



STRATUS CONSULTING

Rebuttal Report of Douglas Beltman United States of America vs. Magnesium Corporation of America et al. Case Number 2:01CV0040 B

Prepared for:

U.S. Department of Justice
601 D Street, NW
PO Box 7611
Washington, DC 20044-7611

Rebuttal Report of Douglas Beltman

***United States of America vs. Magnesium Corporation of
America et al.***

Case Number 2:01CV0040 B

Prepared for:

U.S. Department of Justice
601 D Street, NW
PO Box 7611
Washington, DC 20044-7611

Prepared by:

Douglas Beltman
Stratus Consulting Inc.
PO Box 4059
Boulder, CO 80306-4059

June 8, 2007
SC11144

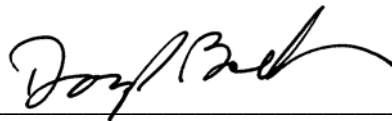
**United States of America vs.
Magnesium Corporation of America et al.**

Case Number 2:01CV0040 B

Rebuttal Report of Douglas Beltman

June 8, 2007

Prepared by:

A handwritten signature in black ink, appearing to read "Doug Beltman", written over a horizontal line.

Douglas Beltman

1. Introduction

This report is the rebuttal report of Mr. Douglas Beltman in the case *United States of America vs. Magnesium Corporation of America et al.* (Case Number 2:01CV0040 B). My expert report for this case (Beltman and Stackhouse, 2007) addresses the endangerment to the environment posed by chemical contamination at the U.S. Magnesium (USM) facility in Rowley, Utah. I concluded that the contamination at and from the USM facility poses a substantial threat to ecological resources in the area.

In this report, I provide a critical review of the expert report on ecological risks at the USM site that was prepared by Dr. William Stubblefield on behalf of USM (Stubblefield, 2007). In that report, Dr. Stubblefield concludes that chemical contamination at most areas at the site pose negligible risks to birds and mammals, that the contamination at the Old Waste Pond poses a low potential risk to shorebirds, and that contamination at the Sanitary Lagoon presents a moderate potential risk to small mammals (Stubblefield, 2007).

In summary, I disagree with the methods and conclusions of the ecological risk assessment in Stubblefield (2007), and in my opinion his methods lead to a substantial underprediction of ecological risk at the USM site. Specifically, his ecological risk assessment has the following deficiencies:

- ▶ It does not assess long-term risks at the USM site. The organochlorine contaminants at the site are long-lived and can remain in the environment for many decades. Although current industrial operations are limiting wildlife exposure at the site, the conditions at the site will very likely change, potentially in the very near future, as a result of natural processes and changes in the operational status of the facility. However, the ecological risk assessment in Stubblefield (2007) assumes that the site will indefinitely remain an industrial facility that is managed just as it is now, and that wildlife use and chemical exposure at the site will forever be limited. It therefore does not take into consideration the inevitable changes that will increase the exposure of wildlife to chemicals at the site, and therefore is not complete in its assessment of the ecological risks posed by the chemical contamination at the site.
- ▶ It uses the hazard quotient approach to calculate risk for each Waste Management Area separately, thereby resulting in greatly reduced hazard quotients compared to what they should be when site-wide exposure is considered. In essence, Stubblefield (2007) treats each area as if it were an isolated area of contamination surrounded by uncontaminated land, but this clearly is not the case. By doing so, Stubblefield (2007) substantially underestimates the true ecological risk at the site.

- ▶ It does not assess risks to plants, invertebrates, or microbes. These organisms form the bases of food chains and are fundamental components of wildlife habitat. However, Stubblefield (2007) purposely does not assess risks to these groups of organisms.
- ▶ It does not include all relevant exposure pathways in the hazard quotient calculations, either because of direct omission or a lack of chemical data. This means that these exposure pathways are assumed to be completely free from contamination in the hazard quotient calculations, which is unrealistic given the pervasive contamination at the site.
- ▶ It relies on hazard quotient calculations that are dependent on unsubstantiated assumptions about what birds and mammals at the site eat. It assumes that birds and mammals eat the different kinds of food items that are available in proportions that reflect what was collected during chemical sampling at the site, but many birds and mammals have specific dietary preferences that most likely are not reflected in the sampling that was conducted.
- ▶ It incorrectly excludes hexachlorobenzene from the calculations of 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) toxic equivalent concentrations (TECs). Hexachlorobenzene meets the criteria for inclusion in the TEC calculation approach, and should be included in the TEC calculations for the USM site. However, even if hexachlorobenzene is excluded from the TEC calculations, the concentrations of TEC without hexachlorobenzene and hexachlorobenzene alone are sufficient to cause risk at the site.
- ▶ Some of the individual toxicity values used in the hazard quotient calculations are too high, which leads to a further underestimation of risk. It also calculates hazard quotients for each chemical separately and does not consider that animals at the site are exposed to multiple chemicals simultaneously.
- ▶ It uses red-tailed hawk to represent avian predators in the hazard quotient calculations, but this species is probably exposed much less to site contaminants than other avian predators, such as the barn owl.
- ▶ Even when the calculated hazard quotients indicate ecological risk, the risk is dismissed or reduced in importance through reliance on qualitative or anecdotal information on animal populations or viability at the site that are not quantitative and cannot be relied on to make determinations about the status of populations or health of individuals at the site.

Some of these problems can be corrected in the risk hazard quotient calculations presented in Stubblefield (2007), and some are deficiencies that are inherent in the hazard quotient approach itself as applied to this site. Revising the problems that can be corrected within the hazard

quotient approach produces site-wide chronic hazard quotients that are much greater than 1 for most receptors at the site (up to 137 for no effect hazard quotients and 14 for low effect level hazard quotients). Since a hazard quotient greater than one means that predicted exposure is greater than the toxicity threshold value, these calculations show that dietary risks to birds and mammals at the site are high, even when multiple additional factors that also lead to an underprediction of risk in Stubblefield (2007) are not taken into account.

Stubblefield (2007) also presents comments on my expert report (Beltman and Stackhouse, 2007). The comments and my responses are provide in Section 3. In addition, in the last section of this report (Section 4), I provide a response to the expert report of Boyle (2007) on wildlife observations at the USM site.

2. Review of Stubblefield (2007)

Attachment A of Stubblefield (2007) is an “Evaluation of Potential Ecological Risks at the US Magnesium, Rowley, Utah Site.” Based on this risk assessment, Dr. Stubblefield concludes that most Waste Management Areas at the site pose no risk to birds or mammals. He concludes that the Old Waste Pond “appears to pose a low potential risk to shorebirds,” and that the Sanitary Lagoon presents a “moderate potential risk” to small mammals.

As stated in Stubblefield (2007), “Exposure of wildlife to contaminants does occur at the USM Site; a fact that is not in dispute” (page B-i). As this statement correctly points out, the issue at hand is not whether ecological resources at the site are exposed to chemicals, but rather the degree of risk posed by that exposure. However, in my opinion Stubblefield (2007) relies on an overall approach and specific details of implementation that lead to a significant underestimate of the real ecological risk at the site.

Stubblefield (2007) relies on the hazard quotient method to estimate ecological risks at the site, which is an approach commonly used in ecological risk assessment. In this method, a hazard quotient is calculated as the ratio of the chemical dose predicted for the site over a toxicity reference dose:

$$\text{Hazard Quotient} = \frac{\text{Predicted Exposure Dose at the Site}}{\text{Toxicity Reference Dose}}$$

When the hazard quotient is greater than one, this means that the predicted exposure dose at the site is greater than the toxicity reference dose, and therefore that animals are exposed to doses that are expected to cause toxicity. Stubblefield (2007) uses two kinds of toxicity reference doses, no observed effect levels (NOELs), which are intended to represent the highest doses at and below which there consistently is no toxicity, and lowest observed effect levels (LOELs),

which are the lowest doses at and above which toxicity consistently occurs and is therefore expected. Doses between the NOEL and LOEL are doses above which no toxicity is expected, but below which toxicity is consistently expected, and the probability for toxicity increases as the dose increases from the NOEL to the LOEL. Within this dose range between the NOEL and LOEL toxicity reference values, some organisms may experience toxicity and some may not. NOEL hazard quotients are necessarily higher than LOEL hazard quotients, since NOEL toxicity reference values are lower than LOEL values.

Stubblefield (2007) calculates hazard quotients separately for each ecological receptor, chemical, and Waste Management Area at the site. For each receptor, chemical, and Waste Management Area, the following outcomes are possible for each NOEL/LOEL hazard quotient combination:

- ▶ The calculated hazard quotient is less than one for both the NOEL and LOEL toxicity reference doses. In this case, there is no or minimal risk, since the exposure dose is less than both toxicity reference doses.
- ▶ The calculated hazard quotient is greater than one for the NOEL reference dose, but less than one using the LOEL dose. This means that the predicted exposure dose falls between the NOEL and LOEL doses, and the dose may be toxic to some organisms and not toxic to others.
- ▶ The calculated hazard quotient is greater than one for both the NOEL and LOEL reference doses. This means that exposure exceeds the concentrations that are predicted to be toxic, and therefore that there is risk from the chemical exposure.

Although the hazard quotient approach provides results in a simple format (values that are less than or greater than one), the multiple hazard quotients that are generated for each receptor, chemical, and Waste Management Area require additional interpretation in reaching overall conclusions about ecological risk at the site. In general, the more chemicals, receptors, and areas with hazard quotients that are greater than one, and the higher that the hazard quotients are, the greater the overall risk at the site. This increase in risk can be an increase in the probability that adverse toxic effects are occurring, an increase in the number and types of organisms that are experiencing the toxic effects, or an increase in the severity of the toxic effects.

The interpretation of hazard quotients also requires consideration of the assumptions used to generate the hazard quotients, and any bias toward an overestimate or underestimate of risk that is inherent in the assumptions. As I describe below, in my opinion the assumptions used in the hazard quotient approach in Stubblefield (2007) have an inherent bias toward underestimating risk – that is, they produce hazard quotients that are consistently too low. This opinion contrasts with repeated statements in Stubblefield (2007) that the assumptions are “conservative,” meaning that they tend to overestimate hazard quotients and risk.

2.1 No Consideration of Long-Term Risks at the Site

The organochlorine chemicals that contaminate the environment at the USM site, which include polychlorinated biphenyls (PCBs), chlorinated dibenzofurans, chlorinated dibenzo-p-dioxins, and hexachlorobenzene, are relatively stable in the environment and can remain in soil and sediment for many decades (Erickson, 1997; Carey et al., 1998). Therefore, an assessment of the ecological risks posed by these chemicals at the site should consider both current and potential future conditions at the site that can affect the exposure of ecological receptors to the chemicals, since these chemicals will remain in the environment for a long time. However, Stubblefield (2007) considers only current conditions at the site and assumes that those conditions will never change. This assumption is stated explicitly in several places in Stubblefield (2007):

- ▶ “The results of this assessment are predicated on the assumption that the Site will continue to be an active industrial facility into the foreseeable future (i.e., if not there are other exposure pathways that would require evaluation and that could change risk predictions)” (page B-iii).
- ▶ “Several of these locations [at the site] lack sufficient habitat and food sources to support chronic (long-term) exposures by avian and mammalian wildlife and only acute (short-term) exposures may be expected, so long as the Site remains an active industrial facility” (page 1).
- ▶ The assumption that the USM site will “continue to be an active and operational facility into the unforeseeable future” is a “key assumption underlying the expert opinion of Dr. Stubblefield regarding ecological risk at the USM facility” (page B-7, top and footnote).

I agree with Dr. Stubblefield that under current conditions at the site there are insufficient food resources or habitat for wildlife to be chronically exposed to chemicals at some of the waste areas at the site. These areas are the Active Waste Pond, SMUT Piles, Barium Sulfate Area, and Gypsum Piles (Stubblefield, 2007). For example, as Stubblefield (2007) points out, the very low pH of the water in the Active Waste Pond prevents any aquatic plants or animals from living in the water. With no aquatic food sources in the pond, wildlife food chain exposure to the chemicals in the pond is therefore limited.

Stubblefield (2007) then makes the assumption that the USM site will forever remain an active industrial facility, and those activities at the site that reduce chronic wildlife exposure at the active waste disposal areas will continue indefinitely. Based on this assumption, he concludes that there is no need to assess chronic risks in these areas (the Active Waste Pond, SMUT Piles, Barium Sulfate Area, and Gypsum Piles). Since these areas are the most contaminated areas at

the site, Stubblefield (2007) thus uses the assumption that conditions at the site will never change to avoid considering the long-term risks posed by the highest levels of contamination at the site.

However, conditions at the site will inevitably change, potentially in the very near future, in ways that will greatly increase the long-term exposure of ecological receptors to the chemicals in these areas. First, natural changes can alter conditions at the site. For example, in the 1980s the water level of the Great Salt Lake was so high that it caused a breach in the dike of the Old Waste Pond, mixing the water and sediment of the Old Waste Pond and Great Salt Lake (Olafson, 1988). Second, USM has been operating at a financial deficit for many years, and the solvency of the operation is in question (Meyer, 2007). If the facility ceases operation, scales back its operations, or changes how it uses and manages the Waste Management Areas, it will probably lead to increased chemical exposure for wildlife. For example, without the continual discharge of the low pH water into the Active Waste Pond, the pH of the pond area will gradually increase, making the area more hospitable to plants, invertebrates, and animals. Similarly, wildlife exposure to chemicals in the SMUT Piles, Barium Sulfate Area, and Gypsum Pile will gradually increase, both as the areas become more natural and as wind and water spread the contamination to adjacent areas. As Dr. Stubblefield explains it:

- ▶ “Should USM elect to discontinue operations and ‘walk away’ from the Site, then the future exposure (and risk) scenario for wildlife may well be different because habitat changes (as they attempt to revert to a more natural state) could increase colonization (and chemical exposure potential) by plants and animals” (page B-7, footnote).
- ▶ “In fact, raising the pH [of the Active Waste Pond] as a sole remedial activity at this WMA would actually increase the risk of chemical toxicity to birds because aquatic organisms, which do not now colonize the pond water and sediments, would be better able to survive in a more hospitable pH condition. . . . Therefore, the current very low pH of the active waste pond (and it is assumed in the future as an active industrial site) actually serves to eliminate the attractiveness of the pond as a dietary source of exposure” (pages B-3 and B-4).

