coveralls could pose a health threat to the G&K

employees who handle the Facility coveralls. My evaluation of off-Facility exposures is presented in Section 8.

After evaluating take-home contamination, I shifted my attention to the remote areas where dioxin and HCB have been detected at very high concentrations. I used the detected concentrations to conduct a human health risk assessment and focused on future workers. According to EPA risk assessment guidance and good scientific practice, all human heath risk assessments must evaluate risks to both current workers and any workers who may come in contact with the remote areas. The importance of evaluating future exposures to different workers cannot be overstated. Once the Facility is sold, the current Facility operator can no longer exert control over the new workers by telling those new workers where they can work and what areas they should avoid. In the more than 300 risk assessments with which I have been involved, future exposures have been shown to be the predominant concern. The persistent nature of the contaminants in the remote areas makes it even more important that future workers be evaluated. This is because dioxins are highly resistant to degradation and will persist at current levels for many decades; in some areas, the dioxins may still be present at high levels 50 to 100 years from now. During this time, it is probable the property use may change.

The wastes streams that ultimately exit the production areas eventually end up contaminating several distinct remote areas of the property. From a human health risk standpoint, the most important areas are the main and central waste ditches, where I have estimated the cancer risk to be in excess of 1×10^{-3}—or, 1 in 1,000. The 400- and 1,200-acre waste ponds also pose elevated risk to future workers. Photograph 2 shows the general location and geological features of these two areas (note the thick gooey sludge residue in the bottom of the waste pond, which was revealed only after I scraped the surface with my boot). The results of my human heath risk assessment for the remote sites are presented in Section 9.

5 DIOXIN AND HCB TOXICITY

The toxicity of dioxin and HCB has been extensively studied in both humans and laboratory animals. Dioxin and HCB produce similar toxic effects, and recent studies suggest that they may produce these effects through a common mechanism. If so, it is important to stress that, although toxicologists conventionally discuss each chemical separately, the resulting health effects associated with simultaneous exposure for Facility workers to both these chemicals will be additive.

The primary toxic effect produced by both dioxin and HCB that is of most concern to toxicologists is cancer. Although the cancer potency of dioxin is still under investigation, the provisional cancer potency factor (which describes the ability of dioxin to produce a tumor) is the highest of any chemical EPA has evaluated. Dioxin has been shown to produce cancer at far lower concentrations than any of the more than 600 other chemicals EPA has studied and for which EPA has developed cancer potency values. Not only is dioxin the most potent carcinogen EPA has ever studied, but the current cancer potency of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD, the parent congener for the family of dioxins discussed below) is approximately 100 times greater than the second-most carcinogenic chemical (diethylstilbestrol) and 1000 times more carcinogenic than the third-most carcinogenic (benzidine).

Like dioxin, the major concern with HCB exposure is cancer. Although the cancer potency is significantly lower than that for dioxin, HCB concentrations detected at the Facility are significantly higher than detected dioxin concentrations.

Dioxin and HCB also produce noncancer toxic effects. The most sensitive toxic effects (the toxic effects that are produced with the smallest amount of chemical) occur in the reproductive and the immune systems.

Due to the sheer volume of published studies on dioxin and HCB, U.S DOJ requested that I conduct an independent and thorough review. For the sake of brevity, I will summarize my findings in the subsequent sections.

5.1 Hexachlorobenzene Toxicity

HCB is readily absorbed from the gastrointestinal tract after ingestion. One of the major complications of exposure is that, after it is absorbed into the body, HCB accumulates in lipid-rich (fat) tissues. It can also be transferred to the fetus across the placenta and via mother's milk in the newborn (Ando *et al.* 1985). The following sections summarize the toxic effects on the immune system and the liver, as well as HCB's ability to produce cancer.

5.1.1 Immune System

The immune system is a complex, highly regulated system that defends against foreign invaders such as bacteria, viruses, parasites, and tumor cells that can develop into cancers. It also recognizes and avoids

reacting against the body's own cells and tissues. Damage to the immune system can lead to allergies, inflammation, autoimmune diseases, and increased susceptibility to infection and cancer. If the immune system is suppressed, an individual is susceptible to infectious diseases and to cancer. In contrast, an over-active immune system can lead to allergies, inflammation, and autoimmune diseases.

HCB damages the immune system. The immunotoxic effects that result from HCB exposure are well known to toxicologists. Numerous studies have shown that there are two different types of HCB-associated immunopathological damage that can lead to (1) cancer or (2) non-carcinogenic autoimmune effects. The first type of immunotoxic response results in malfunctioning of the immune system, which can lead to decreased resistance to infections or development of tumors, while the second type involves autoimmunity, in which a person's own altered immune system attacks the body.

In my evaluation of Facility workers, I identified immunotoxicity as the most sensitive toxic effect. An occupational study conducted by Queiroz *et al.* (1997) revealed that 51 Brazilian workers exposed to HCB for up to 25 years—until the plant was shut down by "judicial order" due to lack of safety measures—had significant functional impairment of their immune systems. Notably, these pathological changes occurred in workers who had blood HCB levels as low as 1 part per billion (ppb). As discussed in subsequent sections, recent measurements show that many Facility workers have HCB blood levels that exceed this low level.

More recently, Volker *et al.* (2001) examined German medical patients who presented with a variety of acute symptoms (mainly, lack of concentration, exhaustion, and common cold) and who had been occupationally exposed for at least 6 months to HCB and other HCHs. Of these patients, 82% complained of a lack of concentration; 80% complained of rapid exhaustion; 50%, frequent common cold diseases; and 14%, insomnia. HCB was strongly correlated with a specific and important type of immunotoxic response. According to the authors, *"The most prominent finding was a strong negative association between HCB and IFN-γ blood levels."* That is, they detected a strong, statistically significant association between increasing blood levels of HCB and decreasing blood levels of interferon-γ (IFN-γ). This finding indicates that HCB has a significant impact on Th1 lymphocytes, which produce IFN-γ. The significance of decreased IFN-γ levels is that the body's normal defenses against viral and bacterial infections are impaired. Additionally, decreased IFN-γ levels indicate compromised immunosurveillance, which is the body's defense mechanism guarding against the development of tumors; compromising of the body's immunosurveillance system can lead to cancer. It has been well

established that both natural killer (NK) and T cells secrete IFN-γ, initiating a potent antitumor response in which tumors are destroyed (Paul 1993).

Loose *et al.* (1981) has shown that the HCB-exposed animals have immune systems that are compromised to the point they cannot attack tumors and that this impairment is specific for HCB and not for organochlorine compounds in general. Indeed, they stated, "…*that, animals exposed to HCB, but not to PCB, had a profound decrease in their resistance to a challenge tumor cell implant which was related to a select alteration in tumor cell killing."* The study showed spleens (part of the immune system) from HCB-treated mice were significantly suppressed and could not kill developing tumors. These findings of reduced *immunosurveillance* are supported by the results of Van Loveren *et al.* (1990).

5.1.2 Liver Toxicity

The toxic effects of HCB in humans have been well documented due to epidemics and inadvertent exposures. One of the earliest mass poisonings occurred in Turkey during the years 1955 to 1959, and numerous toxic effects were observed among the thousands of people who ate bread contaminated with HCB (Ferioli 1987; Clayton and Clayton 1993-1994; *Goomen et al.* 1986). With high levels of chronic HCB exposure, the primary target organ is the liver, which undergoes direct and severe pathological changes. Liver damage triggers toxic reactions in other parts of the body. One of the prominent clinical symptoms seen in HCB poisoning is porphyria cutanea tarda, which is a hallmark of HCB exposure. PCT is classified as a "porphyria," a group of metabolic disorders caused by disruption in the heme biosynthetic pathway, which occurs primarily in the liver.

In some cases, liver damage caused by HCB exposure appears to progress to cancer.

5.1.3 Carcinogenicity

Axelson (1986) conducted a review of the existing peered-review literature and reported a relationship between human exposure to HCB and increased cancer incidence in the liver. Some studies reported links between porphyria and cancer. Observations on porphyria and liver tumors included increased frequency of liver cancer in males manifesting porphyria cutanea tarda and cirrhosis. Axelson reported that lymphoma and porphyria cutanea tarda occurred together in at least five cases.

There is convincing evidence that HCB is a carcinogen based on the results of well-conducted studies on three different species of laboratory animals. Therefore, U.S. EPA has classified HCB as B2, or probable

human carcinogen (2006) (Erturk *et al.* 1986; Cabral *et al.* 1977; Smith and Cabral 1980). The studies show HCB produces tumors in several organs, including the liver, bile duct, and thyroid gland. Based on these studies, there is little doubt regarding the carcinogenic potency of HCB. Smith and Cabral (1980) reported that the incidence of cancer was 100% in their studies, which means that every animal exposed to HCB developed tumors.

Like U.S. EPA, other scientific organizations have identified HCB as a possible carcinogen. The International Agency for Research on Cancer (IARC 1972) has determined that HCB is a B2 carcinogen or *Possibly Carcinogenic to Humans*; the American Conference of Governmental Industrial Hygienists (ACGIH) has classified HCB as an A2 carcinogen, or *Suspected Human Carcinogen* (1995-96); and the U.S. Department of Health and Human Services (DHHS), National Toxicology Program (NTP), has determined that HCB is *Reasonably Anticipated to be a Human Carcinogen* (2001).

Few human studies have been conducted. One investigation by Grimalt *et al*. (1994) looked at the mortality and cancer incidence among village residents in Flix, Spain, near a factory where the measured HCB levels in air and in the blood serum of the residents were unusually high. The average 24-hour air level of HCB in the village was 35-micrograms/cubic meter, and the average serum HCB level in 21 village residents was 26 ppb. An increased level of mortality was noted due to cancers of unknown origin. There was also an increased incidence of cancer in the thyroid gland and cancers in the brain among men, some of whom worked in the factory.

5.1.4 Highly Sensitive Individuals

Women of childbearing age, infants, and small children are more sensitive to HCB toxicity compared with men. This would include female Facility workers and spouses of workers (exposed via take-home contamination). These women should avoid HCB exposure because a fetus or newborn can be exposed *in utero* and/or through breast milk. Ninety five percent of children in the Turkey epidemic who developed Pembe yara (or pink sore) died. It should be noted that, once HCB is absorbed into the body, it is eliminated slowly; it takes approximately 15 years to eliminate HCB from the body once HCB exposure ceases. This means that women who are not currently pregnant could accumulate HCB in fat stores for more than a decade and still affect the health of a fetus in a later pregnancy.

5.2 Dioxin Toxicity

In this report, I use the term "dioxin" to refer not to a single chemical but to a group of compounds that share common toxic properties. In this report "dioxin" actually refers to a group of 17 compounds (sometimes referred to as dioxin-like congeners) that are structurally similar; act through the same toxic mechanism; and, ultimately, produce similar toxic effects. Dioxin-like compounds include seven individual polychlorinated dibenzodioxin congeners (out of a total of 75 congeners) and 10 polychlorinated dibenzofurans. These dioxin-like congeners are a small fraction of the total number of 135 dioxin and furan congeners.

Due to the large number of dioxin-like compounds (which can make studies unwieldy), toxicologists have developed a scheme to simplify toxicological assessments. Instead of independently deriving a toxicity value for each dioxin compound, the toxicity value for each dioxin-like compound is based on its relative toxicity compared with TCDD, which is the parent compound of the group because it has the greatest toxic potency. Based on these comparisons, each of the 17 dioxin compounds has been assigned a specific Toxic Equivalency Factor (TEF) that represents how much less toxic it is than TCDD. In other words, TCDD is considered the parent compound of the dioxin group and has a TEF of 1.0. All other dioxin-like compounds have been assigned TEF values that range from 1.0 to 0.0003. For example, the toxicity of 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) is one-tenth the toxicity of TCDD, so it has been assigned a TEF of 0.1. The total dioxin toxicity equivalence quotient (total dioxin TEQ) describes the total amount of dioxins detected in a sample. The total dioxin TEQ is calculated by multiplying the concentration of each of the 17 detected dioxin-like compounds by their respective TEF values, then summing each product. For example, if 2,3,7,8-TCDF (which has a TEF of 0.1) is detected in a sample at a concentration of 100 parts-per-billion (ppb), the total TCDD equivalent concentration of 2,3,7,8-TCDF would be 10 ppb (100 ppb multiplied by the TEF of 0.1). Likewise, this calculation would be repeated for each dioxin-like compound, and the17 concentrations would be summed to calculate the total dioxin TEQ.

Several TEF schemes have been developed over the years, making dioxin studies somewhat complicated. However, if the scheme used in the study is clearly stated, the total dioxin TEQ can be calculated from the original detected concentrations and the referenced TEQ scheme. For this study, I had to evaluate many laboratory reports from different laboratories, which reported the total dioxin TEQ based on different TEF schemes. For example, one laboratory still uses the ITE Toxicity Equivalency Factor (I-TEF) scheme that was developed nearly 20 years ago (NATO/CCMS 1988). Toxicologists typically use the most up-to-date

TEF scheme developed by the World Health Organization (WHO). (WHO is a diverse international scientific agency represented by 192 Member States through the World Health Assembly that aims to reduce excess mortality, morbidity, and disability around the globe.) WHO updates the TEF values on a regular basis by evaluating any new scientific studies that have come to light since the last TEF scheme was updated. As shown in Exhibit 1, only minor changes were made to the 1998 TEF values in WHO's latest TEF values, which were released in 2005 (suggesting WHO is relatively confident in the veracity of the TEF values). In this report, I have calculated total dioxin TEQ with both 1998 and 2005 WHO TEF values and present the 2005 total dioxin TEQ; however, results are nearly identical to the 1998 TEQ.

EXHIBIT 1
WHO-DERIVED TEF VALUES FOR DIOXIN-LIKE COMPOUNDS

COMPOUND	WHO 1998 TEF	WHO 2005 TEF*
Chlorinated dibenzo-p-dioxins		
1,2,3,7,8-PeCDD	1	1
1,2,3,4,7,8-HxCDD	0.1	0.1
1,2,3,6,7,8-HxCDD	0.1	0.1
1,2,3,7,8,9-HxCDD	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01
OCDD	**0.0001**	**0.0003**
Chlorinated dibenzofurans		
2,3,7,8-TCDF	0.1	0.1
1,2,3,7,8-PeCDF	**0.05**	**0.03**
2,3,4,7,8-PeCDF	**0.5**	**0.3**
1,2,3,4,7,8-HxCDF	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.01	0.01
OCDF	**0.0001**	**0.0003**

Source: The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-like Compounds.

* Van den Berg *et al*. *ToxSci Advance Access.* 7 July 2006

It should be noted that in addition to the 17 dioxin and furan dioxin-like compounds, there are 12 dioxin-like PCB compounds. TEF values have been developed for these PCBs, but as stated previously they are not the focus of my investigation.

5.2.1 Immune System

Dioxins target the immune system. Individuals accidentally or occupationally exposed to dioxin-like compounds have more skin and respiratory system infections (Bekesi 1979; Lu 1985; Jennings 1988; Webb 1989; Zober 1994), and middle ear infections (Chao 1997). In Germany, workers exposed to high levels of dioxin-like compounds had impaired immune responses (Tonn 1996; Ernst 1998). Children in Taiwan who were exposed to dioxin-contaminated rice oil had several functional alterations in their immune systems (Hsu 1994). The thymus gland, which is a central organ in the immune system, has been shown to undergo dramatic shrinking in young animals following dioxin exposure (McConnell 1978; Poland 1982). Dioxin also suppresses the immune system, compromising resistance to infections and cancers (Thigpen 1975; Vos 1978; Thomas 1979; Hinsdill 1980). For example, mice infected with influenza die at a higher rate if they are first exposed to a single dose of as little as 10 ng of dioxin per kg of body weight, which is a miniscule dose—far less than some Facility workers likely receive on a daily basis.

5.2.2 Diabetes

Diabetes mellitus is a group of diseases characterized by high levels of blood glucose resulting from defects in insulin production, insulin action, or both. Diabetes can be associated with numerous serious medical complications and premature death. Dioxin has been shown to interfere with insulin, alter glucose tolerance, and produce diabetes. In a study conducted by Pazderova-Vejlupkova (1981), 50% of 55 workers exposed to dioxin and evaluated 10 years after exposure were diabetic (or showed signs of pre-diabetes). In its updated report, the National Academy of Sciences (NAS), Institute of Medicine (NAS 2001), concluded that there is strong evidence of an association between exposure to dioxin and Type 2 diabetes. Roegner (1991) found that veterans with blood dioxin greater than 33.3 ppb have a relative risk of 2.5 for diabetes.

5.2.3 Carcinogenicity

Various agencies and scientific organizations have concluded that dioxin is a potent human carcinogen. The National Toxicology Program (NTP, which is part of the U.S. Department of Health and Human

Services) upgraded the carcinogenic classification of dioxin from *Reasonably Anticipated to Be a Carcinogen* to a *Known Human Carcinogen* in 2001 (NTP 2001). The International Agency for Research on Cancer has also classified dioxin as Group 1, or Human Carcinogen. Currently, there is general consensus that dioxin should be considered a known human carcinogen. The single most controversial issue is the method EPA used to calculate a cancer slope factor, which only applies when calculating risks for individuals who are exposed to very low levels of dioxins at or near background levels.

As discussed previously, my full evaluation of dioxin toxicity (particularly as it relates to cancer) is presented in Appendix B. In brief, I have concluded that EPA has developed a very comprehensive review of all pertinent dioxin studies, which is contained in its reassessment report titled, *"Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*." The National Academy of Sciences reviewed the EPA reassessment and suggested EPA consider some additional information and studies before finalizing the report. I agree with many of the NAS recommendations, but noted many were targeted at the EPA assumptions regarding low-dose-extrapolation. That is, in the absence of actual data, EPA based its extrapolation on a mathematical model with little supporting information. It should be noted that, in the current study, I have used important information from the EPA reassessment report that *did not* have anything to do with the low-dose model controversy. In fact, I used data and information from the report that NAS apparently agreed was correct, since NAS did not question EPA's handling of that part of the study.

