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Technical Review: Smoky Canyon Mine Site- Specific Selenium Criterion Report

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U.S. Fish & Wildlife Service Technical Review: Smoky Canyon Mine Site-Specific Selenium Criterion Report (August 2010 Draft)

Executive Summary:

In response to a formal Congressional request, the Division of Environmental Quality (DEQ) of the U.S. Fish and Wildlife Service (FWS) prepared a scientific technical review of the draft report titled, “*Interpretive Findings for Field and Laboratory Studies and Literature Review in Support of a Site-specific Selenium Criterion, Smoky Canyon Mine.*” The base report and numerous appendices, in sum totaling more than 1,200 pages of documentation, were closely examined.

Multiple distinct scientific issues warranting reconsideration were identified. Five of the most substantive issues included: (1) The lack of valid field controls for interpretation of subsequent toxicity testing as indicated by the data collected from upstream sampling sites versus a true spatial reference site; (2) The failure to properly incorporate complete absence of “swim-up” among some experimental groups into exposure-response toxicity modeling; (3) Systematically biased low and environmentally unrealistic quantification of larval deformity rates; (4) Unjustified extrapolation of the brown trout ratio for partitioning of selenium between egg tissue and whole body tissue to all other co-occurring species of fish; and (5) The absence of any wildlife risk analysis despite the Clean Water Act’s explicit mandate for protection of fish, shellfish, and wildlife.

An attempt to make reasonable data corrections and recalculate the exposure-response relationship for the endpoint of “15-day post swim-up survivorship” resulted in a set of data unsuitable for estimating an EC-10 or EC-20 because of a lack of intermediate-effects response outcomes. An attempt to make reasonable data corrections and recalculate the exposure-response relationship for the larval deformity endpoint produced EC-10 and EC-20 point estimates that were substantively lower than those originally produced in the draft report. The recalculations for the larval deformity endpoint could be improved by further resolving two key uncertainties: (1) the use of hatchery control data as a surrogate for valid field control data; and (2) the use of confounded data summaries for incidences of larval deformities instead of the unconfounded raw data (which J.R. Simplot Co. declined to provide for this review). An examination of selenium tissue partitioning in brown trout relative to a 12-species community of Colorado River fishes suggested that in the majority of fish species their egg selenium concentrations would exceed the proposed criterion if the whole-body selenium threshold for brown trout were applied to all other species.

Three external, independent, peer reviewers concurred with the DEQ/FWS technical critiques in 94.8% (N=58) of their point-by-point peer review evaluations.

Selected Technical Review Comments for:

Interpretive Findings for Field and Laboratory Studies and Literature Review in Support of a Site-Specific Selenium Criterion, Smoky Canyon Mine (August 2010)

REVIEWER'S PREFACE

Via a letter dated March, 30, 2011, from U.S. Senator Barbara Boxer to U.S. Fish and Wildlife Service (FWS) Acting Director Rowan Gould, FWS received a technical assistance request. The request, on behalf of the Senate Committee on Environment and Public Works (EPW), was for a scientific technical review of a draft report (Report) titled, "*Interpretive Findings for Field and Laboratory Studies and Literature Review in Support of a Site-Specific Selenium Criterion, Smoky Canyon Mine.*" The Report was prepared for the J.R. Simplot Company by two consulting firms and the focus of the Report is J.R. Simplot's Smoky Canyon phosphate mine. As part of earlier investigations resulting from an Administrative Order on Consent (AOC) entered into by Simplot, the Idaho Department of Environmental Quality (IDEQ), the U.S. Forest Service (USFS) and the U.S. Environmental Protection Agency (USEPA) together forming the Site Specific Selenium Criterion (SSSC) Workgroup, it was determined that disposal areas for mining waste rock were the source of selenium pollution to down gradient streams in excess of current water quality standards. Source control actions have been identified and implemented, but will not immediately reduce selenium discharges from a contaminated aquifer. Thus, in the interim, the possibility of relaxing the current water standard on a site-specific basis is being pursued. The Report provides part of the technical basis that would be necessary for a site-specific variance from the currently applicable water quality standards for selenium.