Therefore, by predicating the ecological risk assessment on the unrealistic assumption that the USM facility operations will never change, Stubblefield (2007) ignores the long-term risk posed by much of the contamination at the site. Although I agree with Dr. Stubblefield that current activities at the site are limiting the chronic exposure of wildlife in some waste disposal areas, I disagree that the long-term assessment of ecological risks at the site should assume that these activities will continue indefinitely. In my expert report (Beltman and Stackhouse, 2007), I purposely considered potential changes in conditions at the site by comparing contaminant concentrations in soil, sediment, and surface water from across the site to toxicological benchmarks. By doing so, my analysis addresses the changes that are likely to occur in the future, for it considers the possibility that the site will revert to a more natural state.

2.2 Assessment of Risks for Each WMA and Contaminant Separately

The contamination at the USM site occurs across multiple areas where different management activities have taken place (Beltman and Stackhouse, 2007). These areas include the Old Waste Pond, Active Waste Pond, Gypsum Pile, Barium Sulfate Pile, SMUT Pile, Sanitary Lagoon, and nearby soils in more natural areas. In his assessment of ecological risks at the site, Dr. Stubblefield treats each of these areas as wholly separate, independent areas. He calculates hazard quotients and assesses ecological risk to each area, one at a time, and fails to consider the chemical exposure that receptors receive across the site as a whole. By doing so, he systematically underestimates chemical exposure for ecological receptors at the site, and thereby concludes that there is much less ecological risk than there actually is.

The risk assessment approach used in Stubblefield (2007) includes “Area Use Factors” in the chronic hazard quotient calculations to estimate how much chemical a bird or mammal at the site is exposed to. The Area Use Factor represents the portion of an animal’s diet that the animal receives from a given area of contamination. When the animal’s feeding range is equal to or smaller than the area of contamination, the Area Use Factor is 1, meaning that the organism is assumed to get all of its food from within the contaminated area. When the feeding range is larger than the area of contamination, the Area Use Factor is less than 1, meaning that the organism is assumed to get only a portion of its food from the contaminated area, and the rest of its food from clean areas. In these cases, the Area Use Factor reduces the calculated dietary exposure for an animal when only a portion of its food comes from contaminated areas. When applied appropriately, Area Use Factors can be valid components of ecological risk assessments (U.S. EPA, 1997, 1998).

However, in my opinion Stubblefield (2007) uses Area Use Factors inappropriately and in a way that leads to falsely low risk predictions. For each bird or mammal, a separate Area Use Factor is calculated for each Waste Management Area. Hazard quotients are then calculated separately for each Waste Management Area using these Area Use Factors. For birds or mammals that have ranges that are larger than the size of a Waste Management Area, this equates to an assumption that an animal comes on site and feeds in only that Waste Management Area, and feeds the rest of the time in an area free from any contamination (that is, feeds the rest of the time at some area far from the site). The same process is used to calculate hazard quotients at each of the Waste Management Areas. The birds and mammals that this assumption applies to are tree swallow,

California gull, peregrine falcon, red-tailed hawk, black-tailed jackrabbit, coyote, and badger (Table 5 of Stubblefield, 2007).¹

The effect of this approach is that chronic hazard quotients (and therefore risks) are calculated for each Waste Management Area wholly separately, as if each area existed on its own inside a larger area that is free from any contamination. This approach necessarily underestimates the exposure of (and therefore risks to) birds and mammals at the site by artificially dividing chemical exposure up according to the Waste Management Areas, and assuming that the remainder of a bird's or mammal's diet contains no contamination. This underestimation of exposure and hazard quotients necessarily leads to a conclusion in Stubblefield (2007) of there being much less risk at the site than there actually is.

For the animals with the larger home or feeding ranges at the site, the proper way to estimate their chemical exposure is to estimate all of the chemical exposure that they receive from across the site rather than to estimate exposure for each area separately (U.S. EPA, 1998). Table 1 lists the home or feeding ranges for coyote, badger, and red-tailed hawk from Stubblefield (2007) and for barn owl from the literature.² As the table shows, these receptors have feeding ranges of approximately 1 to 11.5 km². In comparing these home/feeding ranges to the contamination at the USM site, it is important to consider that the total area of contamination at the site most likely extends beyond the boundaries of the specific areas that have been sampled to date. Areas around the site can become contaminated through the deposition of wind-blown dust from the waste areas at the site and through the deposition of stack emissions. For example, the area north of the Smut Pile and west of the Gypsum Pile is not a waste disposal area, yet it is contaminated with contaminants from the site (Beltman and Stackhouse, 2007). Therefore, the actual area of contamination most likely is larger than the sum of the named areas that have been sampled to date.

Table 1. Feeding/home ranges for selected ecological receptors

Receptor	Approximate feeding/home range size	Source
Coyote	11.5 km ²	Stubblefield (2007)
Badger	2.4 km ²	Stubblefield (2007)
Red-tailed hawk	0.85 to 3.9 km ²	Stubblefield (2007)
Barn owl	6.8 km ²	Hegdal and Blaskiewicz (1984)

1. Since the Area Use Factor for deer mouse, American avocet, and sandpiper are 1 for all of the Waste Management Areas where chronic hazard quotients are calculated in Stubblefield (2007), the problem of the separate Area Use Factors for each Waste Management Area does not apply to these receptors.

2. Stubblefield (2007) did not include barn owl as a receptor in his ecological risk assessment, but it should be included as a bird that feeds on small mammals at the site in a species-specific risk assessment approach, as explained in Section 2.8.

Figure 1 shows an aerial image of the USM site with circles of areas of 3 km², 5 km², and 10 km² overlaid on the site for comparison to the home/feeding ranges listed in Table 1. Although birds and mammals do not feed in perfect circles, the figure demonstrates that the size of the USM site is roughly equal to or larger than the feeding ranges of these receptors. Therefore, an Area Use Factor of 1 should be used for these receptors in a site-wide ecological risk assessment that relies on hazard quotient calculations, as Stubblefield (2007) does.

To demonstrate the effect that Stubblefield's Area Use Factors have on the hazard quotient calculations, I used the equations and input data from Stubblefield (2007) to calculate chronic hazard quotients for dietary exposure of coyote, badger, and red-tailed hawk to 2,3,7,8-TCDD equivalents (TCDD-eq, or TEC in Stubblefield, 2007)³, except that I used Area Use Factors of one and calculated site-wide exposure concentrations in the animals' diets. I used the dioxin, furan, and PCB data in Stubblefield (2007) to calculate site-wide exposure concentrations,⁴ and used the food and sediment ingestion rates and body sizes reported in Stubblefield (2007). For purposes only of direct comparison to the hazard quotients in Stubblefield (2007), I also used his dietary toxicity thresholds, even though some of the selected dietary thresholds that he selected are too high (details provided in Section 2.7). I also did not include hexachlorobenzene in the site-wide TEC calculations, even though it should be included (details provided in Section 2.6). Table 2 shows results of the site-wide hazard quotient calculations for dietary exposure to TEC using these assumptions and compares the results with the hazard quotients in Stubblefield (2007) for the individual Waste Management Areas.

3. TCDD-eq, or TEC, is the equivalent concentration of the dioxin congener 2,3,7,8-TCDD that would produce the same dioxin-like toxicity as the mixture of dioxin-like compounds to which an organism is exposed. Further explanation is provided in Beltman and Stackhouse (2007).

4. Badger, coyote, and red-tailed hawk are all assumed to eat only small mammals in Stubblefield (2007). I calculated the site-wide TEC exposure concentrations (upper 95th confidence limit on the mean) in small mammals to be 689 ng/kg (dw) using mammalian toxic equivalency factors, and 1,079 ng/kg (dw) using avian toxic equivalency factors (not including hexachlorobenzene in the TEC concentrations). Due to time constraints, I did not calculate a site-wide TEC exposure concentration in soils without including hexachlorobenzene in the TEC calculations. Instead, I calculated a site-wide TEC soil exposure concentration (upper 95th confidence limit on the mean) that includes hexachlorobenzene in the TEC calculation of 1,700 parts per billion (ppb), then divided this value by two as an estimate of the site-wide soil TEC exposure concentration without hexachlorobenzene in the TEC calculation (producing an estimate of 850 ppb). Soil consumption contributes relatively little TEC exposure to coyote, badger, red-tailed hawk, and barn owl compared to consumption of contaminated mammals. Therefore, the exact estimate of a site-wide soil TEC exposure concentration without hexachlorobenzene has little effect on the hazard quotient calculations.

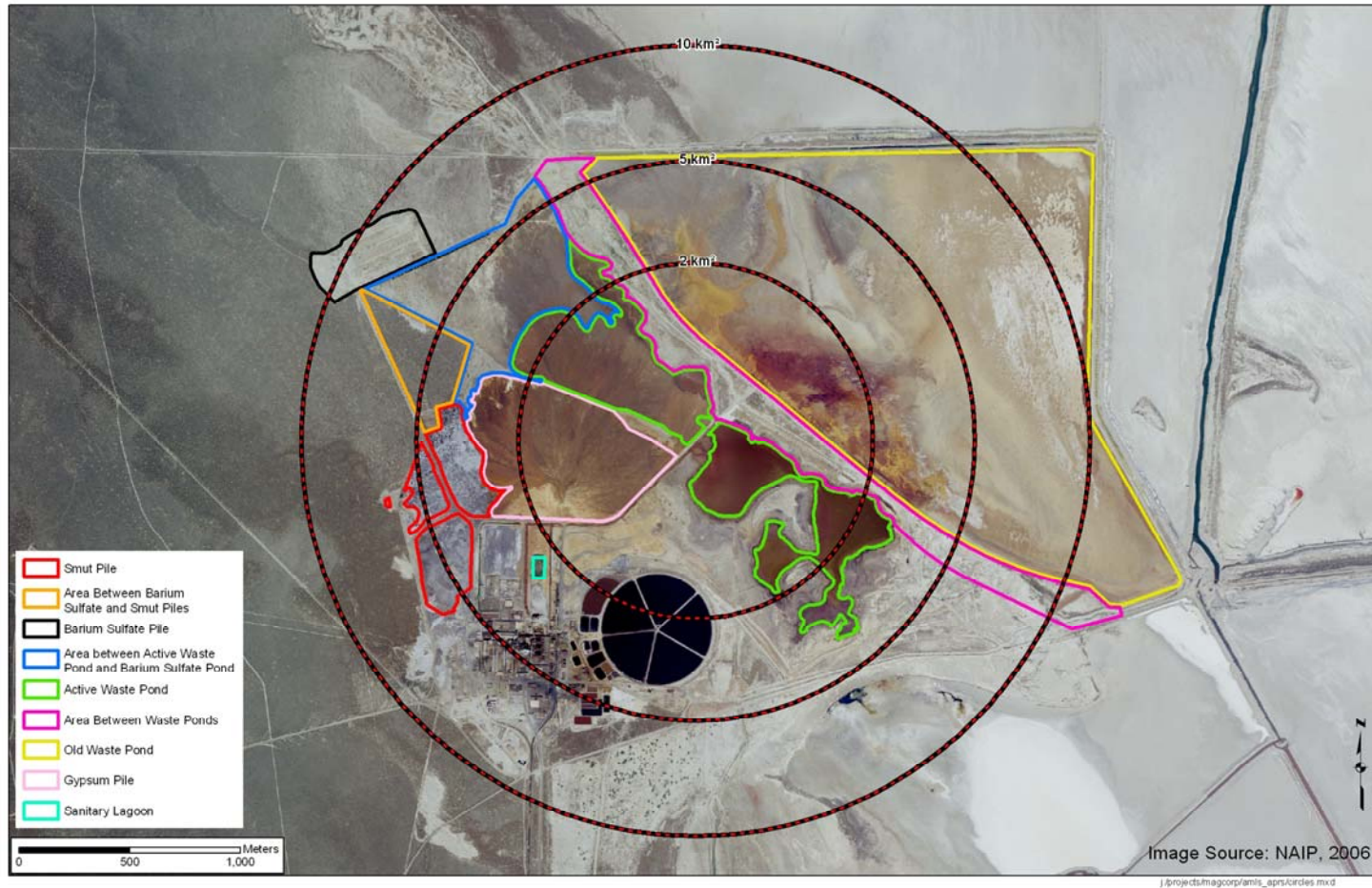


Figure 1. The USM site with circles of areas of 3 km², 5 km², and 10 km² superimposed.

Table 2. TEC hazard quotients for chronic dietary exposure to 2,3,7,8-TCDD TEC

Receptor	Hazard quotients for individual waste management areas from Stubblefield (2007)		Site-wide hazard quotients ^a	
	NOEL ^b	LOEL ^b	NOEL ^b	LOEL ^b
Badger	0.4-4.5	0.05-0.5	79	7.9
Coyote	0.1-1	0.01-0.1	76	7.6
Red-tailed hawk	< 0.01	< 0.01	4.2	0.42

a. Hazard quotients are calculated using all exposure and toxicity assumptions from Stubblefield (2007), except that Area Use Factors are set at 1 and exposure concentrations are calculated on a site-wide basis rather than separately for each Waste Management Area.

b. NOEL = no observed effect concentration, and LOEL = lowest observed effect concentration. Hazard quotients from Stubblefield (2007) are estimated from bar graphs in the report.

Table 2 shows that hazard quotients calculated separately for each Waste Management Area by Stubblefield (2007) are significantly lower than site-wide hazard quotients that consider exposure across the entire site. For example, the chronic LOEL hazard quotients for badger range from about 0.05 to 0.5 when calculated for each Waste Management Area separately, but is 7.9 when calculated on a site-wide basis, which is an increase of approximately 15 to 150 times. Similar increases in the hazard quotients occur for the other receptors on a site-wide basis. Therefore, by not considering site-wide exposure, the hazard quotients reported in Stubblefield (2007) are biased low, by magnitudes of tens to hundreds of times.

In contrast to Dr. Stubblefield's approach of calculating hazard quotients for each Waste Management Area independently of the others, in my expert report I combined the contaminant exposure data from all the areas at the site together for comparison to toxicological benchmarks.