In contrast to my analysis of cancer risk for the Facility workers, it was necessary to use a cancer slope factor to calculate risk for future workers because a low-dose extrapolation was necessary. Since the revised cancer slope factor of 1,000,000 for dioxins presented in EPA's reassessment report is currently under review, I concluded the correct decision is to default to the older provisional cancer slope factor that has been used in human heath risk assessments for the last decade. Accordingly, for the remote areas I have used the cancer slope factor of 150,000 to estimate dioxin risks.

5.2.4 Highly Sensitive Individuals

Like HCB, female Facility workers and employee spouses of childbearing age are also at special risk. They should avoid dioxin exposure because a fetus or newborn can be exposed *in utero* and/or via breast milk. Spouses and young children of employees should avoid exposure to any dioxin brought home by workers via contaminated skin, hair, and work clothes.

6 EPA'S RISK RANGE

Throughout this report I have narrowly focused on toxicological issues relating to human health where I have calculated cancer risk and noncancer health hazards posed by the Facilities contaminants. After calculating risks and health hazards, I have intentionally avoided making any risk management determinations or judgmental statements about my results with regard to the acceptability or non-acceptability of the health threats. I leave those determinations to EPA because the Agency is responsible for making a final determination. However, it may be helpful to put the risks and health hazards I calculate in the subsequent sections into perspective by briefly explaining EPA's risk management framework, which is described in an EPA directive (OSWER directive 9355.0-30, EPA 1991). By convention, any cancer risk below 1×10^{-6} (or 1 in 1,000,000, or 1 in 1 million) is considered insignificant or *de minimus* risk. When cancer risks fall between 1×10^{-6} and 1×10^{-4} (1 in 1,000,000 and 1 in 10,000), EPA takes into account diverse site-specific factors and makes a discretionary decision. Cancer risks higher than 1×10^{-4} typically require remediation to reduce exposures so the risks fall below 1×10^{-4}. This risk scheme is based on the following points stated in EPA directive:

- Where the cumulative carcinogenic site risk to an individual based on reasonable maximum exposure for both current and future land use is less than 10 (to the 4th power), and the non-carcinogenic hazard quotient is less than 1, action generally is not warranted unless there are adverse environmental impacts.

- A risk manager may also decide that a baseline risk level less than 10 (to the 4th power) is unacceptable due to site-specific reasons and that remedial action is warranted.

- The upper boundary of the risk range is not a discrete line at 1×10^{-4}, although EPA generally uses 1×10^{-4} in making risk management decisions. A specific risk estimate around 10^{-4} may be considered acceptable if justified based on site-specific conditions.

7 TOXICOLOGICAL EVALUATION OF THE FACILITY EMPLOYEES

Based on my toxicological evaluation of blood levels of dioxin and HCB measured in Facility workers, I have concluded the following:

- Total dioxin TEQ body burden levels measured in Facility workers are well above U.S background levels.

- The maximum dioxin TEQ body burden measured in Facility workers is about eight times higher than the average U.S. background body burden, which poses a cancer risk of between 6 x 10^{-3} and 2 x 10^{-2} (or between 6 in 1,000 and 2 in 100).

- The noncancer health threat posed by the measured dioxin body burdens is also very high.

- The estimated dioxin exposure for the Facility worker with the highest dioxin burden is 18 times higher than the safe exposure benchmark derived by WHO.

- HCB body burden levels are well above the U.S background level, which is zero.

- The estimated cancer risk for the maximum HCB body burden level in Facility workers is approximately 2 x 10^{-4} (or 2 in 10,000).

- HCB poses a noncancer health to Facility workers-the maximum Facility receptor body burden was 6790 ppb lipid-adjusted, and a safe exposure level was determined to be 180 ppb (ppb) lipid-adjusted serum;

My rationale for these findings is explained in the following sections.

7.1 Dioxin and HCB Exposure

I mainly focused on the Facility employees who work in "high-exposure areas," which are the electrolytic and melt reactor areas. This is where dioxin and HCB are primarily generated. In the melt reactor building, these toxic contaminants are formed when a coal coke mixture is added to the spray dry powder feedstock of $MgCl_2$. It is the addition of the coal coke mixture that provides the hydrocarbon source for the *de novo* production of myriad chlorinated hydrocarbons, including dioxin and HCB. The second operation area of concern is in the electrolytic department, where anode dust is generated and ultimately carried to the grizzly boxes, which are located some distance from the source. Any worker contacting this highly contaminated dust anywhere along the waste stream can be exposed to high concentrations of dioxin and HCB.

NIOSH estimates that, out of approximately 400 total Facility employees, about 52 should be considered to be in high-exposure groups (NIOSH 2005). Within this group, workers with the longest tenure would be expected to be exposed longer and have the highest body burden. This is because, once absorbed into the body, dioxins and HCB remain stored in fat tissue for many years. In fact, the elimination rate for dioxin is so slow that gradual accumulation occurs over the years.

During my toxicological evaluation, I primarily relied on the following site-specific information to form my opinion:

- Measured HCB body burden levels;
- Measured dioxin body burden levels;
- Wipe sample results for dioxin and HCB; and
- Soil, dust, and coverall samples.

Biomonitoring (which is the direct measurement of chemicals in body tissues) is the preferred toxicological method to assess the magnitude of exposures in occupational settings because it directly measures body burden. That is, the body burden represents the cumulative exposure from all three exposure routes: ingestion, inhalation, and dermal absorption. Measuring the amount of HCB in blood circumvents the limitations and uncertainty introduced with mathematical modeling of human exposures based on air, dust, and soil sampling. Once blood samples are collected and analyzed, the results can be compared with normal or expected concentrations for individuals who have had no occupational exposures. Outside this litigation, I am not aware of any biomonitoring the Facility has conducted.

The following sections analyze the available biomonitoring data. The two goals of the analysis are as follows:

1. Determine whether uncontrolled releases of dioxins and HCB have resulted in increased body burdens in Facility workers; and
2. If body burdens are increased, estimate the cancer risk and noncancer health threat associated with those body burdens.

7.1.1 Dioxin Body Burdens

Dioxin body burdens of Facility workers have been determined by NIOSH (2005). By measuring both HCB and dioxin, the NIOSH study provides the most comprehensive picture of body burden in the most highly exposed individuals in the workforce.

The analysis of the NIOSH dioxin body burden study had two components. First, it was necessary to determine whether the exposure pathway is complete for Facility workers. That is, it is necessary to first

confirm that dioxin exposures inside the plant ultimately lead to dioxin absorption into workers' bodies. The second part of the analysis involves estimating cancer risk and noncancer health hazards associated with workers' body burdens.

In determining whether the exposure pathway is complete, I evaluated background levels of dioxin in the U.S. general population. This determination is necessary because it has been well established that historical releases of dioxin in the U.S. (from uncontrolled releases from numerous industrial/manufacturing practices into the general environment) have resulted in many of our foodstuffs being contaminated with low levels of dioxin. Generally speaking, background body burden levels of dioxins are the direct result of eating food contaminated with dioxin. Moreover, people with diets rich in food with high fat content (i.e., meat, poultry, fish) tend to have higher body burdens of dioxin. In addition, because the body fat percentage tends to increase with aging, it is important to make age-adjusted comparisons. The age at which this effect is most pronounced is around 60 years of age.

Individuals with high body fat have a higher body burden of dioxin. As discussed below, the NIOSH study design carefully controlled for both diet and aging bias in dioxin measurements for the Facility group by grouping workers by age. NIOSH also determined the body mass index (BMI), which gives a *rough* approximation of body fat. However, caution should be exercised in interpreting the BMI because it does not necessarily differentiate between muscle and fat. That is, a high BMI can indicate either a muscular *or* "fatty" physique. Typically, a BMI of 20 to 25 is considered normal; the range 25 to 30 is overweight; and more than 30, obese. The problem with the BMI metric is that muscle mass and fat deposition are not taken into consideration. Because muscle is much denser than fat and takes up less space, two individuals of the same height and weight could have the same BMI, even though one may have a significantly higher percentage of body fat than the other. Thus, the BMIs presented in the NIOSH report should not be considered a "fat index."

Finally, NIOSH took into account the tenure of the Facility cohort because longer tenures should be associated with increasing dioxin body burden (if exposures are occurring). Exhibit 2 summarizes the background information for the Facility cohort.

EXHIBIT 2
AVERAGE AGE, BMI, AND TENURE FOR THE FACILITY COHORT

	COHORT	OPERATION AREAS/JOB DESCRIPTION			
		Cell Brick (n=11)	**Electrolytic (n=8)**	**Maintenance (n=6)**	**Reactor (n=5)**
Average Age (range)	50.3 (40-59)	48.2 (42-56)	54.6 (50-59)	50.5 (40-58)	47.8 (46-51)
BMI	30.4 (20.3-42.6)	31.2 (26.9-42.6)	30.0 (20.3-35.0)	30.8 (27.6-40.7)	28.4 (22.4-33.0)
Tenure at the Facility	26.9 (19.9-31.4)	26.9 (22.9-30.3)	26.4 (19.9-31.4)	25.5 (20.0-29.1)	24.4 (20.3-26.7)

There were 30 Facility Workers In the Cohort

There are several sources of dioxin background body burden data for the general population. Studies by Patterson *et al.* (2004) and Ferriby *et al.* (2006) are most applicable to the Facility cohort and present the most recent background levels of age-adjusted blood dioxin levels in the general public. (It should be noted that the authors of the Ferriby *et al.* study are *Chem Risk Inc*. employees, and *Chem Risk* is one of the Facility's consultants). The Patterson *et al.* data represent age-specific reference range levels from a combination of four studies of preexisting serum dioxin data (the analyses were performed by the Centers for Disease Control and Prevention, or CDC). Demographic data on age, race, and gender were available from a total of 588 serum dioxin samples from participants who had no known exposure to dioxin-like compounds other than exposure to background levels of dioxin in food and their general environment.

The Ferriby *et al.* study is based on CDC's National Center for Health Statistics (NCHS) database of the National Health and Nutrition Examination Survey (NHANES), which was conducted during 2001-2002. CDC conducts the ongoing NHANES study by collecting health and nutritional information on the U.S. population every two years to monitor body burdens of 116 chemicals by collecting a blood sample from each volunteer participant selected from the general public.

I chose to use the Ferriby study for the following reasons. Both the Patterson and Ferriby studies appear to be well conducted, but are based on different data sets generated from different populations. Despite

the difference in populations studied, however, both studies yield similar age-adjusted dioxin blood levels for the ages of interest for the Facility cohort, with one important exception. First, the Patterson *et al.* dioxin levels, which are presented as total dioxin TEQ, are the sum of eight dioxins; 10 furans; and four non-ortho substituted, or coplanar, PCBs. These are not the conventional dioxin-like congeners that are summed for the total dioxin-TEQ. WHO (2006) has identified seven dioxins, 10 furans, and 12 PCBs that have common dioxin-like properties, for a total of 29 dioxin-like compounds, as was evaluated in the Ferriby study. Secondly, I have concluded PCBs are not chemicals of concern for the Facility cohort (the levels measured in the Facility samples are not significant for exposures); they should not be included in the total dioxin TEQ calculations. The Ferriby study provides this specific information. Despite these deviations from conventional practices, the Patterson *et al.* study provides a rough approximation for comparison purposes. It should be noted that the Patterson study was used exclusively by NIOSH in the Agency's study of the Facility cohort (2005) because the Ferriby *et al.* study was published after NIOSH completed its study. Lastly, in contrast to the Patterson *et al.* study, the Ferriby *et al.* study provides data that can be directly compared with that of the Facility cohort. This is because NIOSH presents separate dioxin and furan data apart from PCB data, so the total dioxin TEQ based on dioxins and furans can be calculated. Likewise, Ferriby *et al.* present total dioxin TEQ for dioxins and furans apart from PCBs, so it is the preferred study for comparison purposes.

One additional point that needs to be stressed is that all three studies (the Patterson *et al.,* Ferriby *et al.,* and the NIOSH studies) focus heavily on the population *average* or *mean* concentrations as the principal point of comparison. This is in contrast to good toxicological practice in which every individual must be protected. That is, it is necessary to consider the *highest exposed individuals* within the worker population to ensure that the health of the entire group is protected. In addition to good toxicological practice, health agency and regulatory agency policies always call for protecting the highest exposed or most sensitive individuals. For example, U.S. EPA's risk assessment/management policy is based on the *Reasonable Maximum Exposed* individual. While toxicologists often have to *predict* who the reasonable maximum exposed individual is in studies based on exposure conditions, the reasonable maximum exposed individual in the Facility cohort is the Facility worker with the highest body burden. In the NIOSH study, total dioxin TEQ blood results are categorized in such a manner that the reasonable maximum exposed individual is easily identified.

Exhibit 3 presents a summary of the two dioxin background studies of the U.S. general population (Patterson *et al.* and Ferriby *et al.*) juxtaposed with the dioxin body burden levels in Facility workers. The NIOSH data were taken directly from Table 7 in the Agency's 2005 report.

EXHIBIT 3 DIOXIN BODY BURDEN: COMPARING BACKGROUND LEVELS WITH THE FACILITY COHORT [1]

	PATTERSON *et al.* [2] (BACKGROUND)	FERRIBY *et al.* [3] (BACKGROUND)	THE FACILITY COHORT [3] (NIOSH STUDY)
Age	45-49	45-59	40-59
Number Of Individuals	160	228	30
Mean Total Dioxin TEQ	16.9	19.2	38.2 [4]
Range Total Dioxin TEQ	0.8 - 55.4	3.9 - 94.9	12 - 146.7 [5]

Note: All concentrations are parts per billion (ppb).

[1] The Facility cohort data presented in NIOSH study (2005).

[2] Patterson *et al.* (2004) summed eight PCDDs, 10 PCDFs, and 4 non-ortho substituted or coplanar PCBs for a total dioxin TEQ in background population.

[3] Ferriby *et al.* (2006) and NIOSH summed 7 PCDDs and 10 PCDFs to calculate total dioxin TEQ in background population.

[4] This value is the corrected value from the NIOSH report, Table 7; a minor error was present in the NIOSH table.

[5] This range was taken from NIOSH Table 6, which may include PCBs. If so, the true concentration would be approximately 5.5 less

I reached two conclusions based on Exhibit 3. The first is that the entire Facility cohort has dioxin and furan body burdens well above background levels. For example, the average for the Facility cohort is about two times background body burdens. The second is that the individual with the highest body burden has a level that is about 7.5 times the average background body burden. Moreover, the body burden for this individual is significantly higher than the maximum in either the Patterson *et al.* or Ferriby *et al.* background population. There can be no doubt the Facility cohort has been exposed to uncontrolled releases of dioxin, and that some workers in the group have levels that are significantly elevated above background. This is corroborated by the dioxin levels detected in wipe samples (from the melt reactor surfaces), worker coverall samples, and bulk dust samples from inside the plant (discussed in subsequent sections), which show the dioxin contamination is ubiquitous inside the plant. Judging by the coverall sample results, workers come in direct contact with very high levels of dioxin in some job activities.

7.1.2 Toxicity Associated with Dioxin Body Burdens

Cancer Risk

As noted previously, I focused on the reasonable maximum exposed individual who is the worker with the maximum total dioxin TEQ body burden. For this individual, I calculate the cancer risk to be between 6×10^{-3} and 2×10^{-2} (or between 6 in 1,000 and 2 in 100), which is attributable entirely to dioxin exposures at the Facility. This Facility-related risk is exceptionally high. In fact, cancer risks this high are not often calculated in human health risk assessments. This risk is lower than the actual risk because it does not include background risks, since I intentionally deleted the background source (non-Facility related exposures). Furthermore, the method I used was directly based on actual blood levels measured in other worker cohort studies. That is, it was not necessary to use the somewhat controversial low-dose extrapolation model used by EPA in its (2003) dioxin reassessment report, which was recently critiqued in the NAS review. Additionally, I did not need to estimate body burdens based on mathematical models using exposure data (which can introduce uncertainty) because blood levels were measured directly.

The point of departure should always be based on the lowest body burdens at which an increase in cancer can be observed and measured. In the case of cancer risks, the dose of dioxin that results in a 1% increase in cancer incidence above background is termed the effective dose one-percent (ED_{01}). There are several well-designed published studies that identify the dioxin-related ED_{01} for cancer. Exhibit 4 is reproduced from the dioxin reassessment report in which EPA summarized the ED_{01} reported in the Steenland *et al*, (2001); Becher *et al.* (1998); and Ott and Zober (1996) studies.

EXHIBIT 4
SUMMARY OF ED_{01} VALUES FOR DIOXIN

Study	Model and Sex	ED_{10}	ED_{05}	ED_{01}	Unit excess risk for 1 ppt body burden above background
Steenland et. al, (2001)	power male	500 (46.4, 2.91 x 10^7)	33.9 (8.23, 1.59 x 10^4)	1.38 (0.71, 8.95)	0.0079 (0.0027, 0.0132)
	power female[1]	1315 (84.4, 4.5 x 10^8)	64.5 (12.6, 2.50 x 10^4)	1.84 (0.92, 14.9)	0.0064 (0.0022, 0.0107)
	piecewise linear male	• (92.9, •[3])	83.6 (51.8, •[3])	18.6 (11.5, 48.3)	0.00052 (0.00020, 0.00084)
	piecewise linear female[2]	•[3] (108.9, •[3])	100.7 (62.39, •[3])	23.1 (14.3, 59.8)	0.00042 (0.00016, 0.00067)
Becher et al., (1998)	power-male	120.3	41.17	5.971	0.0018
	power-female[4]	170.9	55.44	7.580	0.0014
	additive-male	192.8	93.35	18.22	0.00055
	additive-female[5]	239.1	116.2	22.75	0.00044
	multiplicative-male	258.9	144.4	32.16	0.00030
	multiplicative-female[6]	304.4	173.8	39.82	0.00024
Ott and Zober (1996)	multiplicative-male	411.7 (201.9, □)	229.0 (112.3, □)	50.9 (25.0, □)	0.00019 (0, 0.00039)
	multiplicative-female[7]	478.0 (234.4, □)	272.1 (133.4, □)	62.1 (30.5, □)	0.00015 (0, 0.00032)

Source: EPA 2003 TABLE 8-2: Total cancer risk in humans through age 75 (units are constant body burden in ppb not adjusted for lipid). Upper and lower 95% confidence limits (where available) are in parentheses after ED values.