FWS staff scientists have been continuously involved with the scientific study of the ecotoxicology of selenium since severe toxic impacts on fish and wildlife from selenium pollution emerged as a major issue at Kesterson National Wildlife Refuge (CA) in the mid-1980s. In addition to conducting original research on the ecotoxicology of selenium, FWS scientists are routinely asked to serve as participants on science panels (e.g., Selenium Science Panel for a site-specific selenium criterion for the Great Salt Lake, Utah) and to serve as scientific peer reviewers for prominent professional journals such as *Science*, *Environmental Science and Technology*, and many others. The present review was conducted as a general scientific peer review. In other words, as if the Report had been submitted for publication and a journal editor had requested a peer review. The only exception to that approach is that the time frame for this review was limited relative to the length and complexity of the report and appendices (greater than 1,200 pages), thus only a selected review of prioritized topics was possible. The following review comments follow in the order of the outline of the Report beginning with the Report Introduction. Bold face type was used to identify language extracted directly from the Report.

SECTION I: Scientific Issues Warranting Reconsideration

1.0 INTRODUCTION

Interpretive findings for Yellowstone cutthroat trout (YCT) are brought into question by the disclaimer presented in this section stating that this “... *Interpretive Report precedes the completion of the YCT studies reporting...*” If the YCT toxicity studies are not yet completed, then, by normal scientific protocol, it would be premature to attempt to interpret those studies. [p. 3]

The Report exhibits some confusion regarding the basis for EPA’s current national water quality chronic criterion for selenium of 5 parts per billion (ug/L). It states that it is “...*based on aqueous exposure only and includes no dietary component of exposure...*” which is erroneous. The Report authors correctly state that the criterion is based on field data from Belews Lake in North Carolina, but they fail to link that with the fact that fish at Belews Lake experienced both aqueous and dietary exposure to selenium as is typical of a field study at any selenium-polluted lake. [p. 5]

The Report again exhibits a degree of confusion with regard to the basis for EPA’s draft chronic criterion revision of 2004 stating that it also was based on data from Belew’s Lake in North Carolina. It was not; it was based on a laboratory experiment (Lemly 1993). To add to this confusion, the authors then ask the reader to compare GMCVs (genus mean chronic values) for trout from EPA’s 2004 draft document to the lower bluegill-based draft criterion proposal while failing to mention that the bluegill result is for an entirely different, and more sensitive, toxicity test (multiple stressors of Se and temperature) than the trout GMCVs (single stressor, Se only). Thus, the implication that trout are more tolerant than bluegill is based on a false comparison. [p. 6]

It is correctly noted in the Report that USEPA commissioned a follow-up study meant to replicate and extend the work of Lemly (1993), but the Report fails to note that the follow-up study did not replicate Lemly’s work. Lemly’s work focused on the interactive effects of selenium exposure and winter-stress syndrome (a physiological response to declining water temperature and decreasing photo period expressed as lipid loss). The follow-up study did not decrease photo period and only lowered water temperature, which failed to induce lipid loss (i.e., failed to induce winter-stress syndrome). Because the follow-up study did not mimic “winter”, unlike Lemly’s work, it does not refute or replace Lemly’s original work; furthermore this limited study is of unknown applicability to free-ranging fish. [p. 6]

2.0 SITE SETTING

The field sampling design is predicated on an assumption that sampling locations upstream of selenium discharges to Crow Creek will represent water quality conditions

“...unimpacted by mining activities.” However, as will be discussed below, the biological data contradict that assumption, which has important implications for the laboratory trout toxicity studies (also discussed below). [p. 11]