2.3 Ecological Risks to Plants, Invertebrates, and Microbes

Plants, invertebrates, and microbes (e.g., soil bacteria) are essential components of ecosystems and of habitat for wildlife. As primary producers, plants form the base of the food chain, and they also provide vital shelter and habitat for animals. Invertebrates and microbes perform many essential ecological functions in ecosystems, including breaking down organic matter and litter, cycling energy and nutrients, and serving as food for reptiles, mammals, birds, and invertebrates. EPA guidance is clear that risks to these organisms should be considered in ecological risk assessments at hazardous waste sites (U.S. EPA, 1997, 1998).

Stubblefield (2007) purposely omits plants, invertebrates, and microbes from his ecological risk assessment. As stated on page 1, "no opinions are provided on whether chemical concentrations in soil, sediment, or surface water at the Site are posing an unacceptable risk to microbes,

invertebrates, or plants.” This omission appears to be related to the assumption described above that the site will remain an active industrial facility indefinitely.

Since only risks to birds and mammals are considered in Stubblefield (2007), he concludes that there can be no long-term ecological risks in areas where there is insufficient habitat, such as the Active Waste Pond, Gypsum Pile, Barium Sulfate Pile, and Gypsum Pile (which include the most contaminated areas at the site). However, the possibility that the contamination at the areas is in fact contributing to the lack of habitat is not considered, since risks to plants, invertebrates, and microbes are ignored. Using this reasoning, Stubblefield (2007) assumes that there are and can be no long-term ecological risks at the most contaminated areas at the site, without actually conducting any analysis.

Therefore, Stubblefield (2007) presents no opinion on the risks to plants, invertebrates, and microbes from the contamination at the site. In contrast, in my expert report (Beltman and Stackhouse, 2007) I concluded that chemical concentrations in areas at the site exceed toxicological benchmark concentrations for soil, sediment, and surface water, some of which are based on toxicity to plants, invertebrates, or microbes.

2.4 Failure to Assess all Relevant Contaminant Exposure Pathways

Exposure pathways are the different ways in which an ecological receptor can be exposed to the chemicals at a site. Exposure pathways are site-specific, since they depend on the nature and extent of contamination at a site, the chemical and biological properties of the chemicals, the kinds of ecological receptors being evaluated, and the nature of the habitat at the site (U.S. EPA, 1997, 1998). For an ecological risk assessment to be complete, all potential exposure pathways need to be included (U.S. EPA, 1997, 1998).

However, Stubblefield (2007) does not include chemical exposure from numerous contaminant exposure pathways in the hazard quotient calculations. The omissions occur in two different ways. First, several entire pathways are simply omitted from inclusion in the hazard quotient calculations. The omissions include:

- ▶ The exposure of tree swallows to contamination in brine flies is excluded. Tree swallows feed primarily on flying insects that they catch in the air (Robertson et al., 1992), but the assessment in Stubblefield (2007) only includes exposure to invertebrates that live on the ground (such as ants and spiders) in the hazard quotient calculations (Table 3 of Stubblefield, 2007).

- ▶ The only exposure pathway included for peregrine falcon is drinking water. Peregrine falcon consume mostly other birds (White et al., 2002), and the contamination of birds at the site has been documented through the elevated concentrations of dioxins, furans, PCBs, and hexachlorobenzene in the eggs of birds that nest at the site (Beltman and Stackhouse, 2007). Therefore, the hazard quotient calculations in Stubblefield (2007) ignore a relevant exposure pathway for peregrine falcon.

The second way in which Stubblefield (2007) omits relevant chemical exposures in the hazard quotient calculations is that measurement data are not available for all contaminants and exposure pathways identified in Stubblefield (2007), and where data are unavailable, it is assumed that the pathway is free from contamination. By making this assumption, the hazard quotients necessarily underestimate the chemical exposure at the site, and thereby underestimate risk.

Tables 12 through 17 of Stubblefield (2007) list the availability of chemical data that are used in the hazard quotient calculations. According to those tables, there are no data available for the following contaminant/exposure pathway combinations:

- ▶ PCBs in soil, plants, or invertebrates in the area north of the SMUT pile, west of the Gypsum Pile
- ▶ PCBs in soil, plants, or invertebrates in the area between the Old Waste Pond and the Active Waste Pond
- ▶ PCBs in plants or invertebrates in the Sanitary Lagoon
- ▶ PCBs in the Barrow Pit sediment
- ▶ PCBs, TCDD-TEC, or hexachlorobenzene in the Barrow Pit surface water
- ▶ PCBs, TCDD-TEC, or hexachlorobenzene in the Old Waste Pond surface water
- ▶ PCBs, TCDD-TEC, or hexachlorobenzene in the Active Waste Pond surface water.

The hazard quotient calculations used in Stubblefield (2007) require an input for the contaminant concentration in every exposure pathway. However, where no data are available, the calculations assume a chemical concentration of zero. Therefore, in all the cases listed above where no data are available, the hazard quotient calculations in Stubblefield (2007) are based on the assumption that there are no contaminants present. Given the extent of contamination at the site by these chemicals (Beltman and Stackhouse, 2007), this assumption leads to hazard quotients that are biased low in Stubblefield (2007). However, without data for these pathways, it is difficult to estimate the magnitude of the bias and how the hazard quotients would change as a result.

Nevertheless, it is clearly a source of an underestimation of hazard quotients and risk in Stubblefield (2007).

2.5 Unsubstantiated Exposure Assumptions

The hazard quotient approach in Stubblefield (2007) relies on specific assumptions regarding how much of each potential dietary item at the site is consumed by each of the bird and mammal receptor species being evaluated. The calculations require input variables for the fraction of each potential food item that is consumed by each receptor in each Waste Management Area, and the chemical concentrations in each of those food items. However, the assumptions in Stubblefield (2007) regarding dietary exposure to chemicals at the site are unsubstantiated, and highly dependent on the field sampling scheme used to collect samples from the field for chemical analysis (which most likely does not mimic food selection by wildlife).

Table 3 summarizes some of the dietary intake assumptions used in Stubblefield (2007). At the broadest scale, Stubblefield (2007) makes reasonable assumptions about whether each receptor is primarily a carnivore, herbivore, or insectivore. However, more detailed information about the relative amounts of each specific type of food available to each receptor is required in the hazard quotient calculations. Table 3 shows that these specific assumptions for each receptor vary from area to area in Stubblefield (2007). For example, black-tailed jackrabbits are assumed to eat 50% greasewood and 50% shadscale in the area north of the Smut Pile and west of the Gypsum Pile, 50% saltgrass and 50% shadscale in the area between the Old and Active Waste Ponds, and 33% saltgrass, 33% shadscale, and 33% Chenopodium in the Sanitary Lagoon. These differences in assumed diets are not based on any detailed studies of jackrabbit diet at the site, but rather on the samples collected at each of the Waste Management Areas for chemical analysis. Although the types of tissues collected probably reflect to some degree the types of food available, the selection process used by the samplers in the field does not necessarily mimic the selection process used by birds and mammals as they feed. Therefore, the dietary assumptions in Stubblefield (2007) are more dependent on the field collection techniques that were used than on actual wildlife diets at the site.

The specific assumptions in Stubblefield (2007) regarding wildlife dietary preferences do affect the hazard quotient calculations, because the chemical concentrations vary between the different potential food items that were measured. As an example, Table 4 compares hexachlorobenzene concentrations (upper 95th percent confidence limit on the mean, as reported in Stubblefield, 2007) between different plant and animal tissue samples used in the Stubblefield (2007) hazard quotient calculations. The table shows that the hexachlorobenzene concentrations vary considerably between different plant and animal tissues from within the same area. Differences are greatest in the sanitary lagoon, where hexachlorobenzene concentrations are 280 ug/kg in harvest mice and 8,300 ug/kg in deer mice, and range from 60 ug/kg to 32,500 ug/kg in different

Table 3. Dietary assumptions used for the hazard quotient calculations in Stubblefield (2007)

Receptor	Area	Assumed diet							
		Deer mice	Harvest mice	Invertebrates	Greasewood	Saltgrass	Shadscale	Chenopodium	Cattail
Coyote and badger	Area north of Smut Pile, west of Gypsum Pile	100%							
	Area between Old and Active Waste Ponds	100%							
	Sanitary Lagoon	50%	50%						
Black-tailed jackrabbit	Area north of Smut Pile, west of Gypsum Pile				50%			50%	
	Area between Old and Active Waste Ponds					50%		50%	
	Sanitary Lagoon					33%		33%	33%
Deer mouse	Area north of Smut Pile, west of Gypsum Pile			50%	25%			25%	
	Area between Old and Active Waste Ponds			50%		25%		25%	
	Sanitary Lagoon			50%		17%		17%	17%

Table 4. Stubblefield (2007) exposure point concentrations of hexachlorobenzene in different dietary items at the site (ug/kg dry weight)

Area	Deer mice	Harvest mice	Invertebrates	Greasewood	Saltgrass	Shadscale	Chenopodium	Cattail
Area north of Smut Pile, west of Gypsum Pile	56		440	2		10		
Area between Old and Active Waste Ponds	75		170		93	65		
Sanitary Lagoon	8,300	280	5,160		120		60	32,500

plant species. These differences mean that the specific assumptions used about how much of each of these potential dietary items a bird or mammal eats will have significant effects on the hazard quotients in Stubblefield (2007). Furthermore, we do not know the chemical concentrations in other food items consumed by wildlife but not sampled.

There are several places in Stubblefield (2007) where he acknowledges that these uncertainties about actual animal diets and chemical concentrations in the diets can affect his hazard quotient calculations:

- ▶ “In addition, TCDD-TECs and HCB concentrations were elevated in some potential food items relative to others, which could actually result in higher HQs [hazard quotients] if deer mice were to feed more on these items (the HQs cited above assume an average exposure to several possible food items)” (p. 2).
- ▶ “In other words, there is sufficient uncertainty in the deer mouse diet and variability in chemical concentrations in food items, that the HQs could be even higher than those shown in Figure 13” (p. A-35).
- ▶ “Variability in chemical concentrations between biological media (i.e., food items) and uncertainties in the dietary composition of wildlife that feed in this WMA is potentially an important uncertainty” (p. A-35).

The small numbers of samples of wildlife dietary items collected at the site, in conjunction with the lack of specific knowledge of wildlife diet at the site, means that the hazard quotient approach has an implied precision that is false. As shown in Table 4, chemical concentrations in plants and animals at the site vary greatly, yet our knowledge of what wildlife are actually feeding on is incomplete. It is, in part, for this reason that in my expert report I chose to use an

approach that does not rely on specific assumptions about animal diet (as explained in more detail in Section 3).

2.6 Failure to Include Hexachlorobenzene in TEC Concentration Calculations

Stubblefield (2007) excludes hexachlorobenzene in the calculations of 2,3,7,8-TCDD equivalent (TEC) dose concentrations at the site, and I disagree with this exclusion. Although there has been a substantial amount of research published on the toxicity of hexachlorobenzene, there has been very little discussion in the literature of whether ecological risk assessments should include hexachlorobenzene in the TEC approach (van Birgelen, 2000; Vos, 2000). However, there has been consensus in the literature on how to determine whether a specific compound should be included or excluded in the TEC approach (U.S. EPA, 2003), and I applied those consensus rules to hexachlorobenzene and concluded that it should be included (Beltman and Stackhouse, 2007). Stubblefield (2007), however, criticizes that evaluation, as described below. Regardless, it is worth pointing out that even if hexachlorobenzene is excluded from the TEC calculations, the concentrations of TEC without hexachlorobenzene and hexachlorobenzene alone are sufficient to cause risk at the site, as described in Beltman and Stackhouse (2007) and in this rebuttal report.

Attachment B of Stubblefield (2007) presents an argument as to why hexachlorobenzene should not be included in TEC calculations. I will address the points made in Appendix B in the order presented.

Stubblefield (2007) comment: There exists an extensive database of toxicology literature for HCB that is more than sufficient to support the evaluation of HCB risk at the USM Site without considering HCB in the assessment of toxicity relative to 2,3,7,8-TCDD (p. B-40).

This comment misses the point of the TEC approach. The point is to use TEC calculations to assess the *combined* toxicity of all compounds that act through the same toxic mechanism as 2,3,7,8-TCDD (U.S. EPA, 2003). Excluding hexachlorobenzene from the TEC calculations underestimates the risk from the combined toxicity of dioxin-like chemicals at the site. However, since the toxicity of hexachlorobenzene on its own has been extensively studied, we can also assess the risks from exposure to this chemical alone as another line of evidence in the ecological risk assessment for the site. Therefore, risks from hexachlorobenzene exposure at the site should be evaluated both by including hexachlorobenzene in the TEC approach and by evaluating the toxicity of hexachlorobenzene on its own, as I did in my expert report (Beltman and Stackhouse, 2007).

Stubblefield (2007) comment: The World Health Organization (WHO) expert panel that derived consensus toxic equivalency factors for dioxin, furan, and PCB congeners cautions against including hexachlorobenzene in the TEC approach (p. B-41).

The text in van den Berg et al. (2006) states: “Before inclusion in the TEF concept is considered [for hexachlorobenzene], it should be confirmed that highly purified HCB has indeed AhR agonistic properties, as contamination of HCB with PCDD and PCDFs has been reported.”⁵ The concern of the expert panel is that the hexachlorobenzene that has been used in laboratory studies that demonstrate its 2,3,7,8-TCDD-like properties may have been contaminated with dioxins or furans, and the impurities caused the toxicity observed, not the hexachlorobenzene itself. They cite information from an older study that hexachlorobenzene of high chemical quality contained 16 parts per million (ppm) of octachlorodibenzo-p-dioxin, 6 ppm of octachlorodibenzofuran, 1 ppm of heptachlorodibenzofuran, and 0.088 ppm of tetrachlorinated dibenzo-p-dioxin. Other researchers have also found dioxin and furan contamination in technical grades of hexachlorobenzene as well (Goldstein et al., 1978). Thus, the potential contamination of the hexachlorobenzene used in toxicity experiments is a legitimate concern.