[1] Relative risk RR proportional to (AUC) $^{0.097}$, with 15-year lag

[2] Relative RR proportional to exp (0.000015 AUC). This is based on the linear function in the lower range of the piecewise linear model

[3] When body burden exceeds 133 ppb, the AUC years exceeds 40,000 ppt years and the model cannot achieve the prescribed risk level

[4] Relative risk RR proportional to (0.00017 AUC +1)$^{0.326}$

[5] Relative risk RR proportional to (1+0.000016 AUC)

[6] Relative RR proportional to exp (0.00000869 AUC).

[7] Relative RR proportional to exp (0.0003522 x lipid concentration).

There are several important aspects in Exhibit 4 that should be emphasized. These occupational studies were large and involved thousands of workers who were occupationally exposed to dioxin. Even though the studies were conducted with different cohorts in different locations, the reported ED_{01} values in the

three studies are relatively consistent and differ by less than an order of magnitude. For example, the lowest and highest ED_{01} reported by Steenland *et al.* and Ott and Zober were 18.6 and 50.9 ppb (for males), respectively. Considering the complexity of the studies, these ED_{01} values are fairly close. I used the ED_{01} from these data sets to estimate the cancer risk for the reasonable maximum exposed Facility worker. The last aspect that is important is that the reasonable maximum exposed worker body burden is not only higher than the body burdens corresponding to the ED_{01}, but it was also higher than the body burden that produced a 5% increase (ED_{05}) in cancer in the Steenland *et al.* and Becher studies.

The measured reasonable maximum exposed total dioxin TEQ in the Facility cohort was 147 ppb lipid-adjusted. To calculate cancer risk for this reasonable maximum exposed individual, I first subtracted the dioxin attributable to background, which is approximately 19 ppt lipid-adjusted (mean concentration reported in Ferriby 2006). The excess body burden of dioxin attributed directly to the Facility is, therefore, 128 ppt lipid-adjusted (i.e., 147 - 19 = 128). Next, it was necessary to convert the lipid-adjusted concentration in the Facility workers to non-lipid body burden so it would match the units in the published studies. Assuming a body lipid content of 25%, the mean exposure attributed to working in the Facility is 32 ppt (not adjusted for lipid, i.e., 128/4).

Cancer risk associated with the reasonable maximum exposed body burden is calculated with the following equation (EPA 2003):

EXHIBIT 5
EQUATION TO CALCULATE CANCER RISK ASSOCIATED WITH REASONABLE MAXIMUM EXPOSURE BODY BURDEN

$$\text{ELCR Risk} = BB * 0.01 / ED_{01}$$

Where:

ELCR = Excess Lifetime Cancer Risk

BB = Body Burden (ng/kg; ppt)

ED_{01} = Effective dose to produce a 1% increase in cancer above background

As discussed, there are three different studies that present a range of central tendency values for ED_{01} which are presented in Exhibit 4. These values are 18.6 ppt (Steenland *et al.* 2001), 32.2 ppt (Becher *et al.* 1998), and 50.9 ppt (Ott and Zober, 1996). After reviewing each study, I concluded that, instead of calculating an average ED_{01} values from these three studies, it would be better to calculate a range of

cancer risks by using the highest and lowest ED_{01}. I adopted this approach to avoid introducing unnecessary uncertainty into my analysis. In fact, this source of uncertainty was the basis of another NAS critique directed at the EPA reassessment report. NAS stated the following:

> *"The committee also concluded that EPA did not adequately quantify the uncertainty associated with responses at the estimated value of the POD. The estimated value of the response at a particular effective dose (like the ED01) is typically uncertain for a variety of reasons related to the challenge of conducting an epidemiological study or an animal study. For example, in epidemiological studies, the number of enrolled subjects is small, it can be difficult to estimate the actual level of exposure, other factors (such as smoking or exposure to other chemicals) can also cause cancer, and so forth. The committee concludes that, although EPA discussed many of these factors qualitatively, the agency should strive to more comprehensively characterize the impact of these sources of uncertainty quantitatively."*

It should be noted that this particular critique is not accurate with regard to the three cohort studies (Steenland *et al.* 2001; Becher *et al.* 1998; Ott and Zober 1996) because they were very large studies that controlled for the above-mentioned complicating factor (called confounders). However, in an effort to eliminate any source of uncertainty in my analysis, I decided to calculate the minimum and maximum cancer risk for the reasonable maximum exposed receptor in the Facility cohort by using the ED_{01} from Ott and Zober and Steenland *et al.*, respectively. Using the equation in Exhibit 5, my calculations are as follows:

Based on Ott and Zober the ELCR Risk is $6.3 \times 10^{-3} = 32 * 0.01/50.9$

Based on Steenland *et al.* the ELCR Risk is $1.7 \times 10^{-2} = 32 * 0.01/18.6$

Therefore, my calculated result for the cancer risk for the reasonable maximum exposed Facility employee is between approximately 6×10^{-3} and 2×10^{-2} (or 6 in 1,000 to 2 in 100). Note that there is less than an order of magnitude difference between these cancer risks. It should also be stressed that these estimates are the cancer risks just from dioxin; they do not include the extra cancer risk posed by HCB, which was also measured in the Facility workers' blood. Cancer risk from HCB body burdens is calculated in the later sections.

Noncancer Health Hazard

In addition to cancer, dioxin also poses noncancer effects, which toxicologists refer to as "systemic effects." EPA typically identifies a dose or exposure level, called the reference dose, below which it anticipates no adverse effects from exposure, even among sensitive members of the population. Even though EPA conducted a thorough review and analysis of systemic effects, EPA did not develop a reference dose that could be used to calculate noncancer health hazards, which is required in a comprehensive health assessment, despite the considerable evidence showing that noncancer effects may occur at similar exposures that produce cancer. This oversight was the target of the following pointed critique in the NAS review of the EPA reassessment report:

> *"EPA did not estimate an RfD for TCDD, other dioxins, or DLCs in the Reassessment. The committee suggests that estimating an RfD would provide useful guidance to risk managers to help them (1) assess potential health risks in that portion of the population with intakes above the RfD, (2) assess risks to population subgroups, such as those with occupational exposures, and (3) estimate the contributions to risk from the major food sources and other environmental sources of TCDD other dioxins, and DLCs for those individuals with high intakes."*

In the absence of an EPA-verified reference dose, I identified two well-conducted studies that have developed a reference dose. The first study was conducted by the World Health Organization in its *Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI)* (1989). It states:

> *"During the last years the WHO European Centre for Environment and Health (WHOECEH) has been coordinating a comprehensive programme in collaboration with the International Programme on Chemical Safety (IPCS) on PCDDs, PCDFs and PCBs, aiming at evaluating the possible health risk, and prevention and control of environmental exposure of the general population to these chemicals… Several WHO meetings in the field of the health risk assessment of dioxins and related compounds have been convened. At a meeting held in Bilthoven, The Netherlands (December 1990), a tolerable daily intake (TDI) of 10 pg/kg b.w. for TCDD was established. Since then new toxicological, epidemiological and mechanistic data have emerged, in particular with respect to neurodevelopmental, reproductive and endocrine."*

The methodology used by WHO to derive a tolerable daily intake (TDI) is identical to the methodology used by EPA to derive a reference dose. In its reevaluation of the TDI, WHO reduced the safe exposure level from 10 pg/kg-day dioxin TEQ to 1 to 4 pg/kg-day dioxin TEQ. WHO emphasizes that the upper range of the TDI of 4 pg /kg-day should be considered a maximal tolerable intake on a provisional basis and that the ultimate goal is to reduce human intake levels below 1 pg/kg-day dioxin TEQ.

A study by Greene *et al.* also produced a reference dose after comprehensively reviewing all pertinent studies (2003). (It should be noted that the authors are employees of Chem Risk, which is a consultant for the Facility). Their overall conclusions are as follows:

> *"Although the cancer hazard posed by this chemical has probably received the bulk of attention over the past 20 years, the U.S. Environmental Protection Agency (EPA) and the recent U.S. EPA Science Advisory Board (SAB) that reviewed the "Reassessment" have suggested that the noncancer hazard may well be more important than the cancer hazard at current background doses to the general public. The World Health Organization (WHO) and U.K. Food Standards Agency (FAO) committee (JECFA) on dioxins has reached similar conclusions. This article reviews the published studies involving laboratory animals and humans that address the noncancer effects. Based on our review, developmental toxicity is the most sensitive effect of TCDD consistently seen in mice and rats. Specifically, of the various studies, a no-observed-adverse-effects level (NOAEL) of 13 ng/kg (maternal body burden) was identified as the most pertinent for deriving a reference dose (RfD) for humans. Although more than a dozen different adverse effects have been reported in various studies of humans over the past 25 years, the most consistent clinically important adverse effect of human exposure appears to be chloracne. Following a review of all published studies, we concluded that the best estimate of a LOAEL for production of chloracne is approximately 160 ng/kg (body burden). Based on our analysis, an RfD of between 1 and 10 pg/kg-d (TCDD TEQ) is consistent with the objectives of this risk criterion. Maintaining a lifetime average daily dose below this concentration, based on what is known today, should prevent noncancer effects in virtually all persons."*

Thus, there is general agreement between the two studies.

In order to determine whether Facility workers' exposure rates exceed the derived TDI or reference dose, it was necessary to first convert the dioxin TEQ body burden for the reasonable maximum exposed individual in the Facility cohort to an average daily dose. The calculated average daily dose can then be directly compared with the TDI and reference dose. Both the WHO and Greene *et al.* studies provide the following equation that can be used to convert body burden to average daily dose.

EXHIBIT 6
EQUATION TO CONVERT BODY BURDEN
TO AVERAGE DAILY DOSE

Average Daily Dose = Body Burden * (0.693/Half-life)/Absorbed Fraction

In this equation, the half-life equals the amount of time necessary to eliminate one-half the dioxin body burden, which is assumed to equal 7.5 years, or 2738 days. The absorbed fraction is 50% (WHO 1998). The dioxin body burden for the reasonable maximum exposed Facility individual was derived in the above section and is 32 ppt. Using the equation in Exhibit 6 with these assumptions, the average daily dose is:

16 pg/kg-day = 32 * (0.693/2738)/0.5

From this equation, the average daily dose for the Facility reasonable maximum exposed individual is 16 pg/kg-day. This average daily dose can now be directly compared with the safe TDI level of 1 to 4 pg/kg-day derived by WHO, as well as with the 1 to 10 pg/kg-day reference dose derived by Greene *et al.* From this comparison, I conclude that the reasonable maximum exposed individual is exposed to a far higher dioxin dose than is safe. For example, the dioxin exposure level for the Facility worker is 4 to 16 times higher than WHO's safe TDI exposure level. Likewise, the reasonable maximum exposed is 1.6 to 16 times higher than the 1 to 10 pg/kg-day reference dose derived by Greene *et al.*

It is very important to note that I intentionally subtracted the background exposure from body burden for the reasonable maximum exposed individual. However, for evaluating the health of the reasonable maximum exposed individual, background must be considered because background exposure is occurring together with Facility-related exposures. Both the TDI and the reference dose represent exposures that should not be exceeded, regardless of the source of dioxin (the body does not distinguish between sources). Indeed, the reason EPA did not derive a reference dose for noncancer effects for dioxin is because it realized the reference dose would have no practical use since some toxic effects were observed *below* background exposure levels. In its dioxin reassessment, EPA states:

> *"The decision was made not to calculate a reference dose (RfD) because the U.S. EPA calculations indicated that it would be below the current background body burden (0.5 ng/kg TCDD). Because of the relatively high background levels as compared to effect levels, the Agency is not recommending the derivation of a reference dose (RfD) for dioxin and related compounds. Although RfDs are often useful because they represent a health risk goal below*

> *which there is likely to be no appreciable risk of noncancer effects over a lifetime of exposure, their primary use by the Agency is to evaluate increments of exposure from specific sources when background exposures are low. Any RfD that the Agency would recommend using a traditional approach for setting an RfD using uncertainty factors to account for limitations of knowledge is likely to be below—perhaps significantly below (by a factor of 10 or more)—current background intakes and body burdens."*

Likewise, WHO has also determined that exposures to background levels may produce subtle health effects for some:

> *"It recognized that certain subtle effects may be occurring in some sections of the general populations of industrialized countries at current intake levels (2- 6 TEQ pg/kg bw/day) and body burdens (4-12 TEQ ng/kg bw), but found it tolerable on a provisional basis as these reported subtle effects were not considered overtly adverse and there were questions as to the contribution of non-dioxin-like compounds to the observed effects. The consultation therefore stressed that the upper range of the TDI of 4 pg TEQ/kg bw should be considered a maximal tolerable intake on a provisional basis."*

What these two statements indicate is that subtle toxic effects may be occurring at background levels. Therefore, any exposure added to background exposures will almost certainly have some toxic effect.

7.1.3 HCB Body Burdens

I made four conclusions based on my analysis of HCB body burdens in Facility workers. First, like dioxin, HCB body burdens in Facility workers are well above the U.S background level (which is zero). Second, HCB body burdens have significantly increased between 2002 and 2004. Third, I estimate the cancer risk for the HCB body burden level in the reasonable maximum exposed worker to be approximately 2×10^{-4} (or 2 in 10,000). Fourth, the noncancer health threat is very high, and the estimated reasonable maximum exposed HCB exposure is about 38 times a safe level.

As discussed previously for dioxins, it is important to first determine whether Facility workers have HCB body burden levels that exceed background levels in the U.S. general population. To make this determination, I compared HCB body burden levels in Facility workers, which were recently measured by NIOSH (2005), with background body burden levels reported in the CDC National Center for Health Statistics (NCHS) database of NHANES, which was conducted during 2001-2002. The Second Report

NHANES study, released in January 2003, presents blood data for a similar timeframe. The data in the Second Report clearly show that the levels of HCB in the U.S. general population have fallen below detection limits. That is, HCB is no longer detectable in blood samples from the general U.S. population because HCB has not been produced in the United States for more than 25 years. Therefore, any HCB detected in any Facility employee must result from direct exposure to Facility uncontrolled releases of HCB.

The HCB body burden results from the Facility study conducted by MWH (2002) are shown in Exhibit 7. These results clearly indicate worker exposure is widespread and has led to an increase in body burden. Approximately 77% of the employees had detectable levels of HCB in their blood. Perhaps the most important finding, however, was the widespread dispersal of HCB contamination throughout the Plant area and into non-production areas. An unexpected finding was that employees in non-production positions (such as clerical employees) had higher mean HCB levels than Chemical Operations/Other Production Positions. Non-production employees had a mean concentration of 10 times the concentration detected in workers in Chemical Operations, and the maximum HCB blood level in employees working in Non-production areas, which included clerical workers, was 16 ppb serum.

EXHIBIT 7
THE MWH STUDY: HCB BODY BURDEN

WORKER JOB CLASSIFICATION IN BLOOD VOLUNTEER GROUP	HCB BLOOD SAMPLE RESULTS				
	Number of Samples [(1)]	Number of Samples Detectable HCB	Percentage Samples Detectable HCB	Mean	Maximum
Maintenance	47	43	91%	4.8	20
Reactor Process	8	8	100%	5.4	16
Electrolytics	16	15	94%	4.0	14
Chemical Operations and Other Production Positions	7	2	29%	0.8	1.6
Administrative	15	4	27%	1.8	16
TOTAL	93	72	77%	3.4	20

Notes: All concentrations are µg/L; ppb serum.

Source: Table adapted from MWH 2003.

(1) Note that there were 98 participants; approximately 5 of the samples collected were classified as "assay failed" by the laboratory.

The last important issue associated with elevated HCB levels in Non-production employees is that this group includes women of childbearing age and, as previously discussed, this group is a particularly sensitive population.

The high body burden levels of HCB in Facility workers were confirmed in the second study, which was performed by NIOSH in 2004 (NIOSH 2005). NIOSH measured HCB in blood of 30 Facility workers who worked in electrolytic and melt reactor departments. (See Exhibit 8.)

EXHIBIT 8 NIOSH STUDY: HCB BODY BURDEN

DEPARTMENT	N	METHOD	MEAN	MINIMUM	MAXIMUM
Cell Brick	11	Lipid adjusted	591.70	252.90	2179.83
Electrolytic	8	Lipid adjusted	778.99	316.08	1629.45
Maintenance	6	Lipid adjusted	2465.13	824.68	6788.60
Reactor	5	Lipid adjusted	801.91	472.01	1980.29
Total	**30**	**Lipid adjusted**	**891.1**	**253.0**	**6790.0**

Source: Table adapted from NIOSH 2005, ppb lipid.

Both MWH and NIOSH studies show HCB body burden levels in Facility employees are much higher than they are in the general U.S. population.

For my next analysis, I compared blood levels from the two studies to determine whether HCB blood levels had declined over the two-year period from 2002 to 2004. I was interested (from an occupational health standpoint) in whether steps taken by Facility management were effective in reducing HCB exposures and body burden. Any successful mitigation efforts would have been revealed in a decrease in workers' blood levels since the half-life (the time necessary to eliminate half the body burden of HCB that existed at "time zero") of HCB in the body is approximately 1.5 years. HCB body burden at any point in time represents the difference between intake and elimination. When intake ceases, HCB is slowly eliminated from the body. For example, if HCB exposures and intake ceased completely in 2002 when the Facility collected blood samples, the body burdens in 2004 would have been approximately one-half the 2002 concentration (assuming a half-life of 1.5 years). As the following comparison shows, blood HCB levels did not drop. On the contrary, they increased significantly. This demonstrates the workers continued to be exposed to HCB.

Although direct side-by-side comparisons of individual workers could not be made between the MWH and NIOSH studies (because worker names were not disclosed), general conclusions can be drawn about overall changes in HCB body burdens. For this comparison, I first converted the 2002 HCB blood levels

that were reported as serum concentrations to lipid-adjusted serum units because the 2004 NIOSH study reported lipid-adjusted values. I made the conversions with the following equation (CDC 2005; Needham *et al.* 1990).

EXHIBIT 9
EQUATION TO CONVERT SERUM CONCENTRATIONS TO LIPID-ADJUSTED SERUM UNITS

Lipid-adjusted Serum (ppb) = (Serum [µg/L] * 31) / 0.189

I confirmed that this conversion was correct in a telephone conversation with Drs. Don Patterson and Wayman Turner at the CDC laboratories (24 January 2007). Drs. Patterson and Turner had performed the original analysis for NIOSH and stated they routinely use the same procedure to make unit conversions in their laboratory.