3.0 LITERATURE REVIEW

The literature review is uneven. For example, Besser et al.’s (2008) reporting of a low toxicity threshold for desert pupfish is dismissed based on an unpublished memo (Canton and DeForest 2009) prepared on behalf of the Mining Association and Rio Tinto (a mining corporation); a memo that is not easily accessible to the scientific community, as no URL link to a public posting of this privately held memo is provided in the References section of the report [p. 22 and p. 74]. Yet, results that support a high toxicity threshold reported by Kennedy et al. (2000) are accepted uncritically with no mention of a peer-reviewed, published critique of Kennedy et al. (2000) by Hamilton and Palace (2001) [p. 24 and p. 25]. Hamilton and Palace argued, for example, that the study was compromised by unexplained high mortality in eggs obtained from the reference site and by such a narrow exposure interval among most non-reference eggs that there would be “... *little hope of deciphering [statistically] significant differences in reproductive success.*” Issues also could be taken with many other specifics of the literature review, but ultimately the Report’s derivation of a proposed site-specific selenium criterion is not based substantively on the literature review, so the limited time available for this review will be allocated to higher priority comments.

4.0 FIELD MONITORING – EXPOSURE CHARACTERISTICS

It is stated in the Report that, “***Sulfate has been shown to potentially reduce selenium bioaccumulation and resulting toxicity.***” Presumably this is deemed significant because sulfate concentrations were found to positively co-vary with selenium concentrations in the study area and national selenium standards are not based on studies from high sulfate waters. However, it is not noted in the Report that sulfate inhibition of selenate uptake, while demonstrated in the laboratory for environmentally unrealistic conditions (very short-term exposures without other chemical species of selenium present), has never been demonstrated to meaningfully alter bioaccumulation of selenium into aquatic organisms under field conditions, even in selenate-dominated waters (e.g., Birkner 1978). The authors of lab studies demonstrating sulfate reduction of selenium uptake in aquatic organisms clearly recognized the strict limits of their work for extrapolation to natural waters. For example Hansen et al. (1993:77) wrote, “*Thus, at this time, it does not appear that we have sufficient evidence to justify the consideration of sulfate as a factor in the regulation of Se in aquatic environments.*” Williams et al. (1994:452) wrote, “*At present there is little information available that allows us to assess how relevant this study’s conclusions will be in natural waters containing a complex assemblage of selenium [chemical] species.*” Finally, Ogle and Knight (1996:278) reported that for water concentrations of selenium near the national criterion of 5 ppb, “... *the differences [in selenium bioaccumulation and toxicity] between extremely different sulfate*

concentrations are not significant...” In short, there is not a reasonable scientific basis to expect the sulfate concentrations in the study area to make any difference with regard to a site-specific selenium criterion. [p. 33]

Because of the hydrological connectivity between the upstream putative background sampling sites and the selenium source waters the SSSC Workgroup collectively agreed that a reference site completely outside the phosphate mining district was also needed. The chosen reference site was on the South Fork Tincup Creek [p. 14]. The Report later states, “*With few exceptions, concentrations of selenium in both trout and sculpins tissue from upstream Crow Creek and Deer Creek locations ([putative] background) are not significantly different from the reference location tissue concentrations.*” [p. 34].

However, that statement is contradicted by data presented in Figure 4-8, Table 5-2, Appendix A: Table 4 and the discussion of statistical results on page 6 of Appendix A. Statistical test results presented in Appendix A: Table 4, show that trout whole body tissue selenium at the reference site is statistically significantly lower than at the upstream purported background sampling sites on Crow Creek and Deer Creek. This is also visually evident in Figure 4-8. Table 5-2 reports a trout tissue selenium value of 2.56 ug/g dw at the regionally appropriate reference site outside the mining district, which is typical for fish from waters unpolluted with selenium (see Walsh et al. 1977; Lowe et al. 1985 and summary in Skorupa 1998: Table 1). By comparison, fish tissue values in upstream Crow Creek and Deer Creek, the purported background streams, averaged from about 6-9 ug/g dw which is well above the unpolluted fish tissue norms established by national monitoring programs. Trout movement up and downstream into and out of the exposed stream segments and the purported background segments constitute the most plausible explanation for these data. Accordingly, the assumption that the upstream sampling sites represent background conditions uninfluenced by mining seems unlikely to be valid, especially given the absence of barriers to fish movement in the study system.