However, the literature studies upon which I and others relied to conclude that hexachlorobenzene should be included in the TEC approach used highly purified grades of hexachlorobenzene which were shown to be free from dioxin or furan contamination. As described in my expert report (Beltman and Stackhouse, 2007), those studies are as follows:

- ▶ Hahn et al. (1989) clearly documents the binding of hexachlorobenzene to the Ah Receptor. Hahn et al. (1989) analyzed the hexachlorobenzene they used for contaminants and found none. They report that the hexachlorobenzene that they used contained less than 0.1 ppm (or less than 0.00001%) of dioxin, furan, or PCB congeners.
- ▶ Goldstein et al. (1986) (and references contained therein) conducted tests using hexachlorobenzene that contained less than 0.5 ppm of any dioxin or furan congener (0.00005%) and demonstrated that this pure hexachlorobenzene did indeed induce the Ah Receptor. They also conducted tests using hexachlorobenzene that was known to be slightly contaminated with dioxins and furans (0.5 ppm), and the level of Ah Receptor induction was not changed compared with their highly purified hexachlorobenzene. Therefore, the authors concluded that the binding of hexachlorobenzene to the Ah Receptor is not caused by dioxin or furan impurities in the hexachlorobenzene they used.

5. AhR is the Ah Receptor, which is the receptor in cells to which 2,3,7,8-TCDD initially binds. That binding then produces a cascade within cells that leads to toxicity. Ah Receptor binding is a hallmark of the toxicity of 2,3,7,8-TCDD (Peterson et al., 1993).

- ▶ Carpenter et al. (1985a, 1985b, 1985c) conducted a series of tests that demonstrate that hexachlorobenzene causes toxic responses similar to those caused by 2,3,7,8-TCDD. In their experiments, they started with high quality hexachlorobenzene, then purified it three times through recrystallization. They then analyzed the purified hexachlorobenzene by mass spectrometry and found no impurities. Similarly, Hansen et al. (1978) purified three times (through recrystallization) the hexachlorobenzene they used that showed toxicity similar to 2,3,7,8-TCDD in their tests, and they found no detectable impurities in the hexachlorobenzene when they analyzed it.
- ▶ Sinclair et al. (1997) also conducted tests that show that hexachlorobenzene causes toxic responses similar to those caused by 2,3,7,8-TCDD. They used a highly purified grade of hexachlorobenzene known as “100% organic analytical standard” grade, although they do not report any analytical measurements on the hexachlorobenzene.

Therefore, while the contamination of some commercially available hexachlorobenzene with dioxins and furans has the potential to confound toxicity test results, the studies I and others have relied on clearly demonstrate that hexachlorobenzene itself binds to the Ah Receptor (as does 2,3,7,8-TCDD), and that the toxicity observed in these studies is caused by hexachlorobenzene and not dioxin or furan impurities.

Stubblefield (2007) comment: Other researchers have raised concerns about including hexachlorobenzene in the TEC approach (p. B-41).

Dr. Stubblefield cites Vos (2000) and Finley (1998) as raising concerns about whether hexachlorobenzene should be included in the TEC approach.

Vos (2000) is a letter to an editor of a journal which had published an article that presented evidence that hexachlorobenzene should be included in the TEC approach. Vos (2000) argues that there are differences in the toxicity of 2,3,7,8-TCDD and hexachlorobenzene that do not justify including hexachlorobenzene in the TEC approach. The author of the first article responded (van Birgelen, 2000) that there are in fact differences, but those differences stem from the additional toxic mechanisms through which hexachlorobenzene can act. Hexachlorobenzene acts through multiple toxicity mechanisms, only one of which is the same mechanism as 2,3,7,8-TCDD (Carpenter et al., 1985a; Goldstein et al., 1986).

Finley (1998) is a memorandum written by another of USM’s expert witnesses in this case (Dr. Brent Finley). It raises objections to including hexachlorobenzene in the TEC approach that are similar to those described below.

Stubblefield (2007) comment: Hexachlorobenzene does not meet any of the four criteria for including a chemical in the TEC approach (p. B-42 and B-43).

The four criteria for inclusion of a chemical in the TEC approach are that a chemical must (U.S. EPA, 2003): (1) be structurally similar to 2,3,7,8-TCDD; (2) bind to the Ah Receptor (which is the cellular receptor to which 2,3,7,8-TCDD binds to initiate toxicity); (3) cause toxic effects similar to those caused by 2,3,7,8-TCDD; and (4) bioaccumulate. In my expert report (Beltman and Stackhouse, 2007), I compared the evidence regarding hexachlorobenzene against these four criteria and concluded that it should be included in the TEC approach. Stubblefield (2007) argues that hexachlorobenzene does not meet any of the four criteria.

First, Stubblefield (2007) argues that hexachlorobenzene is not structurally similar to 2,3,7,8-TCDD since hexachlorobenzene has one aromatic ring whereas 2,3,7,8-TCDD has two. However, the exact meaning of “structurally similar” in the context of 2,3,7,8-TCDD toxicity has not been defined (U.S. EPA, 2003), and Dr. Stubblefield’s conclusion that a compound must have two aromatic rings to meet this criterion cannot be supported. What is known is that 2,3,7,8-TCDD is planar, meaning that all of the atoms of the molecule lie flat on the same plane. Only some PCB congeners are planar, and those that are not planar (e.g., the “di-ortho substituted” congeners), show no dioxin-like toxicity and are not included in the TEC approach, even though they have two aromatic rings (U.S. EPA, 2003). Furthermore, dioxin, furan, or PCB congeners that do not have chlorine atoms on directly opposite ends of the molecule also do not exhibit toxicity similar to 2,3,7,8-TCDD are excluded from the TEC approach. Hexachlorobenzene is planar and has opposing chlorine atoms, and thus is similar to 2,3,7,8-TCDD in the two primary features that determine whether other dioxin, furan, or PCB congeners are included in the TEC approach.

Second, Stubblefield (2007) states that “the binding affinity of HCB is far less than that of every 2,3,7,8-substituted PCDD/PCDF compound” as well as other compounds, and that therefore hexachlorobenzene does not bind to the Ah Receptor. In fact, there is direct evidence that hexachlorobenzene does bind to the Ah Receptor, but that the binding is much weaker than the binding of 2,3,7,8-TCDD itself to the receptor (Goldstein et al., 1986; Hahn et al., 1989). It is precisely because of this weak binding that the toxic equivalency factor for hexachlorobenzene is so low (0.0001). Therefore, Stubblefield (2007) is correct that the Ah Receptor binding of hexachlorobenzene is low compared to dioxin and furan congeners, but he is incorrect that this means that there is no binding to the Ah Receptor and that hexachlorobenzene should be excluded from the TEC approach.

Third, Stubblefield (2007) states that the hexachlorobenzene doses required to induce the same types of toxicity caused by 2,3,7,8-TCDD are “orders of magnitude” more than the doses of 2,3,7,8-TCDD required to cause effects. I agree with this statement, for hexachlorobenzene is much less potent than 2,3,7,8-TCDD in causing dioxin-like effects. However, I disagree that this reduced potency is evidence that hexachlorobenzene does not act through a similar toxic mechanism as 2,3,7,8-TCDD. It simply means that the toxic equivalency of hexachlorobenzene is relatively low compared to 2,3,7,8-TCDD (0.0001), not that it does not cause dioxin-like

effects. For example, there are several dioxin, furan, and PCB congeners that have a low potency similar to that of hexachlorobenzene, yet they are still included in the TEC approach (van den Berg et al., 1998). Stubblefield (2007) also points out that metabolites of hexachlorobenzene can cause some types of toxicity whereas it is the parent compound 2,3,7,8-TCDD that binds to the Ah Receptor and causes toxicity, but this fact does not mean that the parent hexachlorobenzene cannot bind to the Ah Receptor and subsequently cause 2,3,7,8-TCDD-like toxicity.

Finally, Stubblefield (2007) argues that hexachlorobenzene does not meet the fourth criterion of bioaccumulation because hexachlorobenzene has a much shorter half-life in humans than does 2,3,7,8-TCDD. While it is true that hexachlorobenzene has a shorter half-life in animals than 2,3,7,8-TCDD, there is ample direct data in the literature demonstrating that hexachlorobenzene does accumulate in tissue (Beltman and Stackhouse, 2007). The fact that it has a shorter half-life than 2,3,7,8-TCDD does not mean that hexachlorobenzene does not accumulate in organisms.

In summary, Stubblefield (2007) wrongly concludes that hexachlorobenzene should be excluded from the TEC approach. However, it is noteworthy that even if hexachlorobenzene is excluded from the TEC calculations, the concentrations of TEC without hexachlorobenzene and hexachlorobenzene alone are sufficient to cause risk at the site, as described in Beltman and Stackhouse (2007) and in this rebuttal report. Nevertheless, the exclusion of hexachlorobenzene from the TEC hazard quotient calculations means that the hazard quotients in Stubblefield (2007) are biased low.

2.7 Use of Toxicity Reference Values that Are Too High

In my opinion, some of the chronic toxicity values used in Stubblefield (2007) in the hazard quotient calculations are too high. Since hazard quotients are the exposure concentration divided by the toxicity value, the use of toxicity values that are too high produces hazard quotients that are too low, and thus underestimates risk. Table 5 lists some of the toxicity values used in Stubblefield (2007), summarizes why they are too high, and presents alternative values (where possible).

For dietary exposure of birds to hexachlorobenzene, Stubblefield (2007) uses NOEL (no effect) and LOEL (low effect) values from Vos et al. (1971), in which Japanese quail were fed hexachlorobenzene. Stubblefield (2007) uses NOEL and LOEL values of 5 ppm and 20 ppm hexachlorobenzene, respectively, from the study. When normalized to ingestion rates and body weight, these values are 0.56 mg/kg/d and 2.25 mg/kg/d. However, birds at the 5 ppm dose level suffered from enlarged livers, increases in coproporphyrin production (which is a sign of

Table 5. Inappropriate toxicity values used in Stubblefield (2007) to calculate hazard quotients

Receptor and pathway	Chemical	Toxicity value used	Why value is incorrect	Correct values
Bird diet	Hexachlorobenzene	NOEL: 0.56 mg/kg/d LOEL: 2.25 mg/kg/d	NOEL value is actually identified as a LOEL value in the original paper.	NOEL: 0.11 mg/kg/d LOEL: 0.56 mg/kg/d
Mammal diet	Hexachlorobenzene	NOEL: 0.014 mg/kg/d LOEL: 0.14 mg/kg/d	The lowest dose tested in the study caused toxicity.	Cannot be determined from the study. Using these NOEL and LOEL values will underestimate risk.
Bird eggs	Hexachlorobenzene	NOEL: none LOEL: 1.5 mg/kg	The lowest dose tested in the study caused toxicity. Egg injection occurred relatively late in egg development.	Cannot be determined from the study. Using this LOEL value will underestimate risk.

porphyria)⁶, and liver lesions (Vos et al., 1971). In fact, the authors of the study state that 1 ppm is a LOEL value (Vos et al., 1971). Therefore, the 1 ppm (or 0.56 mg/kg/d) dose is actually a LOEL, not a NOEL. The true NOEL value from the study is 0.2 ppm (0.11 mg/kg/d). When the correct NOEL and LOEL values from the study are used, the hazard quotients in Stubblefield (2007) would increase by a factor of five.

The LOEL used in Stubblefield (2007) for dietary exposure of mammals to hexachlorobenzene comes from a study by Bleavins et al. (1984), and the NOEL is derived as the LOEL divided by 10. A problem with the study, however, is that toxicity was observed at the lowest dose tested in the study (which is the dose that becomes the LOEL used in the hazard quotient calculations in Stubblefield, 2007). There were no doses tested that showed no toxicity (other than the zero dose control). This means that a no-effect concentration was not determined in the study, and that toxicity may still occur at concentrations much lower than the lowest dose that was tested. This problem is termed the problem of an “unbounded LOEL,” and it means that there is a high amount of uncertainty in the LOEL value because toxicity may still occur at much lower doses that were not tested in the study (Chapman et al., 1998; U.S. EPA, 2005). However, it is difficult to correct for this problem in the hazard quotient calculations directly since the actual toxicity

6. Porphyria is a disruption in the biochemical processes that produce heme, which is a key component of hemoglobin and other enzymes, that causes a buildup of porphyrins and other intermediate compounds. In excess, porphyrins and other intermediate compounds are toxic to the organism (Hansen, 1994).

thresholds cannot be determined from the study. Nevertheless, the results of the hazard quotient calculations should be qualified in that they rely on an unbounded LOEL and therefore most likely underestimate risks. This possibility is not recognized in Stubblefield (2007) in the uncertainty analysis.

The same problem occurs with the toxicity values for hexachlorobenzene in mammal diet. Stubblefield (2007) uses NOEL and LOEL values taken from Bleavins et al. (1984). However, toxicity was observed at the lowest dose tested in the study (0.14 mg/kg/d), and therefore toxicity most likely occurs at lower doses as well. Nevertheless, the 0.14 mg/kg/d value is used by Stubblefield (2007) as a LOEL in the hazard quotient calculations without recognizing that it produces hazard quotients that are biased low.

The same problem of an unbounded LOEL exists with the study that Stubblefield (2007) uses for toxicity values for hexachlorobenzene in bird eggs. Boersma et al. (1986) dosed herring gull eggs with hexachlorobenzene and observed toxicity at the lowest dose tested (1.5 ppm). Furthermore, Boersma et al. (1986) injected the eggs on day 4 of incubation as opposed to immediately after fertilization. Since significant embryo development occurs in the first four days, injecting on day 4 tends to underestimate toxicity compared with injecting immediately after fertilization (DeWitt et al., 2005). Therefore, the 1.5 ppm value from the Boersma et al. (1986) study is most likely too high as a toxicity threshold value. Again, this problem is difficult to correct for directly in the hazard quotient calculations in Stubblefield (2007) and it is not recognized in Stubblefield (2007).

Therefore, some of the toxicity reference values used in Stubblefield (2007) are too high, some in ways that can be corrected in his hazard quotient calculations, and some in ways that cannot be corrected directly. The use of these reference values means that the hazard quotient calculations in Stubblefield (2007) are biased toward producing low hazard quotients, and thus toward underestimating risk.