The results of the 2002 and 2004 HCB body burdens in Facility workers are presented in Exhibit 10. Comparisons were made between the three departments that were measured in both studies. This comparison clearly shows that HCB body burden did not decrease for employees in the Maintenance and Electrolytic departments over the two-year period. Rather, they increased rather substantially for the Maintenance workers. Furthermore, the maximum body burden in 2004 was approximately double the level measured just two years earlier. The overall trend is clear: body burdens have not decreased but have, in fact, increased over the two-year interval.

EXHIBIT 10
COMPARING 2002 AND 2004 HCB BODY BURDENS

WORKER JOB CLASSIFICATION IN BLOOD VOLUNTEER GROUP	HCB BLOOD CONCENTRATION	
	2002 [1]	2004 [2]
	Mean Concentration	Mean Concentration
Maintenance	787	2465
Reactor Process	886	802
Electrolytics	656	779
	Maximum = 3280	**Maximum = 6788**

Note: All concentrations are ppb lipid-adjusted serum

[1] Table adapted from MWH (2003) Serum concentrations were converted to lipid-adjusted serum.

[2] Table adapted from NIOSH (2005)

7.1.4 Toxicity Associated with HCB Body Burdens

I investigated both cancer risk and noncancer health hazards based on the HCB body burden levels measured in the NIOSH study. To estimate cancer risk, I needed to calculate HCB exposures (i.e., the average daily dose) by using the same equation that is routinely used to convert body burden to daily exposure levels that I previously explained for dioxin. Based on an average daily dose of 1.1×10^{-4} µg/kg-day (detailed in the Cancer section below), I estimate the cancer risk to be 2×10^{-4} (or 2 in 10,000). For the noncancer health hazard, I compared the HCB body burden level of 34 ppb in the reasonable maximum exposed Facility worker with the health-based HCB body burden of 1.1 ppb serum that I independently derived. Based on this comparison, the HCB body burden in the reasonable maximum exposed individual poses a health threat. My rationale is explained in the following sections.

Cancer

EPA has derived and verified a cancer slope factor for HCB of 1.6 (IRIS 2006). As discussed in the previous section, the cancer slope factor represents the ability or potency of a chemical to produce cancer. Unlike my previous calculation of dioxin-induced cancer risk where I was able to calculate risk by directly comparing blood levels measured in several published cohort studies with those of the Facility workers, for HCB I needed to first estimate the average daily dose for HCB. This is because EPA derived the cancer slope factor based on HCB intake (average daily dose), rather than blood levels. As shown previously, the conversion between body burden and average daily dose is straightforward (as shown in Exhibit 6, Average Daily Dose = Body Burden * (0.693/Half-ife)/Absorbed Fraction) and can be used to convert the Facility reasonable maximum exposed HCB blood level to average daily dose.

For this conversion, I assumed a half-life of 1.5 years, or 548 days, and an absorbed fraction of 38% (Burton and Bennett 1987; Schlummer *et al.* 1998; Albro and Thomas 1974; Freeman *et al.* 1989). The dioxin body burden for the reasonable maximum exposed Facility individual is 0.034 mg/kg. Therefore, the HCB average daily dose is 1.1×10^{-4} mg/kg-day (i.e., 1.1×10^{-4} mg/kg-day = 0.034 mg/kg * (0.693/548 days)/0.38).

The cancer risk is calculated from the following equation:

ELCR = average daily dose * CSF

Thus, the cancer risk for the reasonable maximum exposed Facility individual is approximately 2×10^{-4}, or 2 in 10,000 (i.e., $2 \times 10^{-4} = 1.1 \times 10^{-4} * 1.6$).

Noncancer Health Hazard

Unlike HCB-induced cancer risk, I was able to determine the noncancer health hazard for the reasonable maximum exposed worker directly from the body burden measurement. However, this required that I first derive a safe health-based reference body burden because no regulatory agency or health organization has yet developed a benchmark body burden level.

The safe body burden benchmark is based on the No Observable Adverse Effect Level (NOAEL), which is the foundation of toxicological evaluations. The standard procedure is to first identify the NOAEL for

a chemical. Simply put, the NOAEL is the body burden level at which no adverse toxic effects are observed. The following are the conventional three steps in deriving a safe benchmark body burden:

1. Identify *the* principal study that presents the best and most pertinent toxicity information for the most sensitive toxic effect;
2. Identify the body burden corresponding to the NOAEL in the principal study; and
3. Modify the NOAEL with appropriate safety factors, if necessary, to ensure human health is protected.

After identifying the NOAEL in the principal study, it may be necessary, depending on the quality and study design of the principal study, to modify the reported NOAEL with safety factors. For, example if the principal study was conducted on laboratory animals, a safety factor of 10 would be used to extrapolate those study results to human exposure. Safety factors account for less-than-perfect studies where uncertainty is introduced by flaws in experimental method or protocol, high variability, species extrapolation, etc.

I conducted a comprehensive review of all available pertinent peered-reviewed studies of HCB and identified approximately 8 to 10 studies providing important toxicological information based on human exposures. From these, I concluded the Volker *et al.* (2001) study to be the principal study because it reveals the most sensitive toxic effect (the toxic effect produced with the lowest body burden) is the immune system. Their results show that HCB produces a very specific and unique immunotoxic effect in humans. They state:

> *"The observed positive and negative associations of cellular and humoral immune parameters with blood levels of PCBs, HCB, and HCHs were relatively weak, with the exception of strongly negative association of IFN-γ with HCB. This finding indicates that HCB has a significant impact on Th1 lymphocytes, is involved in the induction of cellular immune responses against antigens such as viruses by activating NK [natural killer cells], monocytes/macrophages, and granulocytes, and in humoral immune responses by increasing the immunoglobulins secrection of plasma cells."*

The acronym IFN-γ stands for interferon, which is a protein that is important for proper functioning of the immune system. Volker *et al.* concluded that the toxic effect they measured in the immune system could ultimately predispose individuals to viral and bacterial infections, and make them more susceptible to a variety of illnesses. Equally important is that they observed these changes in the immune system at body

burdens as low as 1.1 ppb serum—which is a very low body burden. Assuming that 1.1 ppb serum is the lowest concentration at which Volker *et al.* observed immunotoxic effects, and that they did not observe any effects lower than this body burden, I have concluded the NOAEL for HCB body burden should be 1.1 ppb serum. I also concluded that the Volker *et al.* study was well designed and executed, and, since it was a human study, no safety factor was necessary to modify the NOAEL. To make the comparison of this health-based benchmark with the NIOSH HCB levels, I converted the 1.1 ppb serum level to approximately 180 ppb lipid-adjusted serum (see Exhibit 9 above).

My conclusion is that the 6788 ppb lipid-adjusted HCB body burden of the reasonable maximum exposed Facility worker far exceeds the 180 ppb health-based benchmark, by approximately 38 times.

Explaining Why Dioxin and HCB Body Burden is High

There is clear supporting evidence that explains why the Facility employee HCB body burden levels are high. The HCB is generated as a byproduct during manufacturing operations (NEIC 2003) and, once released, it is an uncontrollable health hazard. After it is released into the air as a dust of fine particles, it settles out as a dust that contaminates all work surfaces, as well as worker clothes, skin, and hair. EPA and NIOSH have collected numerous samples from many different operation areas in many different departments, and there is a clear association between high body burdens and high levels of contamination. In addition to sampling waste and product streams in production areas where they originate, samples have been collected from:

1. The air workers breathe;
2. Work surfaces they touch;
3. The hands of workers;
4. Coveralls at the end of the work shift;
5. Lunchroom table surfaces; and
6. Bulk dust where it has accumulated and deposited in crevices over the years.

When considered collectively, the sampling results indicate that, for some Facility employees, there is no escaping exposure to HCB and dioxin during their work shifts. Contaminants are absorbed into workers' bodies when they inhale airborne contaminated dust, when they inadvertently ingest dust through hand-to-mouth activities, and when contaminants are absorbed through the skin. Contamination is ubiquitous and

provides substantial evidence that these exposures occur on a routine daily basis and are not the result of unique or sporadic contact events. The following is some of the data I relied on for my conclusions.

EPA has collected bulk air samples that have shown high levels of HCB and dioxin in the general workplace air, with some of the highest concentrations detected in the melt reactor building. In addition, I observed many Facility workers not wearing respirators (and some have been inhaling these contaminants for 20 to 30 years). Persuasive evidence that some Facility workers have inhaled unacceptably high levels is presented in the NIOSH study (2005) sampling results. Forty-two personal breathing zone monitors were attached to employees' work coveralls as the workers performed routine assigned job responsibilities. The personal breathing zone monitored what that particular employee was breathing and, of those forty-two personal breathing zone samples, five exceeded the safe recommended exposure level for HCB of 2 microgram/cubic meter (ug/cu.m.) that was developed by the American Conference of Governmental Industrial Hygienists. Dioxin levels were not measured in these samples, but they were presumably high since dioxin and HCB are closely associated in the waste streams.

I observed many employees not wearing respirators during my 26-27 July 2006 tour, despite considerable gas and dust emissions that occurred on a regular basis. For example, Photographs 3, 4, 5, and 6 in Appendix B show a group of employees welding I-beams on the 6th floor of the melt reactor building, which is (according to NEIC data) one of the more heavily contaminated areas of the plant. None were wearing respirators, even though they were also generating considerable welding fumes. As I toured the melt reactor building, I observed workers wearing respirators only sporadically at best. Photograph 7 again shows the workers not wearing respirators; later in the day, the workers unzipped their coveralls (due to the heat), and the work clothes that they wore under their coveralls were clearly contaminated (I discuss the importance of this with regard to take-home contamination). At another point on my tour, I observed approximately five workers using a pressurized air hose to blow dust from the road surface they were repairing inside the electrolytic building. The cloud of dust they generated was so thick that the photograph I took of the activity (Photograph 8) was completely distorted by the dense dust cloud they generated. During this activity, I made three health-related observations. First, none of the men were wearing respirators. Second, more than one employee was wearing a personal work shirt and trousers, and not protective coveralls (which relates to take-home contamination). Third, I later discovered that a NIOSH hand-wipe sample from a worker in this department had the highest HCB residue level on his hands that was measured by NIOSH (indicating workers come into direct contact with HCB).

Perhaps the most significant exposure route for workers is ingestion, as compared with inhalation and dermal absorption. Ingestion occurs when workers put their fingers to their mouths and lips, which occurs surprisingly frequently for all humans. I saw many instances in which workers were in direct contact with likely contaminated surfaces. For example, I observed workers using telephones, such as the one in Photograph 9, that were obviously contaminated. Contacting such surfaces after taking off their gloves, then holding the phone will lead to inadvertent ingestion by workers. Photograph 10 shows an example of a surface that was commonplace on my tour through the melt reactor building. I observed many workers not wearing gloves during my 26 July 2006 tour. Photograph 11 shows a worker's hands that were almost completely black.

Observing workers for a period of time revealed that they repeatedly took off their gloves, touched their faces and lips, and then put their gloves back on many times an hour, as shown for one worker in Photograph 12 (top). This is quite normal mouthing behavior (under the very hot conditions), but a behavior that usually goes unnoticed by the workers themselves. I also saw pairs of gloves lying near workers like those shown in the second photograph in Photograph 12 (bottom), suggesting some workers were not wearing gloves at all. Some of these gloves appeared to be as soiled on the inside as they were on the outside. From these observations, I concluded that, while workers' gloves protect them from physical injury, they provide no protection from contamination via ingestion or dermal absorption. Additionally, because the gloves appeared to be as contaminated on the inside as they were on the outside, and workers wear these gloves until they wear out, once contamination gets trapped inside the gloves, workers' hands will simply be re-contaminated each time they put their gloves back on. Recontamination will occur even after workers have washed their hands. Because hands perspire inside soiled gloves with trapped contamination, the act of wearing gloves may actually enhance contaminant absorption through the skin of workers' hands.

Ingestion also occurs while workers eat and smoke with contaminated hands. The hand wipe samples NIOSH collected from workers clearly shows some workers' hands to be highly contaminated. In fact, wipe samples showed high HCB contamination residue on some workers' hands even after washing. For example, a wipe sample was collected post-washing from a worker's hand in the electrolytic department after he "pigged a header." This sample showed that 1.3 micrograms HCB remained on his hands, despite vigorous washing. As most workers simply assume their hands have no contaminant residues after they wash them, they may unintentionally expose themselves under the assumption that their hands are free from contamination.

I saw evidence of workers eating and drinking within the plant (on both of my trips), and some of this activity was found in some of the most highly contaminated areas. For example, Photograph 13 shows a worker who was eating a sandwich sitting inside the cab of a smutting truck while pumping the molten magnesium solution. Although it is difficult to see, the first photograph shows him eating, and the second photograph shows the dense fumes that had enveloped his truck a short while later. I also observed considerable evidence of smoking and drinking in areas where company policy prohibits it. Photograph 14 shows a cigarette butt and a water bottle in the melt reactor building, which were not rare findings. On a subsequent tour of the Facility, I had the opportunity to observe several workers rebuilding a cell on the 6th floor of the reactor building. None were wearing respirators, and at least two workers were smoking while they applied mortar to refractory bricks. Photograph 15 shows one of two or three workers who repeatedly smoked during a period of 2 to 3 hours. Several contaminant gas and dust clouds were released while the workers labored in that area, which exacerbated this situation. Photograph 16 shows one such uncontrolled release of fumes while I was observing the workers.

Evidence of other potential exposures was observed inside the concrete containment pit where the grizzly boxes accumulate anode waste. While observing employees cleaning out the grizzly box wearing highly protective full-face respirators and hooded Tyvek suits with booties, I noticed a cigarette butt lying just a few feet from the two employees. While the two employees were taking all the necessary precautions handling such toxic dust, it was ironic that there was evidence just a few feet away that other workers were smoking in the area. The first photograph in Photograph 17 shows the fully protected workers carefully removing anode dust from the grizzly box while fully protected, and the second shows the cigarette butt lying a few feet away. It is not clear who is smoking in that particular area. When asked if "anybody smoked cigarettes in the grizzly box area," Mr. Silva (who is the Off Gas Operator foreman; 13 September 2006 deposition) answered that none on his crew smoke. However, the number of cigarette butts in that general area indicates employees frequently smoke in this area.

Although each mouthing incident event may seem trivial, they are not trivial when each hand-to-mouth activity occurs perhaps a hundred times per day for an individual for 20 to 30 years. Based on my observations and review of all the data, I have concluded that ingestion exposure may be one of the most (if not *the* most) important exposure routes.

Recent coverall data clearly shows employees come into direct contact with dioxin and HCB, and these data directly relate to the Facility workers' high body burden levels. EPA (2006) identified three job activities and three the Facility employees who were to perform those activities. According to Facility

management, none of these activities required a high level of protection and, consequently, none of the workers was wearing Tyvek. At the end of their shift, EPA collected the workers' coveralls and sampled a portion on one half of their coveralls (the other half was given to the Facility consultants). According to detailed EPA notes, coveralls were collected from Messrs. Smith and Jones, who were replacing lances in the melt cells in the reactor buildings. EPA also obtained coveralls from Mr. Burnett, who cleaned the electrolytic cell cooling box in the electrolytic building. Photographs were taken to document the condition of the workers' coveralls and some of the conditions they were working in.

Photograph 19 shows the condition of Mr. Burnett's coveralls at the end of his shift. Photograph 20 shows a cloud of anode dust escaping from the electrolytic header as he was cleaning it. This dust is highly contaminated with HCB and dioxins because it is the same anode dust that workers clean from the grizzly box (where they must wear Tyvek, although Mr. Burnett did not). Photograph 21 shows Mr. Burnett brushing anode dust with very little protective equipment.

Photograph 22 shows the condition of Mr. Derric Smith's coveralls at the end of his shift after replacing the lances. Photograph 23 shows Mr. Smith in the middle of a generous chemical release near the lances he replaced. Photographs 24 and 25 show Mr. Jones at the end of his shift cleaning some pipefittings. What is interesting to note from these pictures is that, while all three had very soiled and dirty coveralls at the end of the shift, Mr. Burnett's were perhaps the "cleanest." Despite the appearance of being less soiled, his coveralls were much more contaminated with HCB and dioxin than those of either Messrs. Jones or Smith, suggesting the physical appearance is not a good indicator of contamination. Exhibit 11 presents the coverall sampling results for HCB and dioxin.

EXHIBIT 11
THE FACILITY WORKER COVERALL CONTAMINATION

THE FACILITY EMPLOYEE/JOB DESCRIPTION	JOB RELATED ACTIVITY	AMOUNT OF DIRT AND DUST ON SAMPLE [1] (MG)	TOTAL DIOXIN TEQ [2]	TOTAL DIOXIN TEQ [3]	HCB
Mr. Burnett	Cleaned an Electrolytic Cell Cooling Box Electrolytic Building	795	51	120	5100
Mr. Smith	Working on Lances on Melt Reactor Cell Melt Reactor Building	370	11	15	210
Mr. Jones	Working on Lances on Melt Reactor Cell Melt Reactor Building	990	19	26	400

Note: All concentrations are ppb

[1] Amount of dirt and dust on small piece of coverall sample. Total dirt and dust on coverall would be significantly higher.

[2] Total dioxin TEQ based on WHO 2005 TEF values.

[3] Total dioxin TEQ based on ITE TEF values.

Three conclusions can be drawn from this coverall data. First, the total weight of dirt and dust adhering to the coveralls was substantial. Second, by any measure, these coveralls were highly contaminated with dioxin and HCB, which shows just how much contamination the workers contacted during their shifts. Three, these workers were in contact with high levels of dioxins and HCB during their shifts. Mr. Thayer, who is Vice President of Operations, stated that the company does not consider the jobs performed by Messrs. Jones and Smith (in the melt reactors) to be ones involving "direct contact" (according to his deposition).

One last source of data shows that workers come in contact with contamination where they perhaps least expect it—namely, lunchroom tabletops. NIOSH took wipe samples of lunchroom tabletops and analyzed for HCBs (but not dioxins). NIOSH found detectable HCB levels in the electrolytic lunchroom.