It should be noted in the Report that brown trout are known to undergo vitellogenesis, the stage of egg formation when selenium is incorporated into fish eggs, for 6-7 months prior to spawning (Estay et al. 2003). This extended period allows ample time for long distance movement of fish up and downstream. This interpretation of the fish tissue data is further supported by the results illustrated for periphyton in Figure 4-14. Periphyton is capable of only passive movement with water currents and therefore cannot move upstream. In contrast to the mobile trout, the tissue selenium of periphyton is not significantly different (see also Appendix A, Table 14) between the regionally appropriate reference site on South Fork Tincup Creek and the upstream Crow Creek and Deer Creek sites (see also Appendix A, Table 14).

In summary, Figure 4-1 shows tight concordance of water selenium concentrations (< 1-2 ppb) for the reference site and the purported background upstream sites, Figure 4-14 shows tight concordance of periphyton selenium concentrations (2-3 ppm) for the reference site and the upstream sites, yet the trout selenium concentrations are distinctly

discordant at the reference site versus the purported background upstream sites. The much higher trout selenium at the upstream sites relative to the regionally appropriate reference site would be the unexpected result, not the low reference site trout selenium, according to current models of selenium transfer from a periphyton food web base up to fish (see Presser and Luoma 2010). The fact that the fish sampled at the purported background locations appear to have been influenced by mining has important implications for the brown trout toxicity study in light of the fact that no spawning brown trout that could have served as valid field controls were collected from South Fork Tincup Creek (discussed further below).

The Report indirectly highlights the fundamental problem with population level evaluations of trout when it is stated, *For this study, trout populations are being evaluated to assess if there are any obvious [underlining mine] negative impacts to those fish populations due to selenium.*” [p. 40]

Clearly, “...**obvious negative impacts..**” are rarely evident at the threshold levels of toxicity that are the appropriate focus for establishing water quality criteria; especially in demographically open populations that can be maintained by immigration. Using fish density measurements or age-class profiles as the baseline criteria to establish an effect is indefensible due to the high baseline variability of such coarse metrics (see review of this topic in Janz et al. 2010: Section 6.7.3). By relying on such coarse metrics the Report fails to clearly distinguish between measures of habitat attractiveness and habitat quality.

What these investigators should be looking at in the field are specific demographic parameters that would allow demographic modeling. For example, Vasterling (2003) took that approach to studying riparian birds in the Idaho phosphate district and found that even though the species she studied were highly abundant, they were not self-replacing and that their persistence (at least during the study years) depended on immigration. She also found evidence for localized selenium toxicity, despite documenting abundant populations.

Low quality polluted habitats that poison fish and birds can, nonetheless, be highly attractive. For example, Kesterson Reservoir in California was a site that caused catastrophic levels of reproductive toxicity among birds including complete (100%) reproductive failure, yet year after year very high densities of breeding water birds could be found at Kesterson Reservoir, with these demographically open populations being maintained by immigration of new breeders from outside the system. Yet, if researchers had gone no further than documenting the high densities of breeding birds at the site they would have erroneously concluded that no adverse effects on birds were occurring at Kesterson when in fact this reservoir was a highly dangerous “attractive nuisance” and regionally attracted waterbirds from other “source” populations into a demographic “sink” population. The trout populations in the Crow Creek watershed, just like the bird populations at Kesterson, are open populations with no barriers for movement of individuals into and out of the population. Accordingly, all of the investigative effort spent and space used in the Report to document trout densities and draw toxic risk conclusions from those densities [summarized on **pp. 46-47**] is misleading. These data