2.8 Selection of an Inappropriate Species to Represent Avian Predators

Stubblefield (2007) calculates hazard quotients for individual species that are selected to represent broader groups of species that are expected to have similar chemical exposures at the site. Therefore, the selection of the species to use in the hazard quotient calculations is an important factor in the appropriateness of the risk assessment conclusions.

As noted in Stubblefield (2007), small mammals such as mice are important food items for both bird and mammal predators. Stubblefield (2007) selects the red-tailed hawk to represent birds that feed on small mammals at the site. However, Stubblefield (2007) notes that red-tailed hawks

prefer to hunt for food from perches, and given the lack of suitable perches at the site, probably would feed infrequently at the site. Based on this analysis, Stubblefield (2007) selects a very low Area Use Factor (0.05) for hawk exposure at all areas of the site, which reduces the calculated hazard quotients by a factor of 20.

While I agree that risks to birds that eat small mammals at the site should be assessed, I disagree that the red-tailed hawk is representative of these birds since there probably is limited feeding by red-tailed hawks at the site. A better species for assessing risks to carnivorous birds at the site is the barn owl. Barn owls eat mice almost exclusively (Marti et al., 2005). They also hunt by flying low over open ground rather than perching, and they hunt preferentially in open habitats (Marti et al., 2005), such as occur at and near the USM site. In addition, I observed a barn owl in a small tree along the bank of the Sanitary Lagoon during a site visit in July 2006. Table 6 presents body mass and food and soil ingestion rates for barn owls from the literature. I subsequently use these values in Section 2.10 to calculate hazard quotients for barn owls at the site to compare to the hazard quotients in Stubblefield (2007).

Table 6. Exposure parameters for barn owls

Parameter	Value	Source
Body mass	520 g	Average of values for males and females in Utah from Marti et al. (2005)
Food ingestion rate	0.033 kg food/kg body weight/day	U.S. EPA (2003), allometric equation for non-passerine birds
Soil ingestion rate	0.00066 kg soil/kg body weight/day	2% of food ingestion rate (assumed in Stubblefield, 2007)

Therefore, while I agree with Stubblefield (2007) that risks to carnivorous birds that feed on small mammals at the site should be evaluated, I disagree with his selection of the red-tailed hawk for the hazard quotient calculations, for it produces hazard quotients that are biased low because red-tailed hawks probably feed infrequently at the site. The barn owl is a better species to evaluate as representative of carnivorous birds, and in Section 2.10 I apply the method in Stubblefield (2007) to calculate hazard quotients for the barn owl.

2.9 Dismissal of Hazard Quotients Greater than One

As I described earlier, a hazard quotient greater than one means that the predicted exposure to a particular chemical is greater than a toxicity reference dose. Thus, hazard quotients that exceed one are indicative of ecological risk. The more receptors and chemicals with hazard quotients greater than one, and the greater the exceedences, the greater the risk.

Despite the biases toward producing low hazard quotients in Stubblefield (2007), there are nevertheless several hazard quotients calculated by Dr. Stubblefield that are greater than one. This means that the predicted exposure is greater than applicable toxicity reference value. However, in most cases Dr. Stubblefield dismisses the importance of these hazard quotients with conjecture or anecdotal information. Those cases are the following:

- ▶ LOEL (low effect) hazard quotients at the Old Waste Pond are 2.1 for chronic shorebird exposure to dioxin TEC, meaning that the predicted exposure is 2.1 times higher than the dose at which toxicity is expected. However, Dr. Stubblefield qualifies this result by saying that shorebirds “appear to be unlikely to chronically feed in the Old Waste Pond” (p. 2). However, this statement is not based on any data or studies on shorebird feeding at the pond, but is simply conjecture. Shorebirds have in fact been observed to feed in the Old Waste Pond, and they nest on the bed of the pond as well (Beltman and Stackhouse, 2007). Their eggs are also contaminated with dioxins, furans, PCBs, and hexachlorobenzene, and therefore are exposed to these site chemicals. Despite these facts, Stubblefield (2007) dismisses his own hazard quotient calculations for shorebirds that show that there is risk at the Old Waste Pond without any real basis.
- ▶ LOEL hazard quotients for chronic exposure of small mammals at the sanitary lagoon are 2.4 for TCDD-TEC and 3.8 for hexachlorobenzene. Stubblefield (2007) states that these hazard quotients overstate the risk to these organisms because deer mice were easily trapped in the Sanitary Lagoon during an earlier study, and that a previous qualitative survey at the site reported the deer mouse population to be “robust.” However, the studies cited by Stubblefield (2007) do not address whether there would be more deer mice present in the absence of contamination, or whether the individual mice that are present are healthy. In effect, the studies actually have very limited bearing on the question of whether small mammals at the site are at risk. Therefore, it is presumptuous to dismiss the importance of the hazard quotient calculations for deer mice based on anecdotal observations that deer mice are present and easily trapped at the site.
- ▶ Stubblefield (2007) calculates LOEL hazard quotients in bird eggs that are up to 3.5 for TCDD-TEC and 4.8 for hexachlorobenzene, meaning that chemical concentrations in the collected eggs were up to 3.5 and 4.8 times greater than concentrations expected to cause toxicity. However, he calls these results into question by stating in multiple places that a piece of eggshell fell into the sample vial of the bird egg with the highest chemical concentrations. Since the chemical analysis was intended to be of the egg contents only, Stubblefield (2007) expresses the opinion that the eggshell piece could be the source of the high chemical concentrations in the sample, and he qualifies the chemical results for this sample as “anomalously high.” Based on this, he downplays the importance of the hazard quotients for bird eggs. For example, he states on page A-15:

a piece of egg shell fell into the sample vial and was, therefore, included in the chemical analysis. The elevated total PCB and HCB concentrations associated with this egg sample may have been due to chemical adsorbed to the exterior of the egg and the embryo would not have been exposed to the reported concentration.

Stubblefield cites the field notes of his observer as the source of the information that a piece of eggshell fell into the sample in question (Robinson, 2006). However, Stubblefield (2007) does not mention that his field observer also noted that the piece of eggshell that fell into the sample jar was in fact removed from the jar by the field samplers after it fell in (Robinson, 2006). Therefore, I disagree with Dr. Stubblefield that the high hazard quotients for contamination in bird eggs should be qualified or reduced in importance because a piece of eggshell fell into a sampling jar (and was then removed).⁷

- ▶ Stubblefield (2007) also downplays his calculated hazard quotients for bird eggs that are well above one by noting a lack of observations of embryomortality and gross deformities in the eggs collected from the site. However, the egg examinations that were conducted on the collected eggs were not designed to determine whether egg toxicity was occurring, for the following reasons:
 - The eggs were at different stages of development when collected, including some that were collected within a few days of laying. Toxicity from chemical exposure typically is apparent late in the development of the egg or after hatching, and would not be observable in eggs that were collected early in incubation (Walker and Catron, 2000; Kanzawa et al., 2004).
 - Only embryo death or gross developmental deformities that can be observed with the naked eye were looked for. PCBs, dioxins, furans, and hexachlorobenzene cause many toxic effects to embryos that are not apparent to the naked eye, such as reduced growth, abnormal development of the heart, skeletal deformities, and

7. Perhaps Dr. Stubblefield meant to suggest that contaminants somehow leached from any contaminated soil on the eggshell piece or from the eggshell piece itself before the piece was removed. However, the egg sample in question contained 7,190 ppb hexachlorobenzene and 2,298 ppb PCBs, and soils in the area from which it was collected contain a maximum of approximately 100 ppb hexachlorobenzene and 0.2 ppb PCBs (Beltman and Stackhouse, 2007). Therefore, any soil inadvertently included in the sample would actually dilute the concentrations in the sample. Similarly, if the eggshell piece weighed 1% of the egg contents (entire eggshells typically weigh between 5% and 10% of egg contents, e.g., Dobbs et al., 1998; Garrison, 1999), the eggshell would have to contain 719,000 ppb hexachlorobenzene and 229,800 ppb PCBs, all of which would have to quickly leach from the shell into the sample jar contents for the eggshell piece to be the source of the chemicals measured in the egg sample. This scenario is simply not plausible.

organ malformation that can lead to death at later stages (Walker and Catron, 2000; Kanzawa et al., 2004).

Therefore, the lack of definitive observations of increased mortality or gross deformities in the eggs collected from the USM site does not mean that the chemicals in the eggs pose no risk.

Finally, Stubblefield (2007) also discounts any of his hazard quotients that are greater than one by stating repeatedly throughout his report that the hazard quotient calculations are “conservative,” meaning that they overpredict risk. However, for all of the reasons stated previously in this rebuttal, his hazard quotient approach and assumptions actually lead to hazard quotients that are biased low, meaning that they underpredict risk. Therefore, I disagree that the hazard quotients that are greater than one should be dismissed, but in fact they show that there is ecological risk from chemical exposure at the site.

2.10 Revised Hazard Quotient Calculations

Notwithstanding my comments and disagreements with the hazard quotient approach and methods presented in Stubblefield (2007), some of the problems with his hazard quotients can be corrected through revisions in the hazard quotient equations. This section revises the hazard quotient calculations that were presented in Stubblefield (2007) by using assumptions and inputs that correct for some of the deficiencies of the approach. However, it should be noted that not all of the biases of the hazard quotient approach in Stubblefield (2007) can be corrected for in the hazard quotient approach, and thus the revised hazard quotients still probably underestimate risk to birds and mammals. The revised hazard quotients also do not address risks to plants, invertebrates, or soil microbes, and do not consider risk in the future under different conditions at the site.

Table 7 compares dietary exposure hazard quotients for 2,3,7,8-TCDD TEC and hexachlorobenzene in Stubblefield (2007) with hazard quotients that I calculated using the same exposure and toxicity assumptions as Stubblefield (2007), except:

- ▶ Exposure is assumed to occur on a site-wide basis, and Area Use Factors of one are used (as described in Section 2.2)
- ▶ Hexachlorobenzene is included in the TEC calculations
- ▶ The correct NOEL and LOEL values for hexachlorobenzene in bird diets from the Vos et al. (1971) study are used.

Table 7. Hazard quotients for selected receptors and chemicals^{a,b}

Receptor	Hazard quotients in Stubblefield (2007)		Revised hazard quotients	
	NOEL ranges	LOEL ranges	NOEL	LOEL
Chronic exposure to 2,3,7,8-TCDD TEC in diet				
Tree swallow	0.05	< 0.01	0.95	0.1
Red-tailed hawk	< 0.01	< 0.01	6.1	0.6
Barn owl	Not calculated	Not calculated	7.3	0.7
Badger	0.5-5	0.05-0.5	137	13.7
Coyote	0.1-1	0.01-0.1	131	13.1
Black-tailed jackrabbit	< 0.01-0.5	< 0.01-0.05	10	1.0
Chronic exposure to hexachlorobenzene in diet				
Tree swallow	< 0.01	< 0.01	0.77	0.15
Red-tailed hawk	< 0.01	< 0.01	2.6	0.5
Barn owl	Not calculated	Not calculated	3.1	0.6
Badger	0.2-1.5	0.02-0.15	38	3.8
Coyote	0.1-0.4	0.01-0.04	31	3.1
Black-tailed jackrabbit	< 0.01-1.0	< 0.01-0.1	4.1	0.4

a. The revised hazard quotients used the assumptions of Stubblefield (2007) except for the following revisions: (1) chemical exposure is calculated on a site-wide basis, not area-by-area; (2) hexachlorobenzene is included in the TEC calculations; (3) the dietary hexachlorobenzene NOEL and LOEL values for birds are revised per the discussion in Section 2.7.

b. Site-wide exposure point concentrations were calculated as follows (all are the upper 95th percent confidence interval on the mean) for TEC: soil = 1,705 ng/kg; mice = 1,187 ng/kg (mammal TEFs) and 1,577 ng/kg (bird TEFs); brine flies = 19 ng/kg (bird TEFs); plants = 182 ng/kg (mammal TEFs). For hexachlorobenzene: soil = 16.1 mg/kg; mice = 5.0 mg/kg; brine flies = 0.02 mg/kg; plants = 1.2 mg/kg.

Note that I did not re-calculate dietary hazard quotients for deer mice, spotted sandpipers, or American avocet, which are all species for which an Area Use Factor of one was used in Stubblefield (2007) and for which hazard quotients were already reported as greater than one.⁸

As the results in the table show, revising these three factors produces site-wide chronic hazard quotients that equal or exceed one for all of the receptors listed. NOEL hazard quotients for exposure to 2,3,7,8-TCDD TEC range from 1.0 (for tree swallow) to 137 (for badger). LOEL hazard quotients for 2,3,7,8-TCDD TEC exposure are up to 13.7 (for badger), and equal or

8. Those hazard quotients were up to 28 (LOEL) and approximately 4 (LOEL) for deer mice in the Sanitary Lagoon and 21 (NOEL) and 2.1 (LOEL) for spotted sandpiper, and 1.5 (NOEL) and 0.15 (LOEL) for American avocet in the Old Waste Pond (Stubblefield, 2007).

exceed 1 for black-tailed jackrabbit and coyote as well. For dietary exposure to hexachlorobenzene (alone), NOEL hazard quotients are up to 38 (for badger) and are greater than 1 for red-tailed hawk, barn owl, coyote, and black-tailed jackrabbit. LOEL hazard quotients are greater than 1 for badger, coyote, and black-tailed jackrabbit. NOEL hazard quotients greater than one mean that dietary doses are greater than safe concentrations, and LOEL hazard quotients greater than one mean that dietary doses are greater than concentrations that are predicted to cause toxicity.

Table 7 presents revised dietary exposure hazard quotients only for hexachlorobenzene and TEC. Based on Stubblefield (2007), bird and mammal dietary exposure to total PCBs and arsenic is as high or nearly as high as exposure to TEC and hexachlorobenzene in some areas, and therefore revised hazard quotients for these chemicals are also probably much greater than one. Furthermore, as described previously the revised hazard quotient calculations in Table 7 also do not take into account the effects of simultaneous exposure to multiple contaminants, do not assess risks to plants, invertebrates, or microbes, are based on incomplete contaminant data for exposure pathways, and are based on unbounded LOELs as toxicity values. All of these issues lead to an underprediction of risk using the hazard quotients summarized in Table 7. Nevertheless, the revised NOEL and LOEL hazard quotients for chronic dietary exposure of birds and mammals at the site are much greater than one, for multiple receptors, and for multiple chemicals. Therefore, dietary exposure to chemicals in the food chain at the site is predicted to cause toxicity to birds and mammals that feed at the site, and the contamination at the site poses substantial ecological risk.