8 TAKE-HOME CONTAMINATION

I have concluded it is likely that the Facility employees have unwittingly contaminated their vehicles and homes, and exposed their family members to uncontrolled releases of dioxin and HCB from the Facility. In addition, I have concluded that employees of G&K Services are exposed to Facility contaminants and may not be protecting themselves from exposure.

Exposures to take-home contamination by family members have been well documented. Indeed, some studies have shown that workers very similar to the Facility workforce brought HCB home with them, thereby exposing their families. When workers hug family members; prepare food; and touch the furniture, bedding, and carpet in their homes, they may contaminate them if they have not washed their skin and removed or decontaminated soiled clothing and personal protective equipment.

My conclusion regarding this serious health threat is based on the following:

- Many peered-reviewed studies showing that family members exposed to workplace contamination have manifest toxic symptoms, and some have died;
- Testimony that some Facility workers have taken their work clothes and coveralls home to be laundered;
- Measured levels of dioxin and furans on the Facility worker coveralls at the end of their shifts;
- Observations that the work clothes Facility workers wear under their coveralls get soiled;
- Testimony that Facility workers do not shower or bathe after work;
- Measured levels of HCB on Facility workers' hands even after they have just been washed;
- Testimony that some Facility workers use their own vehicles for transport to and from work; and
- Testimony that wives of some the Facility workers' wash their work clothes.

To determine the likelihood of take-home contamination by Facility workers, I first conducted a review of all pertinent studies and peered review publications to identify studies that had similar exposure conditions. I briefly summarize some of my findings of those studies below to highlight the breadth and magnitude of this often-overlooked health problem. Due to the sensitivity that newborns and infants have

toward HCB, and to a somewhat lesser degree toward dioxins, I have emphasized studies where they have been the main focus of take-home contamination investigations.

Many studies have documented take-home contamination, which is sometimes also referred to as "paraoccupational exposure" or, more vividly, as "fouling one's own nest." The sources of take-home contamination are not only the more obvious work clothes that are taken home to be laundered, but also contaminated worker hair and skin.

In response to growing concern over this health problem, the U.S. Congress passed the Workers' Family Protection Act (Public Law 102-522, 29 U.S.C. 671) in 1992, which requested that the CDC's National Institute for Occupational Safety And Health (NIOSH) conduct a study to "evaluate the potential for, prevalence of, and issues related to the contamination of workers' homes with hazardous chemicals and substances...transported from the workplaces of such workers" (NIOSH 1995). This was prompted by recognition that this was a serious and compelling public health issue, bridging health concerns in the workplace and the home.

NIOSH concluded that take-home contamination was indeed a health problem that was often ignored or overlooked. The Agency concluded that toxic levels of workplace chemicals are brought into workers' homes where family members, particularly spouses and their children, have fallen ill and sometimes show all the hallmark chemical-specific toxic symptoms. Moreover, the Agency found this problem to be worldwide, with incidents reported in 28 countries and in 36 of the 50 United States. NIOSH reviewed incidents that resulted in a wide range of chemically induced diseases and, in some cases, death among family members.

In its review of take-home contamination, NIOSH identified several health effects suffered by family members that were directly related to the type of chemical in the workplace. Some of the toxic effects included the following:

- Chronic beryllium disease;
- Asbestosis and mesothelioma from asbestos fibers;
- Lead poisoning with subsequent neurological effects and mental retardation;
- Pesticide-related deaths and neurological effects;

- Chemical burns from caustic substances used in the workplace;
- Chloracne from chlorinated hydrocarbons;
- Neurological effects from mercury;
- Abnormal development from estrogenic substances;
- Asthmatic and allergic reactions from dust;
- Liver cancer from arsenic;
- Dermatitis from fibrous glass;
- Epileptic seizures; and
- Diseases from infectious agents.

Although many more studies have been conducted in recent years, at the time the NIOSH (1995) report was completed, it concluded that take-home contamination was significantly underreported and that many medical conditions in family members go undiagnosed. The Agency noted the following limitations of their report:

- Little research has documented the frequency and distribution of health effects among the families of workers in various industries and occupations. NIOSH is undertaking one study addressing lead exposure among families of bridge repair workers.
- Lead and pesticides are the only contaminants for which monitoring or reporting programs help to identify and prevent cases of poisoning from contamination of workers' homes.
- Despite various case reports, the prevalence of health effects from workers' home contamination is not known because there are no surveillance systems in place for tracking or monitoring such health conditions.
- Many diseases have long latency periods between exposure and manifestation, making identification and intervention difficult.
- The workplace origin of many common diseases that occur in workers' families (such as asthma, dermatitis, and infectious diseases) is probably unrecognized because physicians and other health professionals fail to inquire about the occupation of family members and to consider whether these diseases are work-related.

- The literature reviewed in this report contained only nominal information about contamination levels in workers' homes. Most measurements were of surface dust, for which there are no guidelines for acceptable levels of contamination.

- Many of these same problems prevent a full health evaluation of take-home contamination from the Facility workers' homes and the possible health threats it may pose to their families.

It is important to note that many take-home contamination studies not only confirm chemical exposures were occurring among family members, but that the dose was sufficient to produce toxic effects, particularly in children because they constitute the high-risk group. That is, significantly lower amounts are required to produce toxic effects in the young. This is due to a variety of factors, including physiology, metabolism, food consumption rates, and hand-to-mouth activity patterns. Children's respiratory rates, heart rates, and metabolism are significantly different from those of adults. Children's activity patterns also place them at risk because they have close contact with the ground and have a greater skin surface area per kilogram body weight than do adults.

The take-home exposure problem has a long history and is not limited to the United States. NIOSH reported that death and health effects from contaminants brought home from the workplace occurred in 28 countries in addition to 36 U.S. states (NIOSH 1995). According to the NIOSH report (1995), one of the earliest reported cases of take-home contamination was reported by Lehmann in 1905. A mother and child of a worker exposed to chlorinated hydrocarbons developed chloracne (as discussed previously, this condition can be caused by dioxin). Lehmann also noted that a laundress working at a commercial laundering service also developed chloracne as a result of washing the contaminated clothing of workers. This case is very relevant to the Facility case because the Facility also uses a commercial laundry service. Moreover, based on testimony provided by a Facility employee, the Facility has not fully disclosed the levels of dioxin and HCB that contaminate workers' coveralls nor has the Facility disclosed the health hazards associated with those contaminants.

Thirty years after Lehmann's report was published, Fulton and Matthews (1936) reported a similar case from the Pennsylvania Department of Labor and Industry. In this case, a child was exposed to hexachloronaphthalene when his father brought his contaminated work clothes home. The researchers showed the wife, 11-month-old daughter, and a 2-and-a-half year-old son of a worker who worked in a PCB-contaminated workplace developed severe chloracne after playing with their father before he changed from his work clothes. Good and Pensky (1943) and Kominski (NIOSH Report No. HETA 84-250, 1987) also found that wives and other family members of electrical workers developed chloracne resulting from contact with PCB-contaminated clothing.

In more recent years, the consequences of take-home contamination have been studied in much greater detail. A study by Flower *et al.* (2004) provides evidence that children whose parents' jobs included applying pesticides had an increased risk for all cancers (SIR 1.36, 95 % CI 1.03-1.79). Their study included 17,357 children and detected an increased cancer risk among children of fathers who did not use chemically resistant gloves (OR 1.98, 95 % CI 1.05-3.76) and who used aldrin prenatally (OR 2.66, 95 % CI 1.08-6.59).

Lead workers have also been shown to contaminate their homes. A study conducted by Pacitelli *et al.* (1997) for NIOSH evaluated exposures among 37 families of construction workers and a reference group of 22 neighborhood families with no known lead exposures. Workers were identified as having blood lead levels at or above 25 µg/Dl. This group had high measured lead contamination on hands and interior surfaces of homes and automobiles. Hands of lead-exposed workers were seven times more contaminated with lead compared with control workers. Surface lead contamination was significantly higher in automobiles driven by the lead-exposed workers. Surface lead concentrations were significantly higher for exposed homes compared with control homes in rooms where work clothing was changed (GM = 370 versus 120 ppm; $p = 0.005$). While environmental sources of lead were also evaluated, study results strongly suggest that construction workers' occupational exposures, together with poor hygiene practices, were the primary causes of lead contamination. Requirements intended to prevent take-home lead exposures were reported by workers in this study to be infrequently followed by employers, which is similar to practices seen in the Facility.

Ballester *et al.* (2000) showed that, in an exposure situation similar to the Facility, serum concentrations of HCB in spouses married to workers employed in an electrochemical factory were elevated. This study of 608 subjects, 412 of whom had never worked in the electrochemical factory, showed that HCB serum concentrations in spouses of workers were elevated 1.28 and 1.23 times the corresponding value of people not living with workers of the factory, respectively, for spouses of current and past workers (however, relatives other than spouses did not show any increase). While the increase in HCB levels in spouses of current workers is not surprising, excessive levels of HCB in spouses of former workers indicates that, once spouses are exposed to HCB brought home by workers, the levels remain high even after worker employment is terminated.

Sala *et al.* (2000) investigated the effect of living in the same household with a worker employed in an electrochemical factory exposed to HCB in Flix, Spain. They identified an exposed and non-exposed cohort; after measuring their HCB levels, the researchers determined that having a spouse who worked in

the factory was associated with elevated HCB concentrations in serum. The adjusted relative increases were higher than the corresponding values of people not living with workers of the factory for spouses of both current and past workers. Relatives other than spouses did not show any increase.

The above summaries show that the problem of take-home contamination is widespread across many industries. These indirect exposures to family members suggest non-workplace-related exposures result from poor personal hygiene practices of workers who unintentionally bring home contaminants on their clothing, hair, and body parts. The likely reason some studies show spouses have higher exposure to non-workplace contaminants is that they launder the contaminated work clothes. As summarized above, significant exposure can also occur in infants and young children, who crawl on carpets, then put their contaminated hands in their mouths. In addition, workers often come home from work, then play with their children before removing their contaminated clothing and showering. Although studies have not specifically examined exposure routes, it is likely that family members are exposed primarily through inadvertent ingestion involving hand-to-mouth activities because most contaminants are brought home in the form of dusts.

I could not directly review the Facility's policies regarding the prevention of take-home contamination. Instead, I relied on deposition testimony from the Facility management, which indicated several inconsistencies. When asked whether there were any Facility policies intended to prevent workers from taking their coveralls home, Mr. Gines (20 September 2006 deposition) stated he only became aware of the potential for take-home contamination about the time the current lawsuit was filed. Under current policy, workers cannot take their coveralls home to be laundered. While this will reduce take-home contamination, it will not completely eliminate it. Workers are still taking contamination home on their bodies since they do not shower or decontaminate themselves. In addition, Mr. Gines stated that workers have been instructed to keep their coveralls out of the locker room so they would not contaminate their "street clothes." Again, this will reduce exposures to workers, but that policy does not instruct workers to take off their coveralls when in the lunchroom where they eat.

The Facility has not collected any dust or soil samples from employees' homes. According to Mr. Thayer (27 September 2006 deposition), workers have never been advised that taking home their coveralls could have exposed their spouses and children, which is somewhat at odds with Mr. Gines's statement. Mr. Thayer also stated that management has never looked into the relative vulnerability of women or children to the Facility contaminants compared with what might be expected in men.

Facility management has confirmed that most employees do not shower after they finish their shifts at the plant. Mr. Ron Thayer stated (2 November 2006 deposition) that nearly all workers simply take off their coveralls and boots, then leave the plant without showering or even changing from the work clothes they wear underneath their coveralls (which also get contaminated, in some cases). Mr. Thayer further explained the steps Facility workers go through in order to shower; based on that description, showering is not convenient for hourly workers, particularly during cold or inclement weather. This is because there are no toilet facilities in the "hourly shower room," so the hourly employees must use the "salaried shower room." (Photograph 18 shows the unused showers; note there are only two shower stalls for approximately 150 employees per shift.) However, the salaried shower room is approximately 35 or 40 feet from where the hourly workers store their clothes and where the workers must return after showering to get back to their assigned lockers. The walk between the two rooms is outside; in inclement weather, the walk must be a deterrent. Mr. Thayer guessed that, out of the Facility's total workforce, only two or three Facility employees shower before leaving the plant. He stated that only a very small number come into direct contact with chlorinated dust, which, according to the Facility policy, is the only time showers are required. When asked about high-exposure tasks for which the Facility requires showering, Mr. Thayer narrowly defined those as direct handling of dust, which he stated most employees do not perform.

Based on coverall samples, the three employees who performed the work during that shift engaged in "direct exposure." For example, the measured contaminant levels detected on the three employee coveralls that were sampled by EPA Region 8 on 8-9 November 2006 were very high, which is the best indicator of who directly contacts high levels of HCHs. Additionally, while their coveralls indicated Messrs. Burnett, Smith, and Jones were directly contacting dioxins and HCB during their shift, it is not known whether they changed from their work clothes and showered after their shifts ended. It is worth noting that, according to Mr. Thayer, no employee has ever received a citation for not showering, even though some are required to shower.

In addition to the coverall data, NEIC (2006) has also shown high dioxin and HCB levels in bulk dust that contaminates most surfaces within the melt reactor building. Consequently, workers are unknowingly directly contacting contaminated dust on a routine basis just by virtue of working in that area. When asked about the high contaminant levels that have been detected in bulk dust and air samples, Mr. Thayer stated that, unless workers "actively handle chlorinated dust," showering is not required by the Facility. Whether "actively handling dust" or unknowingly coming into direct contact with dust, the contact will contaminate workers' coveralls and work clothes. When asked about a specific activity in a specific area

(as an example) like a worker working around a "reamer shaft in the reactor building" and whether such a worker would be required to shower, Mr. Thayer stated, "It is not our policy on that particular dust." He stated the Facility believes the "concentration of chlorinated organics in that particular dust are much lower than we see in electrolytics." As for exposure conditions, Photograph 23 shows Mr. Derric Smith working in the reactor building the day his coveralls were collected, suggesting that he is in direct contact with "chlorinated organics," as he is in the middle of a dense cloud of gas and dust. Since Mr. Smith would not be required to shower after such an exposure, he likely took home contamination on his body and clothes.

My understanding of routine practice is that workers arrive at the plant in their personal work clothes. When they get to their lockers, they don their coveralls over their personal work clothes, which they wear to and from work. At the end of their shifts, workers simply take off their coveralls and go home, with little or no effort to decontaminate their bodies or work clothes. This is an important aspect of normal practice because, based on personal observations, some work clothes worn underneath workers' coveralls become very soiled. That is, some workers wear their coveralls unzipped because it is extremely hot inside the electrolytic and melt reactor buildings and, consequently, their exposed personal work clothes become contaminated (an example is shown in Photograph 7).

In order to determine whether take-home contamination has occurred from the Facility, I re-reviewed the employee coverall sample data that were presented in Exhibit 11 (discussed with regard to body burden levels). The total weight of dirt and dust adhering to the coveralls that could have been taken home is substantial. For example, just the small patch sampled from Mr. Jones's coveralls had nearly a gram of extractable dirt and dust. If the other side of the coverall, which was given to the Facility's consultants, had approximately the same amount, and the remaining coverall portions (not sampled) were also contaminated, the total dirt and dust could have been more than 2 grams. That is a significant amount of contamination that could be taken home. Second, the dioxin and HCB concentrations in this dirt and dust were excessive by any measure. Cumulative dioxin and HCB take-home contamination would be significant if workers were routinely taking that level of contamination home on a weekly basis for 25 years. For example, Roger Francom, who is the Facility Environmental Coordinator, stated (12 September 2006 deposition) that, when he was an equipment operator, he was responsible for cleaning his own coveralls, which he laundered at home. He also said that that was a common practice among his co-workers. When asked if some employees are still laundering their coveralls at home, he answered that he did not know. Mr. Francom also stated that it was his wife who laundered his coveralls.

No samples have been taken from any workers' personal vehicles to determine whether they are contaminated with dioxins and HCB. However, published studies (Curl *et al.* 2002; Sanderson *et al.* 1999; Pacitelli *et al.* 1997) have shown that workers' vehicles become highly contaminated when workers do not shower and change into clean (non-contaminated) clothing before they leave work. When they leave work and get into their vehicles, the contaminated dirt and dust workers carry on their bodies contaminates the surfaces they contact. Since the Facility employees do not shower and remove their personal work clothes (which I have observed to be dirty and soiled because they do not wear their coveralls properly), there is no reason to believe Facility workers' vehicles used in the carpools are not similarly contaminated. Mr. Francom (12 September 2006 deposition) confirmed that some workers carpool together to the Facility in their own personally owned vehicles. Having multiple workers in a single vehicle exacerbates the problem since, instead of one worker contaminating the vehicle, the contaminant dust and dirt would be the sum of contamination from several workers.

The health problem associated with take-home vehicle contamination is that family members also likely use the contaminated vehicles and, therefore, would be directly exposed to dioxin and HCB. For example, family members would get the same contamination on their hands from touching the steering wheel as was left by the worker.

The last concern regarding off-site exposures to dioxin and HCB from the Facility involves employees of the contract laundry service the Facility employs to clean the workers' rented coveralls. As studies have shown, laundry employees do not take the same precautions as workers because (1) they do not know the contaminant levels on the clothes, and (2) they do not know how toxic the contaminants are. For example, Lehmann (1905) investigated a laundress who developed chloracne when she laundered HCH-contaminated clothes. According to Mr. Gines (20 September 2006), employees can either purchase their own coveralls (which they are responsible for cleaning) or they can rent them, in which case they are laundered by G&K, a commercial laundering service. According to Facility management, they have provided no information or data to G&K regarding the concentrations or the toxicity of dioxin and HCB that contaminate the coveralls. Since it is likely that G&K launders more than 50 pairs of Facility coveralls per week, they could pose a health threat to G&K workers (especially if they are women of childbearing age). If each pair of coveralls is soiled with the levels of contaminants detected in the recent EPA samples, the total amount of dioxins and HCB could be considerable. Even if G&K employees were aware of the risks, they could not likely implement the necessary health-protective procedures to protect themselves without considerable cost and effort. Mr. Gines stated that the Facility has not given G&K any warnings or information about the toxic contaminants on coveralls. When asked what personal

protective steps G&K is taking or equipment they may be using to launder the coveralls, Mr. Gines answered that Facility management has not "been involved in that aspect." He also stated that he has never been to G&K's facilities or observed how employees handled the coveralls. He has never met any of G&K's workers, and he states that G&K has never requested information about the contaminant on the coveralls. Lastly, the health risks may be greater for the one or two G&K employees who handle the coveralls because they are laundering not just a single pair, as Facility wives are reported to do, but are handling many pairs of contaminated coveralls.