2.11 Conclusions

In my opinion, the ecological risk assessment in Stubblefield (2007) underestimates ecological risk at the USM site for the following reasons:

- ▶ It assumes that conditions at the USM site will never change. Based on this assumption, chronic risks to wildlife from potential exposure at the most contaminated areas of the site are not addressed. However, conditions at the USM site are very likely to change as a result of natural processes and changes in the operational status of the facility (Section 2.1).
- ▶ It quantifies risk for each Waste Management Area separately, thereby greatly reducing the calculated hazard quotients over what they should be when site-wide exposure is considered. In essence, Stubblefield (2007) treats each area as if it were an isolated area of contamination surrounded by uncontaminated land, but this clearly is not the case (Section 2.2).

- ▶ It does not assess risks to plants, invertebrates, or microbes, which form the base of the food chain and are fundamental components of wildlife habitat (Section 2.3).
- ▶ It fails to include all relevant exposure pathways in the hazard quotient calculations, either through direct omission or because of a lack of chemical data. This means that these exposure pathways are assumed to be free from contamination, which is unrealistic given the pervasive contamination at the site (Section 2.4).
- ▶ The hazard quotient calculations are based on unsubstantiated assumptions about what birds and mammals at the site eat. The calculations assume that birds and mammals eat food in proportions that reflect what was collected during sampling at the site, but many birds and mammals have specific dietary preferences that most likely are not reflected in the sampling that was conducted (Section 2.5).
- ▶ Hexachlorobenzene is incorrectly excluded from the calculations of 2,3,7,8-TCDD TECs. Hexachlorobenzene meets the criteria for inclusion in the TEC calculation approach (Section 2.6).
- ▶ Some of the individual toxicity values used in the hazard quotient calculations are too high, which leads to a further underestimation of risk (Section 2.7).
- ▶ It uses red-tailed hawk to represent avian predators in the hazard quotient calculations, but this species is probably exposed much less to site contaminants than other avian predators, such as the barn owl (Section 2.8).
- ▶ Even when the calculated hazard quotients predict risk, they are dismissed or reduced in importance through reliance on qualitative or anecdotal information on animal populations or viability at the site that have no basis in fact (Section 2.9).

Stubblefield (2007) recognizes some of these shortcomings in the hazard quotient calculations in the discussion of uncertainties, but wrongly concludes that, despite these shortcomings, the hazard quotient calculations are “conservative,” meaning that they tend to overpredict risk. In fact, the ecological risk assessment in Stubblefield (2007) underestimates ecological risk at the site, for the reasons described above. The calculations of the revised hazard quotients that I presented in the previous section demonstrate this fact, even though they can account for only some of the assumptions that produce low biases in the Stubblefield (2007) hazard quotients. The revised hazard quotient calculations presented in Section 2.10 show that addressing only some of these issues produces hazard quotients that are much greater than one for many of the birds and mammals at the site, and many of the hazard quotients are much, much greater than one. Since the revised hazard quotients are much greater than one for multiple receptor species (which are

each intended to represent larger groups of species), and for multiple chemicals, these results demonstrate that the contamination at the site poses a high ecological risk.

3. Response to Dr. Stubblefield's Comments on My Expert Report

In this section I provide responses to the comments made in Stubblefield (2007) regarding my expert report titled "Environmental Endangerment at the U.S. Magnesium Facility, Rowley, Utah" (Beltman and Stackhouse, 2007).

Stubblefield (2007) raises concerns about the general approach I used and about some specific aspects of my methods, results, and conclusions. The general concerns are (verbatim):

- ▶ EPA ecological risk assessment guidance was not followed
- ▶ Benchmarks lack relevance to the USM site
- ▶ Technical concerns with analysis procedure aspects
- ▶ Facility management goals for active industrial facility ignored
- ▶ Important site-specific evidence was not considered
- ▶ Uncertainties were not recognized resulting in a biased presentation.

My responses to these general concerns are presented here. My responses to specific concerns raised in Stubblefield (2007) are either included in the responses in this section or were addressed in the previous section.

3.1 EPA Ecological Risk Assessment Guidance

Stubblefield (2007) raises the concern that the technical analysis in my expert report does not follow EPA guidance for conducting ecological risk assessments. The aspects he cites as being inconsistent with the guidance are that I used toxicological benchmarks to compare to chemical concentrations at the site, that the analysis I did constitutes a "screening-level" ecological risk assessment which should not be used for decision-making, and that I did not use site-specific assumptions in estimating chemical exposure at the site.

First, I am very well versed in and knowledgeable about EPA's ecological risk assessment guidance. Prior to my current position I was an ecologist in the Superfund office of EPA

Region 5, which includes Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin.⁹ During my tenure there, I authored ecological risk assessment guidance for Superfund sites in the region; provided training on conducting ecological risk assessments at Superfund sites to site managers, EPA management, and State waste management agencies; participated in national meetings and forums with other Superfund ecological risk assessors from across the country to discuss ecological risk assessment methods and guidance; participated in regional and national workgroups on defining and dealing with ecological risks from contaminated sediment; and served as the co-chair of the regional Biological Technical Assistance Group, which is a group of scientists from state and federal agencies within the region that provides technical input on ecological risk issues to Superfund site managers. I also wrote, conducted, or reviewed ecological risk assessments at over 60 Superfund sites in the region.

One of the main points in Stubblefield (2007) is that the analysis that I conducted is a screening-level risk assessment, which is a preliminary assessment of risks at a site and typically is not adequate as the basis for concluding that a site poses ecological risk (U.S. EPA, 1997, 2001). A screening-level risk assessment is often the first step in the ecological risk assessment process, and it is used to screen out those sites, contaminants, or exposure pathways that it can safely be assumed pose no risk (U.S. EPA, 1997, 2001). The key feature of a screening-level risk assessment is that it compares the maximum chemical exposure concentrations at a site against minimum toxicity thresholds (U.S. EPA, 1997). When this comparison shows that there is no risk, it can be safely concluded that the chemical, pathway, or site in question poses no risk and requires no further evaluation. When a maximum exposure concentration is greater than the lowest toxicity threshold, then additional risk assessment work is necessary to conduct a more detailed assessment of risks (U.S. EPA, 1997, 2001).

Stubblefield (2007) is incorrect that the assessment I conducted is a screening-level assessment. In the assessment I conducted, I based my conclusions on a comparison of the entire range of chemical concentrations measured at the site against wide ranges of toxicity thresholds. I did not merely compare the highest concentrations at the site against the lowest toxicity values from the literature, and my conclusions were not based on such a comparison. This key fact clearly distinguishes my analysis from a screening-level risk assessment.

Stubblefield (2007) also suggests that the analysis I conducted is a screening-level assessment because I relied on a comparison of a wide range of site chemical concentrations against ranges of toxicity reference values, rather than calculating single-value hazard quotients. However, screening-level risk assessments typically rely on hazard quotients, but the quotients are between the highest possible exposure concentrations and the lowest available toxicity thresholds (U.S. EPA, 1997). Although the use of hazard quotients is quite common for more detailed risk

9. A more complete description of my qualifications as an expert in this matter is included with my expert report (Beltman and Stackhouse, 2007).

assessment as well, EPA guidance does not specify that detailed risk characterization must be conducted using hazard quotient equations, and other approaches are possible (U.S. EPA, 1997, 1998). The guidance recognizes that different approaches are appropriate under different situations: “It [ecological risk assessment] is a flexible process for organizing and analyzing data, information, assumptions, and uncertainties to evaluate the likelihood of adverse ecological effects” (U.S. EPA, 1998).

In fact, the analysis that I conducted and the analysis in Stubblefield (2007) are similar in that both rely on a comparison of site chemical concentrations against values from the literature that are intended to represent concentrations that are toxic. This comparison between chemical contamination at the site and toxicity threshold levels forms the basis of any ecological risk assessment that relies primarily on site chemistry data to evaluate risks (U.S. EPA, 1997, 1998). A key difference in the two approaches is that I did not attempt to derive single-point exposure concentrations or to select single-point toxicity reference values for the comparison, whereas Dr. Stubblefield did. In reviewing the available data for the site, I considered several different ways to evaluate the site chemistry data, including the hazard quotient method, and I considered the strengths and limitations of the data that are available for the site. I concluded that the risks from chemical contamination at the site can be assessed by comparing the full range of site concentrations against a broad array of toxicological reference values or benchmarks. This approach requires far fewer specific assumptions about receptor diets, contamination in the diets, how the diets or contamination can change over time, and on exactly what literature toxicity study best represents the conditions at this site. I chose not use a hazard quotient approach because it requires many data inputs and assumptions to capture completely all of the exposure pathways that occur at the site, and as described above in Section 2, there are not sufficient data available for the site to quantify all of the exposure pathways and define the dietary habits of the receptors on the site. Such an approach necessarily includes many uncertainties and assumptions, gives a false sense of precision to the risk assessment, and inevitably underestimates risk at the site. The hazard quotient approach is essentially a detailed model of exposure and toxicity for the site, and as stated in EPA guidance on ecological risk assessment, “Because models simplify reality, they may omit important processes for a particular system and may not reflect every condition in the real world” (U.S. EPA, 1998).

Dr. Stubblefield himself recognizes the uncertainties of the hazard quotient calculations in several places in his report (Stubblefield, 2007):

- ▶ In describing the results of the hazard quotient calculations for shorebirds feeding at the Old Waste Pond, he states “However, it must be emphasized that actual bird usage of the Old Waste Pond has not been quantified in anyway and the potential risk could be higher if it were determined that shorebirds *chronically* probe in sediment with elevated TCDD-TECS or HCB, particularly in the old inlet area” (p. A-44, emphasis in original).

- ▶ “The LOAEL-based HQs [hazard quotients] of 1-2 for the spotted sandpiper are on the threshold of being of concern, where refined exposure estimates (either higher or lower) would have a significant influence on estimating whether a potential risk exists for shorebirds at this location” (p. A-45).

In my opinion the data available for the site are sufficient for the approach that I used in my assessment. In my assessment, I compared all of the available site contamination data against a series of toxicological benchmarks from the literature. Those benchmarks included benchmarks for soil and sediment (which address direct toxicity to plants and invertebrates as well as dietary toxicity to birds and mammals through the food chain), mammal diet, bird diet, and bird eggs. The approach I used also considers ecological risks in the future as site conditions change. Based on these comparisons, it is evident that chemical concentrations at the site, and particularly at the most contaminated areas (the Active Waste Pond, Old Waste Pond, and Gypsum Pile), far exceed toxicity threshold concentrations. Because the contamination at the site is so high, the more general approach that I used is sufficient to reach a conclusion that contamination at the site poses a risk to ecological receptors, and a detailed model of exposure and toxicity is not necessary.

In summary, the approach I used is not a screening-level risk assessment as Dr. Stubblefield claims, and in fact it is a better and more reasonable approach given the available data and the high degree of contamination at the site. The hazard quotient approach that Dr. Stubblefield uses gives a false appearance of precision to his risk calculations and underestimates risks at the site.

3.1.1 The use of toxicological benchmarks

In my assessment, I termed the toxicity values that I used to compare to chemical concentrations at the site “benchmarks.” The values I used represent chemical concentrations in soil, sediment, water, bird diet, and mammal diet that are either safe, cause a low level of toxicity, or cause a high level of toxicity.¹⁰ As I describe in more detail in Beltman and Stackhouse (2007), these benchmarks were developed by various state, federal, and international agencies for use in evaluating the level of environmental contamination at sites. Some of the soil and sediment benchmarks I used are based on direct toxicity to organisms that are in contact with the soil or sediment (plants and invertebrates), and some are based on food chain effects to birds and mammals. The bird diet and mammal diet benchmarks were developed to determine the level of risk associated with contamination in bird and mammal prey items.

10. I used benchmarks only to assess the contamination of hexachlorobenzene, dioxins, furans, and PCBs at the site. The discussion presented here does not apply to the issue of the acidity of the Active Waste Pond.

Dr. Stubblefield criticizes the use of these benchmarks because they do not take into account any site-specific information and are not directly relevant to the conditions at the site. There are several points to respond to in this criticism. First, as stated above there are no site-specific data on toxicity that can be applied here. In fact, Stubblefield (2007) also uses input values for calculating wildlife dietary exposure that are either from the literature or are simply assumed, and none are site-specific. For example, he calculates most of the values for how much food and water an animal at the site drinks in a day using generic equations from the literature that are based only on how much an animal weighs (for which he also uses values from the literature that are not site-specific). Similarly, the toxicity reference values in Stubblefield (2007) are from the literature and are not specific to the site, and some of the studies he uses for toxicity reference values also form the basis of the benchmark values that I rely on. For example, I used bird diet toxicity benchmarks for 2,3,7,8-TCDD TEC from the U.S. EPA (1993) and Canadian Council of Ministers of the Environment (CCME, 2003) that are based on a pheasant dietary toxicity study by Nosek et al. (1992), and Stubblefield (2007) uses the same values from the Nosek et al. (1992) study for his bird diet toxicity thresholds. Therefore, while it is true that the benchmarks that I used are not site-specific, there in fact are no site-specific values to rely on, and Stubblefield (2007) also uses values from the literature that are not site-specific.

Second, one of my objectives was to evaluate the potential toxicity of soils and sediments across the site, both in areas where there currently is habitat and in areas where there is not, but will or could be in the future. The purpose of my analysis was to consider risks at the site as conditions change in the future, either because of natural changes (e.g., rises in the Great Salt Lake level) or changes in the facility operations that could allow increased wildlife exposure at the site, such as the facility shutting down, scaling back its operations, or changing how it uses the Waste Management Areas. To evaluate food chain risks in areas where there currently are no or limited data on contaminants in the food chain, soil or sediment-based toxicity thresholds or contamination benchmarks are the only option. The benchmarks I selected are based on substantial efforts by the authors in compiling, reviewing, and synthesizing data from the original literature and are intended to be widely applicable under many different site conditions. Further refinement of site-specific soil and sediment toxicity values is possible through site-specific studies of chemical toxicity, bioavailability, and accumulation, but given that site soil and sediment concentrations are so much higher than the available benchmarks, such refinement is not necessary to reach a conclusion about risks at the USM site.

3.1.2 Technical concerns with analysis procedures

Stubblefield (2007) also criticizes two specific aspects of the method I used:

- ▶ He criticizes the fact that I included hexachlorobenzene in the calculation of 2,3,7,8-TCDD TEC concentrations in samples. I addressed the specifics of Dr. Stubblefield's opinions, and my responses, in Section 2.6.
- ▶ He criticize the bird egg toxicity values that I used for 2,3,7,8-TCDD TEC as being too low.

The bird egg toxicity values for 2,3,7,8-TCDD TEC that I used came from a study by Hoffman et al. (1998) on American kestrels in which kestrel eggs were injected with dioxin-like chemicals and monitored for toxicity. Based on the results reported in Hoffman et al. (1998), I selected a value of 23 parts per trillion (pptr) TEC¹¹ as a low effect toxicity level and 230 pptr TEC as a more severe effects level for comparison to the TEC concentrations in bird eggs at the site. Dr. Stubblefield raises two concerns with the selection of the 23 pptr value: (1) there was not a clear dose-response relationship for the toxic effects observed at 23 pptr; and (2) it ignores the results of tests reported in the same study on the toxicity of PCB congener number 77, which showed a much higher LOEL value of 5,000 pptr TEC.

Figure 2 plots the mean data from the Hoffman et al. (1998) paper for reductions in liver weight and increases in edema or malformation. As shown in Figure 2, liver weight and percentages of edema or malformation show clear dose-response relationships. Starting at the 23 pptr dose, liver weight decreases consistently with increasing dose, and the percent of embryos or hatchlings with edema or malformations increases consistently with increasing dose. Both of these adverse effects thus show consistent trends with TEC dose, starting at 23 pptr. At the 23 pptr dose, the difference in liver weight is statistically different from controls, whereas the percentage with edema or malformations is not. The lack of statistical significance in edema or malformations at the 23 pptr dose is probably because of small sample sizes used in the study. Based on these clear trends, and the fact that total embryo weight and femur length were statistically significantly reduced at 23 pptr compared to controls, I selected the 23 pptr dose as a low effect dose. In my opinion, this selection is reasonable.

11. In the Hoffman et al. (1998) study, American kestrel eggs were dosed with PCB 126. The TEC concentrations reported here are the concentrations of PCB 126 used in the study times 0.1, which is the consensus toxicity equivalency factor for PCB 126 relative to 2,3,7,8-TCDD (van den Berg et al., 1998).

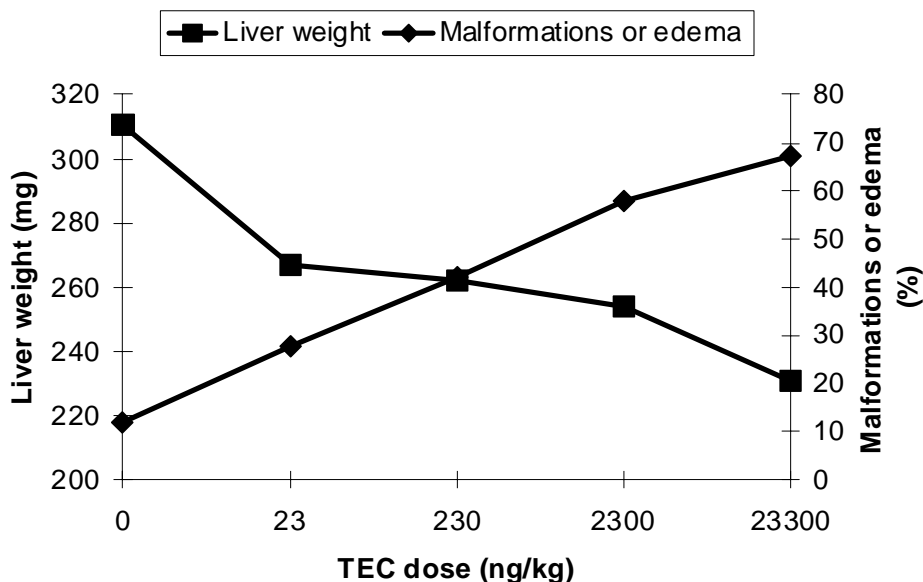


Figure 2. Mean liver weight and percentage of embryos or hatchlings with malformations or edema in the American kestrel egg injection study by Hoffman et al. (1998).

A study that supports the results of the Hoffman et al. (1998) has recently been published (Lavoie and Grasman, 2007). In that study, the authors injected chicken eggs with PCB 126, the same PCB congener as used in the Hoffman et al. (1998) paper. Two different trials were run, with similar results. At a concentration of 25 ppb TEC, 61% (trial 1) and 69% (trial 2) of the eggs died compared to 21% and 22% mortality in control eggs. Similarly, 31% and 32% of the eggs with 25 ppb TEC had deformities compared with 5% and 13% with deformities in controls (Lavoie and Grasman, 2007). All of these effects at the 25 ppb TEC dose were statistically significantly different than controls. The higher severity of effects observed in this study (at 25 ppb TEC) compared with the Hoffman et al. (1998) study (at 23 ppb TEC) is most likely because chickens are more sensitive to 2,3,7,8-TCDD-like toxicity than American kestrels. Nevertheless, the results of the Lavoie and Grasman (2007) study provide additional evidence that the 23 ppb TEC dose that I used from the Hoffman et al. (1998) study is a reasonable value.

Second, in the American kestrel tests with PCB77, the 5,000 ppb TEC dose was the lowest dose tested. At that dose there were significant reductions in embryo survival, hatching success, and hatching or pipping percentage, and a significant increase in malformations or edema. Therefore, this dose is an unbounded LOEL, and since the TEC dose is much higher than the lowest doses

at which effects were seen in the other tests, I used the 23 pptr and 230 pptr TEC values instead of the values from the PCB77 test.

Third, it should be pointed out that the low effect and more severe effect levels of 23 pptr and 230 pptr TEC in bird eggs that I selected from this study bracket the range of the NOEL and LOEL values of 40 pptr TEC and 80 pptr TEC used in Stubblefield (2007). TEC concentrations measured in bird eggs at the site are up to 788 pptr¹² (Beltman and Stackhouse, 2007).

3.1.3 Facility management goals were ignored

In essence, this concern is that I did not assume that conditions at the USM site will never change, and that I assessed risks to plants, invertebrates, and soil microbes as well as birds and mammals. I have addressed this concern in my comments on Dr. Stubblefield's report (sections 2.1 and 2.3). In short, it is unreasonable to assume that operating conditions at the site will not change, and a comprehensive assessment of the risks from contamination at the site must include consideration of risks to plants, invertebrates, and microbes.

3.1.4 Important site-specific evidence was not considered

There are several pieces of evidence that Stubblefield (2007) claims I did not consider but should have. First, he claims that I ignored the results of bird toxicity tests conducted with water from the Active Waste Pond. To the contrary, I considered the results very carefully in developing my opinion that the high acidity of the water makes it unpalatable to birds. In fact, the birds consumed so little of the water that no signs of toxicity were observed in the tests. The water did cause irritation to bird tissue exposed to it, which I noted in my report.

12. Stubblefield (2007) relies on the same bird egg concentration data and makes no mention of concerns regarding the quality of the data. Wait (2007), in a separate expert report on behalf of USM, points out that the supporting quality assurance/quality control (QA/QC) documentation for the egg samples was not available for him to review, and without that documentation he calls into question the validity of the egg chemical data. However, it is my understanding that supporting QA/QC documentation for the egg samples is available and is being provided to USM. The egg contaminant data are one piece of evidence documenting ecological risks at the site; contamination in soils, sediment, and wildlife dietary items also demonstrate risk to ecological receptors (Beltman and Stackhouse, 2007). Furthermore, the chemical contamination measured in the bird eggs is consistent with the contamination measured in soils, sediments, and dietary items at the site, and the egg contamination data show consistency across the three years of sampling and analysis (2004, 2005, and 2006). Therefore, I am continuing to rely on the egg contaminant data in forming my opinions regarding ecological risk at the site, and if those data are deemed to be invalid, my conclusions regarding ecological risk at the site do not change.

Second, he points to the results of the embryological exams that were conducted on the eggs collected from the field and analyzed for contaminants as evidence that there was no toxicity to the eggs from the contamination, and that I ignored this information. As I described in Section 2.9, because of the way they were designed and conducted, the results of these embryological exams cannot be used to determine whether the egg contamination is toxic. Dr. Stubblefield also states that I should have considered the fact that a piece of eggshell fell into one of the sampling jars when the egg contents were placed into the jar, and that this eggshell could have contaminated the sample and produced falsely high chemical concentration measurements. However, as I discussed in Section 2.9, the piece of eggshell was removed after it fell in, and it is simply not plausible that any external contamination of the eggshell could be responsible for the high chemical concentrations measured in the egg contents.

Third, he states that I ignored that mice can be easily trapped at the site and that this fact contradicts my conclusions regarding chemical risks at the site. As I describe in Section 2.9, although studies have shown that mice are present at the site, the studies do not address whether there would be more mice present in the absence of contamination, or whether the individual organisms that are present are healthy. Moreover, I did in fact consider the fact that mice are present at the site in that I used the chemical concentration data measured in them to evaluate potential dietary toxicity to the birds and mammals that eat them.

Fourth, Stubblefield (2007) states that I did not consider the fact that the dietary exposure pathway at the Old Waste Pond is incomplete, and that I should not have evaluated this pathway. This statement presumably is based on the difficulty that Dr. Stubblefield's colleagues had in collecting brine fly larvae from the Old Waste Pond. However, there have not been any detailed studies of shorebird feeding at the site, and it is not in fact known that they do not feed in the Old Waste Pond. Nevertheless, I deliberately compared chemical concentrations in Old Waste Pond sediments to toxicity thresholds that include food chain bioaccumulation to account for potential changes in the future should the Old Waste Pond become improved habitat, such as if the Great Salt Lake breaches the berm around the pond as occurred in 1986 (Olafson, 1988). Therefore, I evaluated potential risks from food chain exposure to chemicals in the Old Waste Pond as part of the evaluation of potential long-term conditions at the site, regardless of what shorebird exposure to chemicals in brine flies is under current conditions.

3.1.5 Uncertainties not recognized

The final methodological concern raised in Stubblefield (2007) is that I did not include a description of how the uncertainties in my analysis lead to "significantly overestimating risk." However, there are two points to make in response to this comment. First, in my expert report I did in fact describe the uncertainties inherent in attempting to compare measured chemical

concentrations at the site with toxicity values from the literature. Those uncertainties include (from Beltman and Stackhouse, 2007):

- ▶ *Variability in organism exposure to chemicals in the environment.* Organisms can differ in their actual exposure to chemicals depending on their feeding habits and life history. Exposure can vary from season to season or year to year, depending on environmental conditions. As a result, organisms of the same species living in similar areas can have different exposure to chemicals in the environment.
- ▶ *The inherent biochemical sensitivity of the exposed organisms to the chemical.* Chemical sensitivity often varies considerably between species, as well as between individuals of the same species.
- ▶ *The life stage of the exposed organism.* Dioxins, furans, PCBs, and hexachlorobenzene tend to be most toxic to developing embryos and young, but other life stages are affected as well.
- ▶ *The susceptibility of the exposed organism to chemical toxicity.* Organisms that are under stress from hunger, thirst, nutritional deficiencies, exhaustion, disease, cold, or heat can be more susceptible to chemical toxicity. For example, organisms in the desert may be more susceptible to the adverse effects of pollutants because they live in a highly demanding environment with its own inherent stresses (Everts, 1997).
- ▶ *Physical and geochemical properties of the soil, sediment, or surface water that can affect the bioavailability of the chemicals.* For example, chlorinated hydrocarbons tend to be more bioavailable from soil or sediment with low organic carbon content than from soil or sediment with high organic carbon content.

I also stated in my expert report that another potential source of variability is the application of laboratory toxicity data to field conditions. Most of our knowledge about the toxicity of chemicals comes from laboratory toxicity studies on relatively few bird and mammal species. These studies use animals that are healthy, well-fed, and under benign laboratory conditions, and that typically monitor only a handful of toxicological endpoints at a time. Therefore, the uncertainty in translating the results from laboratory toxicity studies to field conditions also makes it difficult to rely on single concentrations as toxic thresholds. It was precisely because of these uncertainties that I chose to assess the contamination at the site using the approach I did, and why an area-specific, chemical-specific, and receptor-specific hazard quotient approach that requires many detailed inputs and assumptions like that in Stubblefield (2007) gives a false sense of precision to the risk analysis.

Second, in the approach that I used, I compared all of the available site concentration data points against multiple toxicity reference benchmarks. With this approach, the uncertainty in exposure concentrations and in toxicity reference values is much more explicit than in the hazard quotient approach. I rely on many fewer assumptions, and do not attempt to define exposure and toxicity using single point values. In essence, comparing every site concentration data point to a range of toxicity reference values involves very little uncertainty since it is a direct use of all of the available data. Moreover, because the contamination at the site is so high, there is little uncertainty about the conclusion that the site contamination poses a substantial threat to ecological resources in the area.

3.2 Conclusions

Stubblefield (2007) raises numerous criticisms of my expert report and its conclusions. Some of the criticisms are directed at the approach I used to evaluate risks, and some are directed at specific aspects of my analysis. I disagree with the criticisms raised by Stubblefield (2007), and stand by my opinions as presented in my expert report.

4. Response to Boyle (2007)

Boyle (2007) presents opinions regarding wildlife abundance and behavior at the USM site, and includes a review of the opinions in Beltman and Stackhouse (2007) that relate to wildlife abundance and behavior. In summary, the opinions in Boyle (2007) are as follows:

- ▶ The field methods that Beltman and Stackhouse (2007) relied on to observe wildlife activity and behavior at the USM site were reasonable.
- ▶ The conclusions in Beltman and Stackhouse (2007) regarding wildlife presence at the USM facility are correct.
- ▶ The USM facility is much less attractive to birds than the Great Salt Lake shoreline.
- ▶ The observations reported in Beltman and Stackhouse (2007) of unusual behavior of birds that come into contact with the water of the Active Waste Pond (e.g., gagging, alternately lifting feet) are in fact normal and not indicative of stress or harm.