In summary, the coverall sample results for dioxin and HCB, together with the poor Facility worker personal hygiene, strongly suggests contaminants are being "taken away" from the Facility. The individuals at risk are family members and G&K employees. The health threat to these individuals is much greater than to the workers themselves because infants (especially those that are nursing), young children, and women of childbearing age are most susceptible to the toxic effects. As discussed earlier, children under two years of age in the Turkey HCB poisoning were the most susceptible and had a 95% mortality rate after developing pembe yara. Although the take-home contamination levels are not nearly as high as the Turkey incident, the results strongly suggest infants and young children are far more susceptible to HCB than adults (under 15 years of age, there is a 10% mortality rate).

9 RISK ASSESSMENT FOR REMOTE AREAS

Dioxins and HCB that were produced inside the plant were intentionally and unintentionally released, and now contaminate many remote areas of the Facility property. This section summarizes the human health risk assessment conducted for the outlying areas located in different parts of the property away from operation/manufacturing areas. To expedite the process of developing an opinion about potential risks, I carefully reviewed the human heath risk assessment conducted by MWH (2003), which was submitted to EPA for its review. To the best of my knowledge, EPA concluded the results and conclusions in the MWH (2003) were correct and were reasonably based on default assumptions. Very little site-specific information was used to modify default parameters, which is the next step in the risk assessment process. Although not required, site-specific information can often reduce the uncertainty introduced into the risk estimates when site-specific conditions are considerably different from default conditions.

To facilitate investigations, U.S. EPA has defined 12 discrete areas of the plant as Waste Management Units. They are:

- Barium Sulfate Area
- Gypsum Pile
- Smut Pile
- Courtyard
- Landfill
- Boron Ditch
- Central Ditch
- Chlorine Ditch
- Main Ditch
- Western Ditch
- Old Waste Pond
- Waste Pond
- 400 Acre Waste Pond

Although waste management units are classified based on similar characteristics or wastes, the size of the waste management unit does not always translate into exposure units in a human heath risk assessment. This is because an estimate of risk is based on exposure units, which are defined as geographical areas that an individual can reasonably be expected to randomly contact on a daily basis. For, example it would be unrealistic to expect a worker to come into contact with soils or sediments in all the areas within the Old Waste Pond (even if all the water were removed), which totals approximately 1,200 acres. A more reasonable size for an occupational exposure would be approximately 1 acre or less. Consequently, it is usually necessary to first evaluate the entire waste management unit, then smaller areas within the waste management unit, to determine if "hot spots" representing exposure units pose elevated risk.

The human heath risk assessment presents an estimate of potential carcinogenic risks and noncarcinogenic hazards associated with current and future exposures. These estimates are then used by U.S. EPA risk managers to determine whether action is necessary to mitigate cancer risk or other threats to human health to acceptable levels.

The scientific methodology used to calculate lifetime cancer risk was initially developed by the National Academy of Sciences (1983) and later adopted by U.S.EPA, which has developed numerous detailed risk assessment guidance documents. The guidance followed in this human heath risk assessment includes:

- *Risk Assessment Guidance for Superfund, Volume I, Part A* (U.S. EPA, 1989) (HHEM);

- *Exposure Factors Handbook* (U.S. EPA, 1989b);
- Integrated Risk Information System (IRIS; U.S. EPA, 2006); and
- *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds (Dioxin Reassessment).*

In accordance with U.S. EPA risk assessment guidance, this human heath risk assessment was conducted without regard for any controls to limit exposure, such as worker protective equipment, fences, etc. Indeed, the purpose of the risk assessment is to determine the *necessity* of institutional controls. Furthermore, any institutional controls currently in place at the Facility are voluntary and self-imposed, which the Facility is not legally bound to enforce. The persistence and resistance to degradation of the chemicals of concern (dioxin and HCB) make it particularly important that no institutional controls be assumed in this human heath risk assessment because the chemicals of concern are extremely persistent and will still be present long after the Facility ceases operations, at which time the property use could change. For example, some dioxin-like compounds can persist unchanged in the environment for perhaps a century.

Following the risk assessment paradigm presented in U.S. EPA (1989a), the current human heath risk assessment follows the four steps:

- Data Assessment;
- Exposure Assessment;
- Toxicity Assessment; and
- Risk Characterization.

The following sections briefly describe the each of these four steps.

9.1 Data Assessment

U.S EPA has recently collected environmental samples of high quality to characterize the 12 separate waste management units. Although these data were specifically collected to begin the process of characterizing the units, they must be used to estimate risks. The shortcoming of using this type of data in

a human heath risk assessment, however, is that they may not represent the average concentration for exposure once they are aggregated.

An evaluation of the data quality determined it is sufficiently high for purposes of estimating risks in this human heath risk assessment. The criteria MWH used to eliminate chemicals as chemicals of concern was acceptable and routinely used to reduce the number of chemicals evaluated in a risk assessment. The exposure point was estimated as the lesser of the 95 percent upper confidence limit upper confidence limit on the mean and the maximum detected concentration according to EPA guidance (U.S. EPA 1989). I was able to reproduce all of the MWH's estimated upper confidence limit values. The only disagreement I have is with the approach MWH took in estimating the upper confidence limit for the Old Waste Pond. The approach was as stated:

> *"At the Old Waste Pond, data were stratified into two populations. The area proximate to the pond inlet has been sampled much more intensively than the rest of the Old Waste Pond. Calculating an EPC using these data as a single population would bias the calculation due to the greater sampling density in the inlet area. To avoid this bias, separate statistics have been calculated for data collected from the inlet area and the rest of the pond. The data from these two areas have been combined as a weighted average to yield a single EPC for the entire pond. The weighting was based on the size of each area; 13 acres for the inlet area, and 787 acres for the rest of the pond."*

This approach is not consistent with EPA guidance because the exposure point concentration should represent the average concentration within a reasonable exposure area. EPA (HHEM 1989) guidance states:

> *"When evaluating chemical contamination at a site it is important to review the spatial distribution of the data and evaluate it in ways that have the most relevance to the pathway being assessed. In short, consider where the contamination is with respect to known or anticipated population activity patterns. Maps of both concentration distribution and activity patterns will be useful for the exposure assessment. It is the intersection of activity patterns and contamination that defines an exposure area. Data from random sampling or from systematic grid pattern sampling may be more representative of a given exposure pathway than data collected only from hot spots."*

And:

> *"In some cases, contamination may be unevenly distributed across a site, resulting in hot spots (areas of high contamination relative to other areas of the site). If a hot spot is located near an area, which, because of site or population characteristics, is visited or used more frequently, exposure to the hot spot should be assessed separately. The area over which the activity is expected to occur should be considered when averaging the monitoring data for a hot spot. For example, averaging soil data over an area the size of a residential backyard (e.g., an eighth of an acre) may be most appropriate for evaluating residential soil pathways."*

The MWH approach calculates a *single* exposure point concentration for the entire pond, which is 800 acres. It would be impossible for a single worker to be exposed to all areas with the 800 acres on a daily basis, which is what MWH's estimated exposure point concentration for the entire Old Waste Pond assumes. While the weighted average approach can be used to correct sampling density bias, it can only be used when all sampling locations are within a reasonable exposure unit size. The fact that the inlet area (not surprisingly) is much more contaminated is ignored. For this reason, a minimum of two upper confidence limits should be estimated for: (1) the inlet area of 13 acres, and (2) the remaining 787 acres. However, the optimum and scientifically tenable approach is to subdivide the pond into a grid, with each grid being perhaps one or two acres, which is a reasonable size for a single worker to be exposed to on a daily basis (assuming the property is sold and the property continues to be used for industrial purposes in the future). However, it would not be cost effective to generate such a database because if just 3 samples were taken within each 1-acre grid (to derive an average upper confidence limit for each grid), the cost of sampling and analysis of dioxins alone could exceed $2 million. The next-best option is to estimate separate risks for the Old Waste Pond inlet area (IA) and the Old Waste Pond areas away from the inlet (AAI).

The MWH human heath risk assessment does not estimate an upper confidence limit for areas within other waste management units, based on a reasonable exposure unit. However, an evaluation of the datasets for those waste management units indicates that the other waste management units do not indicate a clear concentration gradient, as does the Old Waste Pond.

9.2 Exposure Assessment

The exposure assessment includes an evaluation of typical worker exposures under default conditions. These conditions have been well described with regard to routes, magnitude, frequency, and duration of exposure in the MWH report. The human heath risk assessment evaluated all possible worker exposures

to all the chemicals identified in the previous section. It should be noted that a human heath risk assessment is conventionally conducted under the assumption of unrestricted land use where residents could contact contamination in the future. However, this human heath risk assessment evaluated only an occupational receptor because it is unlikely residential exposures will occur, at least in the foreseeable future

This human heath risk assessment evaluated cancer risk for the following worker following routes of exposure:

- Ingestion;
- Inhalation; and
- Dermal Absorption.

However, the preliminary analysis showed that the risks associated with only one route of exposure—namely, oral ingestion—was sufficient for U.S. EPA's risk management purposes. The following default exposure assumptions were used to estimate risks:

Chronic Daily Intake (mg/kg-day) = (C x IR x CF x EF x ED)/BW x AT

CDI	=	Chronic daily intake of each contaminant (mg per kilogram body weight - day, [mg/kg-day])
C	=	Chemical concentration in contaminated medium (mg/kg)
IR	=	Ingestion Rate (mg soil/day)
CF	=	Conversion Factor (1E-6 kg/mg)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged - days).

I evaluated all the exposure assumptions and default parameters presented in the MWH human heath risk assessment, and they are reasonable and appropriately represent current and future industrial exposures at the waste management units.

9.3 Toxicity Assessment

The information presented in the preceding sections provides toxicological information with regard to the type of toxic hazards associated with exposure to the Facility contaminants. For the purpose of *quantifying* carcinogenic risk and health hazards, U.S. EPA has derived toxicity values based on the mathematical dose-response relationships. U.S. EPA categorizes chemicals based on two toxic responses; chemicals produce carcinogenic and noncarcinogenic toxic responses (IRIS 2006).

Cancer risks must be calculated based on dose-response relationship where the exposure level corresponds to a particular level of risk of developing cancer. Carcinogenic risks are quantified based on the chemical-specific carcinogenic slope factor. The cancer slope factor is directly proportional to the potency for a chemical to produce cancer; the higher the cancer slope factor, the greater the cancer potency.

Noncancer health hazards are based on a reference dose that represents the average daily dose that will not produce toxic effects when exposed over a lifetime.

9.4 Risk Characterization

Risk characterization combines the chronic daily intake with the toxicity of contaminants represented by the cancer slope factor. This provides the numerical estimates of cancer risk to workers. Risks associated with exposure to carcinogens are calculated as follows:

$$\textit{Excess Lifetime Cancer Risk (ELCR)} = \textit{LDD} * \textit{CSF}$$

Where:

ELCR =A unitless probability of an individual developing cancer over a 70-year lifetime

LDD = Lifetime daily dose (mg/kg-day)

CSF = Carcinogenic slope factor expressed in $(mg/kg\text{-}day)^{-1}$

Noncarcinogenic health hazards are estimated as a hazard quotient.

Hazard Quotient (HQ) = CDI / RfD

Where:

HQ =Hazard Quotient

CDI = Chronic daily intake (mg/kg-day)

RfD = Reference Dose (mg/kg-day)

The total cancer risk for each waste management unit is estimated by summing the risk for each chemical of concern at that waste management unit. The hazard index is the cumulative noncancer hazard at the site. The hazard index is estimated by summing the chemical-specific hazard quotients for each chemical of concern that has the same target organ.

The total cancer risk and hazard index were estimated for each waste management unit in the MWH human heath risk assessment. The results are presented in Exhibit 12. I have concluded that MWH has likely *overestimated* risks for current exposures. Based on my knowledge and observations of the Facility, very conservative assumptions were used. For example, MWH assumes a current worker will perform some job related activity in the courtyard 8 hours per day, 100 days per year, for 25 years. Based on Facility design and my observations, that is highly unlikely. Nevertheless, the estimates serve the purpose of setting a very high health protective benchmark. The true risks are likely to be significantly less than MWH estimates.

For future exposures, I have MWH's calculations and determined that it has derived reasonable cancer risk estimates and hazard indices for each waste management unit, with one exception. As previously mentioned, the Old Waste Pond is 800 acres, and the MWH human heath risk assessment calculated a single exposure point concentration. The assumption, therefore, is that a single worker could be exposed to the entire 800-acre area on a daily basis. An alternative approach should have been used in which two exposure point concentrations should have been calculated. One exposure point concentration should have been estimated for the inlet and another for the area away from the inlet. Even with this approach, the exposure units are unreasonably large. However, a careful evaluation of the data suggests that chemical concentrations within each of the two respective areas are somewhat uniform and homogeneous.

This suggests that, if the two areas were subdivided, the risks for the subdivisions would not differ significantly.

As shown in Exhibit 12, MWH estimated the cancer risk for the entire Old Waste Pond to be 1×10^{-4}. However, when two separate exposure point concentrations are used to define the inlet and the area away from the inlet, the estimated risks are different. The total risk for the 13-acre inlet area is 5×10^{-4}, and the risk for the area away from the inlet is 1×10^{-4}. (See Exhibit 13.) It can be concluded that the net effect of the "weighted average" approach implemented was to "dilute" the risks within the 13-acre inlet area. EPA risk managers may be able to use this information to develop the most cost-effective approach for mitigating risks if the Agency deems action is necessary.

DOJ also requested I evaluate the 400 Acre Waste Pond (that was not include in the MWH risk assessment). After reviewing the data, I concluded that unlike the Old Waste Pond, no clear gradient or hot spot could be identified. Therefore, I averaged the data for the entire waste pond. The calculated risk is 4×10^{-4}.

EXHIBIT 12
CANCER RISK AND HAZARD INDEX CALCULATED BY MWH

Area	Receptor	Medium	Cancer Risk	Hazard Index
Current Exposure Scenarios				
Gypsum Pile	Facility Worker	Soil	4E-05	0.2
SMUT Pile	Facility Worker	Soil	4E-07	0.0004
Courtyard	Facility Worker	Soil	1E-04	0.5
Old Waste Pond	Pond Inspector	Soil	2E-06	0.005
Future Exposure Scenarios				
Barium Sulfate Area	Industrial Worker	Soil	2E-07	0.0005
	Construction Worker	Soil	1E-08	0.001
	Subsistence Rancher	Beef	1E-09	4E-06
Gypsum Pile	Industrial Worker	Soil	9E-05	0.4
	Construction Worker	Soil	6E-06	0.6
	Subsistence Rancher	Beef	9E-06	0.04
SMUT Pile	Industrial Worker	Soil	1E-06	0.001
	Construction Worker	Soil	7E-08	0.002
	Subsistence Rancher	Beef	8E-08	0.00007
Courtyard	Industrial Worker	Soil	2E-04	1.0
	Construction Worker	Soil	2E-05	3
	Subsistence Rancher	Beef	2E-07	0.0008
Former Boron Ditch	Industrial Worker	Soil	9E-06	0.03
	Construction Worker	Soil	5E-07	0.07
	Subsistence Rancher	Beef	2E-08	0.00009
Central Ditch	Industrial Worker	Soil	8E-03	2
	Construction Worker	Soil	5E-04	6
	Subsistence Rancher	Beef	1E-04	0.009
Chlorine Ditch	Industrial Worker	Soil	2E-03	2
	Construction Worker	Soil	1E-04	5
	Subsistence Rancher	Beef	3E-06	0.002
Main Ditch	Industrial Worker	Soil	2E-03	7
	Construction Worker	Soil	1E-04	10
	Subsistence Rancher	Beef	1E-03	0.2
Western Ditch	Industrial Worker	Soil	3E-04	4
	Construction Worker	Soil	2E-05	5
	Subsistence Rancher	Beef	2E-06	0.05
Old Waste Pond	Industrial Worker			
	Deterministic	Soil	1E-04	0.3
	Probabilistic (95th percentile)	Soil	3E-05	0.2
	Construction Worker	Soil	7E-06	0.7
	Subsistence Rancher	Beef	2E-05	0.04
Landfill	Construction Worker	Groundwater	2E-07	0.06

In summary, Exhibit 13 presents the most reasonable estimates of risk for each waste management unit in descending order from highest to lowest risk for a future industrial worker.