The first two conclusions require no response, as Mr. Boyle is agreeing that our methods and conclusions regarding wildlife presence at the USM site are sound. His third opinion, that the USM facility is much less attractive to birds than the Great Salt Lake shoreline, is based in part on a bird survey that Mr. Boyle's company conducted in 2006. Without providing an opinion on

that survey itself, Mr. Boyle's opinion that the USM facility is less attractive habitat than the Great Salt Lake is not directly relevant to assessing the ecological risks posed by the chemical contamination at the site. The fact that many birds and other types of wildlife inhabit, visit, and feed at the site is well established through several years of direct observation, and the fact that these organisms are exposed to contaminants at the site is not disputed (Stubblefield, 2007). Therefore, the key point is that many birds and wildlife do use the site and are exposed to chemicals at the site, not whether there are as many birds using the USM site as use the adjacent Great Salt Lake. The description of the importance of the Great Salt Lake as a habitat for birds in Beltman and Stackhouse (2007) was included to point out that the USM site is located in an area of high bird use and that therefore birds are exposed to chemicals at the site, not that bird use at the USM site is equal to bird use at the lake.

Mr. Boyle's fourth opinion, that the behaviors exhibited by birds in the Active Waste Pond are normal, is based primarily on the observations of Mr. Coen Dexter. This opinion is in direct contrast with the opinion of Mr. Mark Stackhouse that many of the behaviors that he observed are in fact signs of distress in the birds that came in contact with the Active Waste Pond (Beltman and Stackhouse, 2007). Mr. Stackhouse is an expert in bird behavior, and particularly the behavior of sick or distressed birds. For six years at the Dayton (Ohio) Museum of Natural History, and for 12 years at Tracy Aviary in Salt Lake City, Mr. Stackhouse rehabilitated and closely observed thousands of birds suffering from a wide variety of injuries and sicknesses. He was in charge of the Tracy Aviary (Salt Lake City) bird rehabilitation center, which received approximately 600 birds per year for rehabilitation that represented over 200 species of birds native to Utah and many other species native to North America. Mr. Stackhouse was able to observe closely and learn about the behavior of birds suffering from many different kinds of illnesses and injuries. Mr. Stackhouse based his opinions about the behavior of the birds at USM's Active Waste Pond on this experience as well as his thousands of hours spent in the field observing birds under natural conditions.

References

- Beltman, D. and M. Stackhouse. 2007. *Environmental Endangerment at the U.S. Magnesium Facility, Rowley, Utah. Expert Report*. Prepared for U.S. Department of Justice. February 5.
- Bleavins, M.R., R.J. Aulerich, and R.K. Ringer. 1984. Effects of chronic dietary hexachlorobenzene exposure on the reproductive performance and survivability of mink and European ferrets. *Archives of Environmental Contamination and Toxicology* 13:357-365.
- Boersma, D.C., J.A. Ellenton, and A. Yagminas. 1986. Investigation of the hepatic mixed-function oxidase system in herring gull embryos in relation to environmental contaminants. *Environmental Toxicology and Chemistry* 5:309-318.

- Boyle, S. 2007. An evaluation of conclusions concerning wildlife abundance and behavior. Presented in *Environmental Endangerment at the U.S. Magnesium Facility, Rowley, Utah. Expert Report*. Prepared for Parsons Behle & Latimer. April 23.
- Carey, J., P. Cook, J. Giesy, P. Hodson, D. Muir, J.W. Owens, and K. Solomon (eds.). 1998. *Ecotoxicological Risk Assessment of the Chlorinated Organic Chemicals*. Society of Environmental Toxicology and Chemistry (SETAC), Pensacola, FL.
- Carpenter, H.M., D.E. Williams, and D.R. Buhler. 1985a. A comparison of the effects of hexachlorobenzene, β -naphthoflavone, and phenobarbital on cytochrome P-450 and mixed-function oxidases in Japanese quail. *Journal of Toxicology and Environmental Health* 15:93-108.
- Carpenter, H.M., D.E. Williams, and D.R. Buhler. 1985b. Hexachlorobenzene-induced porphyria in Japanese quail: An in vitro study of changes in cytochrome P-450 and monooxygenases. *Journal of Toxicology and Environmental Health* 16:207-217.
- Carpenter, H.M., D.E. Williams, and D.R. Buhler. 1985c. Hexachlorobenzene-induced porphyria in Japanese quail: Changes in microsomal enzymes. *Journal of Toxicology and Environmental Health* 15:431-444.
- CCME. 2003. Canadian Environmental Quality Guidelines, Update 3.2 (CD-ROM). Canadian Council of Ministers of the Environment. December.
- Chapman, P.M., A. Fairbrother, and D. Brown. 1998. A critical evaluation of safety (uncertainty) factors for ecological risk assessment. *Environmental Toxicology and Chemistry* 17(1):99-108.
- DeWitt, J.C., E.B. Meyer, and D.S. Henshel. 2005. Environmental toxicity studies using chickens as surrogates for wildlife: Effects of injection day. *Archives of Environmental Contamination and Toxicology* 48:270-277.
- Dobbs, R.C., P.R. Martin, and T.E. Martin. 1998. Green-tailed towhee (*Pipilo chlorurus*). In *The Birds of North America*, No. 368, A. Poole and F. Gill (eds.). The Birds of North America, Inc., Philadelphia, PA.
- Erickson, M.D. 1997. *Analytical Chemistry of PCBs*. 2nd ed. Lewis Publishers, New York.
- Everts, J.W. 1997. Ecotoxicology for risk assessment in arid zones: Some key issues. *Archives of Environmental Contamination and Toxicology* 32:1-10.
- Finley, B. 1998. Expanding the TEF Approach to Include Hexachlorobenzene. Memorandum submitted to Susan Brager Murphy, TEF Workshop Coordinator. In Appendix C-F of USEPA

2001, *Workshop Report on the Application of 2,3,7,8-TCDD Equivalence Factors to Fish and Wildlife*. Risk Assessment Forum, Washington, DC. USEPA/630/R-01/002. August 2001.

Garrison, B.A. 1999. Bank swallow (*Riparia riparia*). In *The Birds of North America*, No. 414, A. Poole and F. Gill (eds.). The Birds of North America, Inc., Philadelphia, PA.

Goldstein, J.A., P. Linko, M.E. Hahn, T.A. Gasiewicz, and H.N. Yeowell. 1986. Structure-activity relationships of chlorinated benzenes as inducers of hepatic cytochrome P-450 isozymes in the rat. In *Hexachlorobenzene: Proceedings of an International Symposium*, C.R. Morris and J.R.P. Cabral (eds). International Agency for Research on Cancer, IARC Scientific Publications No. 77.

Goldstein, J.A., M. Friesen, T.M. Scotti, P. Hickman, J.R. Hass, and H. Bergman. 1978. Assessment of the contribution of chlorinated dibenzo-*p*-dioxins and dibenzofurans to hexachlorobenzene-induced toxicity, porphyria, changes in mixed function oxygenases, and histopathological changes. *Toxicology and Applied Pharmacology* 46:633-649.

Hahn, M.E., J.A. Goldstein, P. Linko, and T.A. Gasiewicz. 1989. Interaction of hexachlorobenzene with the receptor for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin *in Vitro* and *in Vivo*. *Archives of Biochemistry and Biophysics* 270(1):344-355.

Hansen, L.G. 1994. Halogenated aromatic compounds. In *Basic Environmental Toxicology*, L.G. Cockerham and B.S. Shane (eds.). CRC Press, Boca Raton, FL.

Hansen, L.G., S.B. Dorn, S.M. Sundlof, and R.S. Vogel. 1978. Toxicity, accumulation, and depletion of hexachlorobenzene in laying chickens. *Journal of Agricultural and Food Chemistry* 26(6):1369-1374.

Hegdal, P.L. and R.W. Blaskiewicz. 1984. Evaluation of the potential hazard to barn owls of TALON (Brodifacoum bait) used to control rats and house mice. *Environmental Toxicology and Chemistry* 3:167-179.

Hoffman, D.J., M.J. Melancon, P.N. Klein, J.D. Eisemann, and J.W. Spann. 1998. Comparative developmental toxicity of planar polychlorinated biphenyl congeners in chickens, American kestrels, and common terns. *Environmental Toxicology and Chemistry* 17(4):747-757.

Kanzawa, N.K., M. Kondo, T. Okushima, M. Yamaguchi, Y. Temmei, M. Honda, and T. Takahide. 2004. Biochemical and molecular biological analysis of different responses to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in chick embryo heart and liver. *Archives of Biochemistry and Biophysics* 427:58-67.

Lavoie, E.T. and K. Grasman. 2007. Effects of in ovo exposure to PCBs 125 and 77 on mortality, deformities, and post-hatch immune function in chickens. *Journal of Toxicology and Environmental Health* 70(A):547-558.

Marti, C.D., A.F. Poole, and L.R. Bevier. 2005. Barn owl (*Tyto alba*). *The Birds of North America Online*, A. Pole (ed.). Cornell Laboratory of Ornithology, Ithaca, NY; Retrieved from The Birds of North American Online database:
http://bna.birds.cornell.edu/BNA/account/Barn_Owl/.

Meyer, J. 2007. Expert Report of Joan K. Meyer, U.S. vs. U.S. Magnesium et al. Prepared for U.S. Department of Justice. February 6.

Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and reproductive effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in ring-necked pheasant hens. *Journal of Toxicology and Environmental Health* 35:187-198.

Olafson, E.O. 1988. Memorandum to Bureau of Water Pollution Control re: Renewal of NPDES Permit No. UT0000779. June 16. [DMA-0141743-744; M111699-700]

Peterson, R.E., H.M. Theobald, and G.L. Kimmel. 1993. Developmental and reproductive toxicity of dioxins and related compounds: Cross-species comparisons. *Critical Reviews in Toxicology* 23(3):283-335.

Robertson, R.J., B.J. Stutchbury, and R.R. Cohen. 1992. Tree swallow. In *The Birds of North America*, No. 11, A. Poole, P. Stettenheim, and F. Gill (eds.). The Academy of Natural Sciences, Philadelphia, PA; The American Ornithologists' Union, Washington, DC.

Robinson. 2006. Egg processing notes for May 22, 2006. Pages 1-8. [USM219565-72]

Sinclair, P.R., H.S. Walton, N. Gorman, J.M. Jacobs, and J.F. Sinclair. 1997. Multiple roles of polyhalogenated biphenyls in causing increases in cytochrome P450 and uroporphyrin accumulation in cultured hepatocytes. *Toxicology and Applied Pharmacology* 147:171-179.

Stubblefield, W. 2007. Summary of Opinions on Potential Ecological Risks at the US Magnesium, Rowley Utah Site and Summary of Rebuttal to Stratus Consulting's Expert Report "Environmental Endangerment at the U.S. Magnesium Facility, Rowley, Utah" (February 5, 2007). Expert Report. April 23.

U.S. EPA. 1993. *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin Risks to Aquatic Life and Associated Wildlife*. EPA/600/R-93/055. Prepared by Environmental Research Laboratory, Office of Research and Development, Duluth, MN. March. [US-SP-000335-491]

U.S. EPA. 1997. *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments. Interim Final*. EPA 540-R-97-006.

U.S. EPA. 1998. *Guidelines for Ecological Risk Assessment. Final*. EPA/630/R-95/002F.

U.S. EPA. 2001. *The Role of Screening-Level Risk Assessments and Refining Contaminants of Concern in Baseline Ecological Risk Assessments*. Office of Solid Waste and Emergency Response. USEPA/F-01/014. June.

U.S. EPA. 2003. *Framework for Application of the Toxicity Equivalence Methodology for Polychlorinated Dioxins, Furans and Biphenyls in Ecological Risk Assessment. External Review Draft*. EPA/630/P-03/002A. June. Available: <http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=55669>. Accessed 9/28/2006.

U.S. EPA. 2005. *Guidance for Developing Ecological Soil Screening Levels*. OSWER Directive 9285.7-55.

van Birgelen, A.P.J.M. 2000. Hexachlorobenzene: van Birgelen's response. *Environmental Health Perspectives* 108(2):A58.

van den Berg, M., L.S. Birnbaum, M. Denison, M. De Vito, W. Farland, M. Feeley, H. Fiedler, H. Hakansson, A. Hanberg, L. Haws, M. Rose, S. Safe, D. Schrenk, C. Tohyama, A. Tritscher, J. Tuomisto, M. Tysklind, N. Walker, and R.E. Peterson. 2006. The 2005 World Health Organization re-evaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. *Toxicological Sciences* Prepublication version, July 7.

van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, R.X.R. van Leeuwen, A.K.D. Liem, C. Nolt, R.E. Peterson, L. Poelinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives* 106(12):775-792.

Vos, J.G. 2000. Health effects of hexachlorobenzene and the TEF approach. *Environmental Health Perspectives* 108(2):58A.

Vos, J.G., H.L. van der Maas, A. Musch, and E. Ram. 1971. Toxicity of hexachlorobenzene in Japanese quail with special preference to porphyria, liver damage, reproduction, and tissue residues. *Toxicology and Applied Pharmacology* 18:944-957.

Wait, A.D. 2007. Expert Report of A. Dallas Wait, Ph.D. Regarding: Reliability of Measurements Produced for Various Site Investigations at the US Magnesium Facility in Rowley, Utah. Prepared for Parsons Behle & Latimer. April 23.

Walker, M.K. and T.F. Catron. 2000. Characterization of cardiotoxicity induced by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and related chemicals during early chick embryo development. *Toxicology and Applied Pharmacology* 167:210-221.

White, C.M., N.J. Clum, T.J. Cade, and W.G. Hunt. 2002. Peregrine falcon (*Falco peregrinus*). In *The Birds of North America Online*, A. Poole (ed.). Cornell Laboratory of Ornithology, Ithaca, NY; retrieved from The Birds of North American Online database:
http://bna.birds.cornell.edu/BNA/account/Peregrine_Falcon/.