EXHIBIT 13
HIERARCHY OF ESTIMATED CANCER RISK FOR EACH FACILITY WASTE MANAGEMENT UNIT

WASTE MANAGEMENT UNIT	ESTIMATED CANCER RISK
Central Ditch	8×10^{-3}
Western Ditch	3×10^{-3}
Chlorine Ditch	2×10^{-3}
Main Ditch	2×10^{-3}
Old Waste Pond-Inlet Area	5×10^{-4}
400 Acre Waste Pond	4×10^{-4}
Courtyard	2×10^{-4}
Old Waste Pond-Area Away From Inlet	1×10^{-4}
Gypsum Pile	9×10^{-5}
Boron Ditch	9×10^{-6}
Smut Pile	1×10^{-6}
Barium Sulfate Area	2×10^{-7}

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APPENDIX A:
CURRICULUM VITAE

Dr. RICHARD L. DeGRANDCHAMP
President and Principal Toxicologist

EDUCATION

University of Colorado, Medical School, Department of Physiology, National Institutes of Health Postdoctoral Fellow, 1988-1991

Rutgers University School of Pharmacy and Toxicology, Rutgers Postdoctoral Fellow, 1986-1988

Cornell University Medical School, Department of Pharmacology, Research Associate, 1987-1988

University of Michigan, School of Public Health, Ph.D., Toxicology, 1986

Eastern Michigan University, B.S., Biochemistry, 1978

ACADEMIC APPOINTMENTS

Adjoint Assistant Professor, University of Colorado, Health Sciences Center, School of Pharmacy, Department of Molecular Toxicology and Environmental Health, Denver, Colorado (May 1998-Current)

Adjoint Assistant Professor, University of Colorado, Health Sciences Center, Graduate Programs in Environmental Sciences, Denver, Colorado (2006-Current)

Teaching Faculty, Naval Civil Engineer Corps Officers School (CECOS), Port Hueneme, California (1996-2002)

PROFESSIONAL POSITIONS

March 1997-Current President and Principal Toxicologist, Scientia Veritas, L.L.P., Evergreen, Colorado

November 1996-March 1997 Corporate Director of Medical Toxicology and Health Sciences and Principal Toxicologist, Terranext, Lakewood, Colorado

February 1996-November 1996Director of Toxicology and Risk Assessment and Principal Toxicologist, GeoTrans Inc., Boulder, Colorado

February 1992-November 1995 Toxicology and Atmospheric Science Discipline Leader and Principal Toxicologist, PRC Environmental Management Inc., Denver, Colorado

May 1991-February 1992 Senior Toxicologist, PTI Inc., Boulder, Colorado

1984-1986 Consulting Toxicologist to EPA Neurotoxicology Division, Research Triangle Park, North Carolina

1980-1986 Consulting Toxicologist and Research Assistant, University of Michigan School of Public Health, Department of Industrial and Environmental Health, Ann Arbor, Michigan

1978-1980 Research Assistant, University of Michigan School of Public Health, Department of Water Quality

PROFESSIONAL SOCIETIES/ASSOCIATIONS

Society of Toxicology
Society For Risk Analysis
Society of Environmental Toxicology and Chemistry

EXPERTISE OVERVIEW

Dr. DeGrandchamp is an expert in toxicology and risk assessment who has more than 23 years of professional experience. He has served on numerous scientific review panels and has been a toxicological consultant for: U.S. Environmental Protection Agency (U.S. EPA), Department of the Navy (DON), Department of Energy (DOE), Department of Defense (DOD), Massachusetts Department of Environmental Protection as well as many chemical, pharmaceutical, and manufacturing companies. He has conducted or reviewed more than 300 human health risk assessments regulated under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA; Superfund); Resource Conservation and Recovery Act (RCRA); and Underground Storage Tank (UST) programs. . He has been the lead negotiator in over 150 regulatory meetings and provides expert support as well as expert witness testimony in cases involving toxicological and risk assessment issues resulting from toxic chemical exposure. He has provided expert toxicological legal support in the private sector as well as in the legal department of U.S EPA Regions 3, 5 and 8 in environmental cases involving hazardous releases. Currently he is part of the U.S. Department of Justice, Expert Witness Unit and is providing expert witness testimony pertaining to risk assessment and toxicological issues in two high profile cases. He has been a member of numerous expert scientific panels and authored risk assessment guidance documents for U.S. EPA and DOD.

TEACHING AND TRAINING EXPERIENCE

Dr. DeGrandchamp has two faculty appointments at the University of Colorado, Health Sciences Center. He is an Adjoint Assistant Professor in the School of Pharmacy, Department of Molecular Toxicology and Environmental Health (DMTEH) as well as in the Graduate Programs in Environmental Sciences in the Health Sciences Center. He is responsible developing course material and teaching toxicology and risk assessment to doctoral candidates, medical students and physicians.

Dr. DeGrandchamp taught at the Naval Civil Engineering Corps Officers School (CECOS), Port Hueneme, California. He was responsible for developing the curriculum for a risk assessment and risk management course that included detailed instruction on tiered risk-based screening, baseline risk assessment, statistical analysis, probabilistic risk assessment, and bioavailability. These three-day courses were presented nationwide at all Navy divisions.

Dr. DeGrandchamp has developed and presented a hands-on training, three-day toxicology/risk assessment workshop to risk assessors, physicians, and industrial hygienists at the Navy Environmental Health Center, Bureau of Medicine, in Norfolk, Virginia.

Dr. DeGrandchamp has instructed many U.S. EPA CERCLA and RCRA personnel, and Navy project managers in the practice and application of risk assessment, statistics, and toxicology at petroleum-contaminated sites.

LITIGATION EXPERTISE

Dr. DeGrandchamp testifies as an expert witness in toxic tort litigation and provides pretrial legal and negotiation support on issues involving toxicology, chemical fate and transport, risk assessment and risk management, and environmental laws and regulations.

Dr. DeGrandchamp served as a consulting expert for the Navy Office of General Council and the Navy Bureau of Medicine, Navy Environmental Health Center in a toxic tort suit involving more than 6,000 claimants alleging toxic exposures and medical conditions from Navy activities over a 60 year period. The defense asked for damages more than $1B. He was responsible for analyzing hundreds of historical documents and medical records generated over a period of 60 years. Additionally, he was responsible for analyzing hair sample results to determine the level of toxic metal exposures as well as the current cancer registry. Dr. DeGrandchamp's toxicological study concluded the toxicological/medical claims were without merit. Ultimately the case was dismissed based on a legal ruling.

Dr. DeGrandchamp was the expert toxicologist for the U.S. DOJ and EPA Region 3 at a PCB and dioxin contaminated site in Pennsylvania. He provided expert reports, rebuttal reports, and supplemental reports, depositions, interrogatories, and assisted U.S.DOJ in preparing for depositions. Ultimately the court ruled for U.S. DOJ's and awarded $25M. He is currently providing expert witness support in the second phase of the trial where he is developing a risk-based remedial strategy for mitigating risks to acceptable levels.

Dr. DeGrandchamp was an expert witness for U.S. DOJ in a bankruptcy trial for three sites in Pennsylvania. He was responsible for conducting a toxicological assessment to determine whether the court should secure the funds necessary to protect public health The court ruled for U.S. DOJ and required $15M to be secured for additional studies and remediation.

Dr. DeGrandchamp served as the expert toxicologist for U.S. DOJ and U.S. EPA Region 5 in a case against a steel manufacturing facility in Ohio. He was responsible for conducing toxicological evaluations for residents who live near the facility who have been eating PCB-contaminated fish. Upon completion of expert reports, a settlement was reached for approximately $25M.

Dr. DeGrandchamp provided toxicological support representing the City of Platteville regarding public health threats and risk associated with bacterial infection from livestock in the state of Colorado. He developed the overall scientific strategy for evaluating the risks and potential health threats to residents from agricultural chemicals and the mutant strain of *Escherichia Coli* 0157:H7.

Dr. DeGrandchamp has provided expert testimony in several toxic tort litigation cases for a potentially responsible party at a chrome-plating facility in Texas. His responsibilities included reviewing medical records, preparing pretrial reports, giving depositions, presentations during arbitration and mediations, preparing guardian *ad litem* documents.

Dr. DeGrandchamp has worked extensively with the U.S. Navy attorneys on diverse health and environmental issues. Dr. DeGrandchamp provided toxicological expertise and negotiation support in their Navy CLEAN program. He was a member of a multifaceted installation-wide technical panel that evaluated the legal basis for developing innovative remediation strategies to streamline the CERCLA process for all Navy bases scheduled for closure or transfer. He prepared position papers, developed the Navy's overall remediation strategy; and negotiated with local, state, and federal regulation agencies. He has been the technical expert in numerous negotiations and dispute resolution meetings.

Dr. DeGrandchamp served as the toxicological expert in a toxic tort case filed against a major pesticide manufacturer that involved domestic exposure to a pyrethroid pesticide. He prepared an expert report that was used to have the case dismissed.

Dr. DeGrandchamp provided litigation support for a toxic tort case involving a PRP in Montana involving exposure to petroleum constituents. His responsibilities included developing the overall scientific strategy and designing a sampling plan for the defense.

Dr. DeGrandchamp provided legal support for a chlorinated solvent site in Montana. He also served as the technical advisor on community relations for this project. He was responsible for interacting with the U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR).

SUMMARY OF PROJECT EXPERIENCE

Dr. DeGrandchamp was retained by the Air Force to provide toxicological expertise on health issues and risk assessment related to asbestos exposure at the Lowry Air Force Base. In addition to providing toxicological support focusing on potential exposures, he participated in developing experiments to simulate exposures, site-specific risk assessments and developing risk management strategies and responding to evolving regulatory requirements for cleanups.

Dr. DeGrandchamp routinely develops new toxicity values and information for DON for those chemicals that do not currently have U.S. EPA-verified toxicity values. To date he has developed toxicity values for more than 95 chemicals. In addition, he routinely conducts toxicological reviews to determine if U.S. EPA-toxicity values need to be modified or up-dates based on new toxicological studies.

Dr. DeGrandchamp has prepared a comprehensive guidance document on sampling and analysis and conducting risk assessments at PCB and dioxin contaminated sites for DOD. These documents were used to train Navy personnel in the environmental restoration program who are responsible for remediating Navy installations that will be returned to civilian use.

Dr. DeGrandchamp conducted a geostatistical analysis of background conditions for dioxin, furans, and PCB for the Rocky Mountain Front Range for EPA Region 8. This analysis was based on new statistical method he developed based on geochemical analyses using linear regression and principal component analysis.

Dr. DeGrandchamp developed and negotiated a geochemical method for evaluating background conditions in the state of Florida for the Department of Defense (Navy). After conducting a pilot study to demonstrate the geochemical technique can be used to define background conditions and identify chemical release areas, the Florida Department of Environmental Protection (FDEP) formally approved the technique for use on Superfund and Federal Facilities throughout Florida.

Dr. DeGrandchamp has conducted a toxicological evaluation of chemicals detected at NAS Atsugi (Japan) for the Department of the Navy. This project involved developing new toxicity values for unique chemicals and their breakdown products. This was a sole source contract resulting from specific recommendations by the National Academy of Sciences and Navy Surgeon General. Ultimately, Dr. DeGrandchamp used these toxicity values to show that contaminant levels did not pose risks to Japanese citizens.

Dr. DeGrandchamp was selected by U.S. EPA to serve on an expert External Peered Review Panel to provide technical oversight for: “Draft Human Health Risk Assessment Protocols For Hazardous Waste Combustion Facilities And Screening Level Ecological Risk Assessment Protocols For Hazardous Waste Combustion Facilities.” He was responsible for providing expertise in risk assessment and toxicology on the panel and participated in a 2-day public hearing/workshop to field and respond to public comments to prepare for finalization and release of the guidance.

Dr. DeGrandchamp the Technical Lead for EPA Region 6 in developing a new technical guidance document for RCRA sites: "Risk Management Strategy." He was responsible for all technical sections and responding to public comments.

Dr. DeGrandchamp provided EPA Region 8 with toxicological and risk assessment technical support at two RCRA sites involving hazardous solvent exposure to off-site residents. He was responsible for evaluating risks and health hazards associated with vapor entering homes from contaminated ground water ground water into nearby homes. He was responsible for evaluating current toxicological peered-reviewed toxicological studies on formaldehyde to identify current health problems in residents, determine acceptable levels of exposure, and identify homes that may require interim measures or evacuation of residents.

Dr. DeGrandchamp conducting a background analysis implementing "Procedural Guidance for Statistically Analyzing Environmental Background Data", which he authored for the Navy, at NAS Whiting (Milton Florida). This approach is being used to identify chemicals of concern for risk assessment, evaluate Applicable or Relevant and Appropriate Requirements (ARAR), and identify chemical releases. Successful completion of this project is expected to save DOD and the state of Florida $30 Million in potential remediation costs.

Dr. DeGrandchamp has conducted a comprehensive review and analysis of diverse scientific methods used to evaluate risks associated with lead exposure for DON. He is prepared a Navy position paper that evaluated all lead risk assessment model including the scientific veracity of the U.S. EPA Integrated Exposure Uptake Biokinetic Model (IEUBK) software code, the California Lead Spread Model, and the new probabilistic Integrated Stochastic Model to make recommendations for improvement. He is also developing the DON risk assessment strategy to evaluate adult lead exposure to expedite lead cleanup at closing Naval installations.

Dr. DeGrandchamp has developed a cost-effective, risk-based corrective action approach for a hazardous waste site for Lockheed Martin in Denver, Colorado. The approach incorporated Monte Carlo simulation techniques to accurately estimate actual site-specific risks based on realistic exposures. A cost-benefit matrix was being developed to guide risk management decisions.

Dr. DeGrandchamp provided technical expertise on wide-ranging issues to EPA Regions 8 and 6 RCRA and CERCLA programs. He provided toxicological and statistical support on all remedial investigations and feasibility studies conducted at the Rocky Flats Nuclear Weapons Plant (RFP) and was involved in all investigations pertaining to the analysis of human health risks resulting from chemical and radionuclide exposures. He developed data quality objectives and risk assessment methodology, statistical analysis, sampling and analysis plans, and oversaw all chemical and radiological fate and transport modeling. He compiled a database for conducting Monte Carlo simulations and provided technical review on supplemental guidance for conducting Monte Carlo simulations for EPA Region 8. He developed a cost-effective risk assessment template for RFP to streamline and provide consistency for all risk assessments.

Dr. DeGrandchamp was responsible for evaluating DOE's statistical analyses and risk assessments and ensured results were consistent with U.S. EPA, the International Commission on Radiation Protection (ICRP) and Nuclear Regulatory Commission (NRC) methodologies. He assisted EPA Region 8 in negotiating numerous disputes and was a participant in a workgroup of nationally recognized experts in binding arbitration involving statistical analyses. He was

selected as a member of an interagency committee that included the Colorado Department of Natural Resources, Colorado Department of Health, Colorado Fish and Wildlife Service, EPA Region 8, and DOE to scope, design, and implement a comprehensive installation-wide human health and ecological risk assessment for Rocky Flats.

Dr. DeGrandchamp provided scientific expertise to DOE on toxicological, risk assessment, and statistical issues at the Savannah River Site (SRS) in South Carolina. He reviewed human health risk and dose assessments conducted for numerous operable units and participated on a task force responsible for establishing background conditions. He was invited to lecture on risk assessment and statistical issues by EPA Region 4, DOE, and the South Carolina Department of Health project managers and toxicologists.

Dr. DeGrandchamp conducted numerous baseline risk assessments at Naval Air Station (NAS) Lemoore in California. These risk assessments were ultimately combined into a comprehensive installation wide risk assessment that involved fate and transport modeling of contaminants coupled with the analysis of current and potential future health risks. He was responsible for all negotiations with federal and state regulators. He successfully negotiated cost-effective management of human health risks during remedy selection by using a risk-based approach to avoid unnecessary and expensive remediation

Dr. DeGrandchamp conducted all risk assessments and coordinated feasibility studies for NAS Moffett Field in California. He carried out a detailed future land use analysis that was used to focus risk mitigation strategies based on probable future land use. The land use analysis was also used to focus human health risk assessments on realistic exposure conditions to avoid unrealistic conservative default assumptions. He negotiated all aspects of the risk assessment approach with state and federal regulatory agencies. The Navy requested Dr. DeGrandchamp to assist the Department of Justice to avert formal dispute resolution.

Dr. DeGrandchamp conducted risk assessments for NAS Alameda in California. He was responsible for developing the overall risk assessment approach and negotiating all technical aspects of the project Navy with local, state, and federal regulators. He was also tasked with preparing innovative approaches to establish anthropogenic and nonanthropogenic background conditions, preliminary remediation goals, and data aggregation to estimate exposure-point chemical doses. He was also responsible for developing a Navy policy document for risk-based corrective action (RBCA) at petroleum sites.

Dr. DeGrandchamp provided oversight to DOD for risk assessments conducted for NAS China Lake. He was responsible for implementing a risk-based cost-effective approach for remediation and alternative cleanup levels based on actual site exposures.

Dr. DeGrandchamp provided technical expertise to the Massachusetts Department of Environmental Protection for radionuclide risk assessments, compliance, and cleanup standards. He worked with the state to develop state guidance for radionuclide cleanup of all Department of Defense and Nuclear Regulatory Commission operated sites within the state.

Dr. DeGrandchamp provided EPA Region 8 with technical oversight for all remedial investigations and risk assessments for F.E. Warren Air Force Base in Wyoming and Tooele Army Depot in Utah. He conducted a risk assessment in response to an emergency exposure

condition for off-site residents at F.E. Warren AFB who were directly exposed to high concentrations of organic solvents.

Dr. DeGrandchamp led the human health and environmental risk assessment task force for EPA Region 6 in studying potential adverse health effects associated with emissions from several incinerators in Midlothian, Texas. This investigation was prompted by strong public concern about adverse health effects on humans and livestock. In this evaluation, Dr. DeGrandchamp analyzed the potential for dioxin to produce birth defects, spontaneous abortions, and other potential toxic effects.

Dr. DeGrandchamp investigated the human health risks associated with RCRA facilities in southern California. He conducted the risk assessment for the onsite human receptors as well as the surrounding community to determine the potential risks to pregnant woman from benzene, arsenic, and cadmium exposure in groundwater. He also evaluated the risks to fetuses via in utero exposure. At another RCRA facility, he conducted a risk analysis to determine potential risks associated with arsenic-laden fly ash used as landfill material.

Dr. DeGrandchamp provided oversight and technical support to the EPA Region 8 (Montana office) RCRA division for remediation of oil refineries in Billings, Montana, Mandan, North Dakota, and Commerce City, Colorado. He oversaw all phases of the RCRA process involving preliminary investigations and corrective measures studies. His developed health-protective cleanup levels, and evaluated facility permitting and remediation enforcement. Together with Colorado Department of Health officials, he worked to negotiate remediation goals and a cost settlement.

BIOMEDICAL RESEARCH

Dr. DeGrandchamp investigated the neurotoxic mechanisms associated with exposure to mercury and acrylamide. This information was incorporated into the toxicological database developed by U.S. EPA and the Occupational Safety and Health Administration to set regulations and establish safe exposure conditions for occupational workers.

Dr. DeGrandchamp investigated the neurotoxic effects of alcohol on the developing nervous system, which produces fetal alcohol syndrome. He was responsible for developing new research methodologies and approaches to investigate subtle molecular changes in the nervous system.

Dr. DeGrandchamp designed experimental paradigms to study the bioavailability of mineralogical forms of heavy metals, such as arsenic and cadmium, from mining tailings for a CERCLA site in Montana.

Dr. DeGrandchamp worked on a project for the National Institutes of Health to investigate the neurophysiological mechanisms of strychnine poisoning. In this capacity, he coordinated a team of experts and managed all technical personnel in a multifaceted research program to elucidate the steps that result in central nervous system damage.

Dr. DeGrandchamp further refined the neurotoxic esterase in vivo enzyme assay used to evaluate neurotoxic damage resulting from nerve agents and pesticides. This laboratory method has

become a standard methodology to screen neurotoxic compounds in the chemical industry and to evaluate the neurotoxic potential of chemical weapons. He also developed a correlative animal model for U.S. EPA to quantify chemical-induced neuropathies associated with exposure to pesticides and nerve agents.

PUBLICATIONS

Dr. DeGrandchamp has authored over 100 major toxicological and human health risk assessments that have undergone extensive peered-review, however, the reports could not be published due to confidentiality or proprietary information.

RISK ASSESSMENT GUIDANCE DOCUMENTS

1. DeGrandchamp, R.L (1997). Risk-based Screening Using A Back Calculating Approach Prepared for the Naval School, Civil Engineer Corps Officers (CECOS). Port Hueneme, CA

2. DeGrandchamp, R.L (1998). Developing Monte Carlo for Probabilistic Risk Assessment Prepared for the Naval School, Civil Engineer Corps Officers (CECOS). Port Hueneme, CA

3. DeGrandchamp, R.L (1998). Applying a Tiered Risk Assessment Approach. Prepared for the Naval School, Civil Engineer Corps Officers (CECOS). Port Hueneme, CA

4. DeGrandchamp, R.L (1998). Using Geostatistics in Risk Assessment. Prepared for the Naval School, Civil Engineer Corps Officers (CECOS). Port Hueneme, CA

5. DeGrandchamp, R.L (1998). Evaluating Future Land Use in Risk Assessment. Prepared for the Naval School, Civil Engineer Corps Officers (CECOS). Port Hueneme, CA

6. DeGrandchamp, R.L (1998). Applying RAGS Part C in Risk Assessment. Prepared for the Naval School, Civil Engineer Corps Officers (CECOS). Port Hueneme, CA

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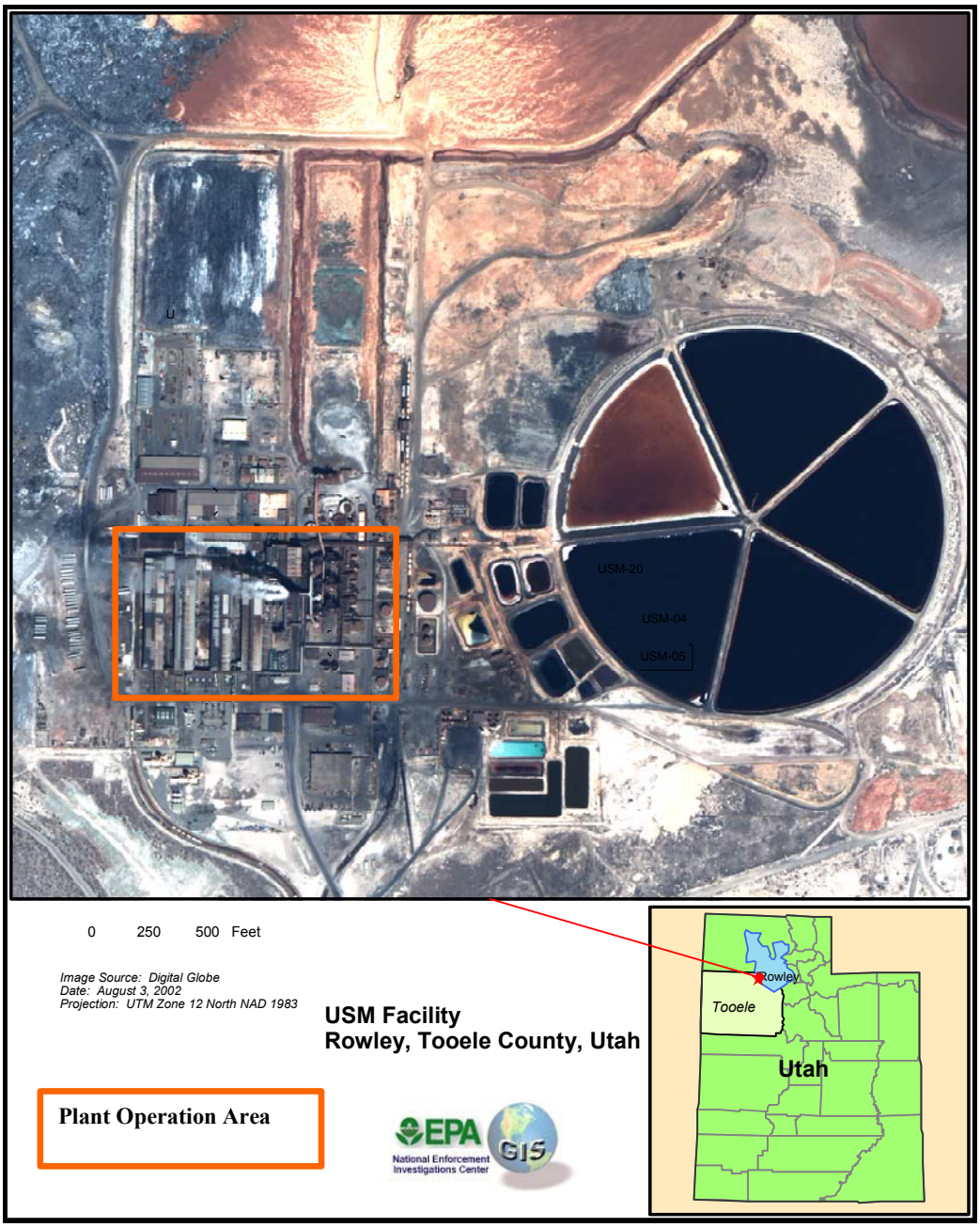
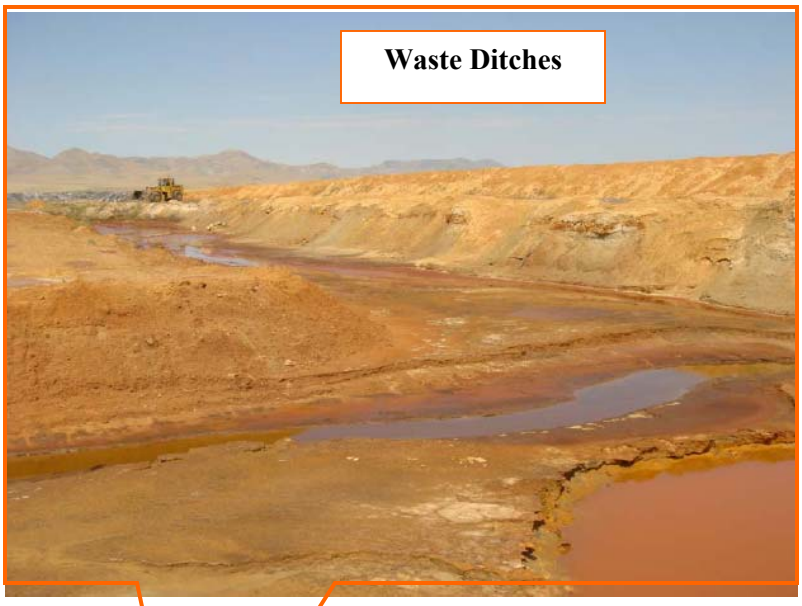
APPENDIX B.

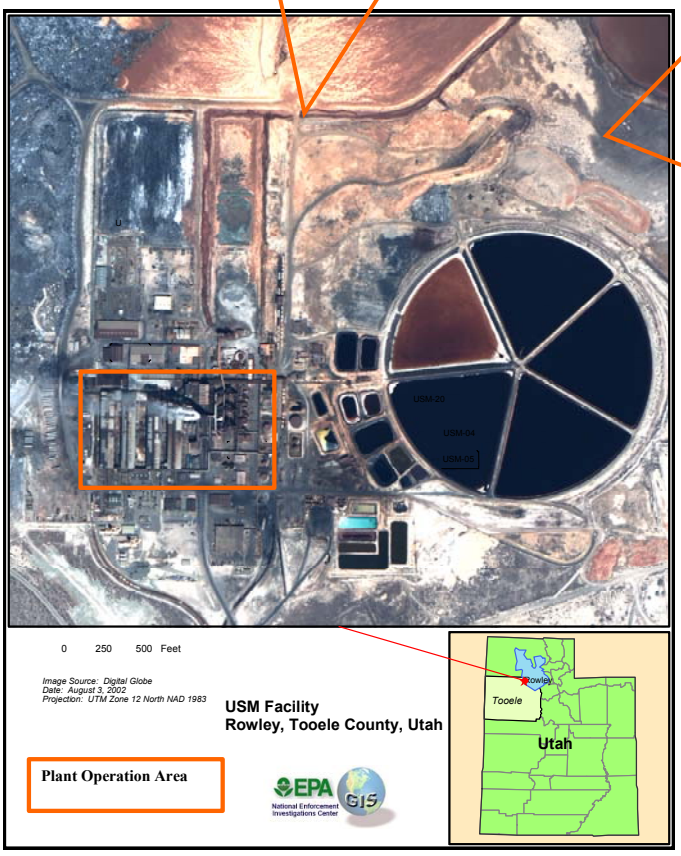
PHOTOGRAPHS

PHOTOGRAPH 1
SATELLITE IMAGE OF FACILITY (NEIC 2003)
SHOWING PLANT OPERATION AREA



PHOTOGRAPH 2
REMOTE AREAS OF THE FACILITY LOCATION OF
WASTE DITCHES AND WASTE POND





PHOTOGRAPH 3
EMPLOYEES WELDING I-BEAMS-6TH FLOOR REACTOR BUILDING
WORKING WITHOUT RESPIRATORS
PHOTOGRAPHS TAKEN JULY 26 2006

PHOTOGRAPH 4
EMPLOYEES WELDING I-BEAMS-6TH FLOOR REACTOR BUILDING WORKING WITHOUT RESPIRATORS OR GLOVES
PHOTOGRAPHS TAKEN JULY 26 2006

PHOTOGRAPH 5
EMPLOYEES WELDING I-BEAMS-6TH FLOOR REACTOR BUILDING WORKING WITHOUT RESPIRATORS AND WITH COVERALLS OPEN
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 6
WORKER WELDING I-BEAMS-6TH FLOOR REACTOR BUILDING WORKING WITHOUT RESPIRATORS GENERATING WELDING FUMES
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 7
EMPLOYEES WELDING I-BEAMS-6TH FLOOR REACTOR BUILDING WORKING WITHOUT RESPIRATORS, OPEN COVERALLS, AND CONTAMINATED UNDERSHIRT
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 8
EMPLOYEES CREATING DENSE DUST CLOUD WITH PRESSURIZED HOSE NOT WEARING RESPIRATORS
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 9
REACTOR BUILDING CONTAMINATED SURFACES-HANDS MOUTH CONTACT
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 10
CONTAMINATED SURFACES CONTACTED BY EMPLOYEES
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 11
EMPLOYEE IN MELT REACTOR BUILDING WITH SOILED HANDS NO GLOVES
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 12
TOP: EMPLOYEE REPEATEDLY TAKING GLOVES OFF AND ON
BOTTOM: WORKER NOT WEARING GLOVES
PHOTOGRAPHS TAKEN JULY 26 2006

PHOTOGRAPH 13
EMPLOYEE OPERATING SMUTTING TRUCK
EATING SANDWICH IN TRUCK AND FUMES EMITTED FROM HOLDING TANK
PHOTOGRAPHS TAKEN JULY 26 2006

PHOTOGRAPH 14
EVIDENCE OF SMOKING AND DRINKING INSIDE THE MELT REACTOR BUILDING
PHOTOGRAPH TAKEN JULY 26 2006

PHOTOGRAPH 15
REACTOR CELL REBUILD WORKERS-6TH FLOOR REACTOR BUILDING NO RESPIRATORS AND SMOKING WHILE REBUILDING REACTOR CELL
PHOTOGRAPH TAKEN SEPTEMBER 20 2006

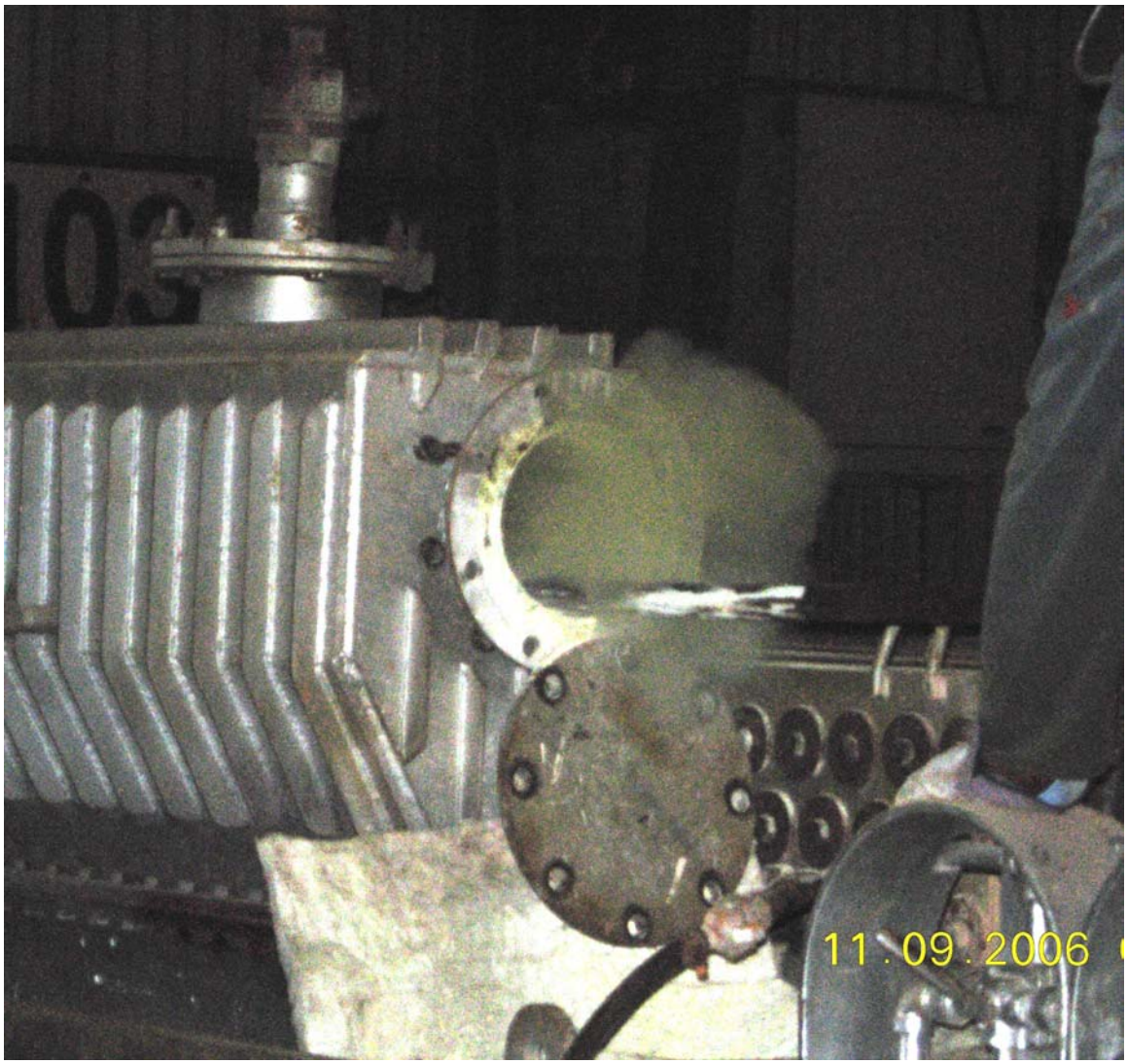
PHOTOGRAPH 16
6TH FLOOR REACTOR BUILDING-THICK CHEMICAL VAPOR/DUST RELEASE WHERE CELL REBUILD WORKERS WERE NOT WEARING RESPIRATORS AND WERE SMOKING
PHOTOGRAPH TAKEN SEPTEMBER 20 2006

PHOTOGRAPH 17
FIRST PHOTOGRAPH: EMPLOYEE REMOVING DIOXIN AND HCB CONTAMINATEED ANONDE DUST FROM GRIZZLY BOX
SECOND PHOTOGRAPH: EVIDENCE OF SMOKING INSIDE GRIZZLY BOX CONTAINMENT PIT
PHOTOGRAPHS TAKEN JULY 26 2006

PHOTOGRAPH 18
LOCKER ROOM WITH NO TOILET FACILITIES AND UNUSED SHOWERS
PHOTOGRAPHS TAKEN JULY 26 2006

PHOTOGRAPH 19
MR. CARL BURNETT-OFF GAS OPERATOR
HOLDING HIS COVERALLS AT THE END OF THE SHIFT
PHOTOGRAPH TAKEN NOVEMBER 9 2006

PHOTOGRAPH 20
ANODE DUST EMISSION FROM THE ELECTROLYTIC CELL COOLING BOX
MR. BURNETT CLEANED
PHOTOGRAPH TAKEN NOVEMBER 9 2006



PHOTOGRAPH 21
MR. CARL BURNETT CLEANING ANODE DUST EMISSION FROM THE ELECTROLYTIC CELL COOLING BOX
PHOTOGRAPH TAKEN NOVEMBER 9 2006

PHOTOGRAPH 22
MR. DERRIC SMITH-OFF GAS OPERATOR IN HIS COVERALLS AT THE END OF THE SHIFT
PHOTOGRAPH TAKEN NOVEMBER 9 2006

PHOTOGRAPH 23
MR. DERRIC SMITH-MELT REACTOR DUST AND CHLORINE EMISSIONS AS LANCES ARE LOWERED INTO MELT CELL
PHOTOGRAPH TAKEN NOVEMBER 9 2006

PHOTOGRAPH 24
MR. RAY JONES-ASSISTANT REACTOR OPERATOR AT THE END OF HIS SHIFT
PHOTOGRAPH TAKEN NOVEMBER 9 2006

PHOTOGRAPH 25
MR. RAY JONES-USING A HOSE TO CLEAN EOG PIPE CONNECTION
PHOTOGRAPH TAKEN NOVEMBER 9 2006