simply show that trout are in the stream just as simple counts of breeding birds at Kesterson showed birds there were abundant. In neither instance would these data address the issue of whether the water quality was impaired, or not, from selenium or any other chemical. This comment also applies to the portion of the literature review that was devoted to reviewing the many other fish count studies that also erroneously conclude that high habitat occupancy, simply one measure of habitat attractiveness, equates to high habitat quality (i.e., low toxic risk). These studies fail to account for the mobility of the species and the confounding effect of immigration to data interpretation. Studies of that type, as opposed to the type represented by Vasterling (2003), are simply not designed to rule out the possibility that they are dealing with contaminant-induced demographic sink populations that would collapse in the absence of continual immigration from outside the site. Janz et al. (2010:191) recount a good case example of this issue. The measured whole body selenium concentrations in Thompson Creek trout were 4-14 ug/g dw and fish in this creek persisted across about 20 years of standardized monitoring. In a nearby tributary and adjacent pond the selenium concentration in trout averaged 13 ug/g dw whole body, yet the trout in these water bodies collapsed and disappeared within the first three years of monitoring. The primary difference between Thompson Creek and the nearby water bodies was Thompson Creek was open to fish movement into and out of the resident population from other areas, while the other nearby waters were not.

5.0 LABORATORY STUDIES – EFFECTS CHARACTERISTICS

Because the proposed site-specific criterion is based on the brown trout toxicity study (Appendix F), review comments for this section will largely relate directly to the material presented in Appendix F of the Report and in Appendix A of Appendix F.

There are two fundamental concerns with the interpretation of the brown trout toxicity study:

The first concern is the lack of any true field control fish from which to calibrate point estimates of effects. The authors themselves state, ***“For the brown trout studies, the response observed at [hatchery] controls was adjusted to the response observed at background since true controls for this study were not practical.” [p. 50]***

This is problematic given the evidence (discussed above) that the upstream Crow Creek and Deer Creek fish didn't represent normal or background exposures to selenium, but probably something on the order of about 2-times to 4-times normal background exposures of 2-3 ug/g dw whole body fish tissue as found at the appropriate reference site. Until spawning fish can be collected from South Fork Tin Cup Creek or a comparably mining-independent reference site, with whole body tissue selenium < 3 ug/g dw and egg selenium < 4 ug/g dw (see Table 5-2 for South Fork Tin Cup Creek data), the point estimates of toxic effects cannot be confirmed to have been based on proper controls. If the fish sampled at upstream study area sites are compromised controls, then toxicity point estimates such as EC-10s or EC-20s will systematically overestimate the tissue concentration of selenium that causes a 10% or 20% toxic effect. To check for this

potential source of systematic bias, comparison toxicity response curves could be calculated based on the hatchery controls where the data for hatchery fish seem suitable or on theoretical full performance values where the hatchery control data are problematic.

The second concern is that the calculated point estimates of toxic effects are not based on the most sensitive reproductive endpoint. The point estimates are based on the endpoints of alevin survival to 15 days post-swim-up and sum total normal fry across four categories of deformity/abnormality. The EC-20s for those two endpoints are 21.6 ug/g and 21.7 ug/g egg selenium respectively (Appendix F: Table 8), yet Appendix F, Figures 12 and 13, show that the EC-100 for swim-up occurs somewhere between about 20 ug/g and 27 ug/g egg selenium. Although many of the brown trout alevins that had failed to swim-up by the time this study was terminated may have still been living (2.4-33%), in nature total failure of swim-up would be equivalent to total reproductive failure. Analysis of the swim-up endpoint for less severe point estimates of effect than the EC-100 should be explored.

Related to the reported observation that “... **yolk fry never swam up...**” (Appendix F: page 17) for the five sets of eggs with the highest selenium exposures (also recorded as 0 “% Swim-up” in Table 5-2 of the Report’s main body and illustrated that way in Figure 12 of Appendix F), it is very perplexing as to how the 15-day Post Swim-up feeding trials produced any data for those same sets of eggs (reported in Appendix F, Figure 37)? Clearly, before a 15-day Post Swim-up Feeding Trial can be conducted alevins have to actually swim-up. If “... **yolk fry never swam up...**”, how is Post swim-up data presented in Figure 37 generated?

The formulation of the “sum total normal fry” endpoint across four deformity/abnormality categories is not an environmentally realistic way to formulate that data [Appendix F, page 30 and Figure 44]. For example suppose five fry are each assessed for the four categories of deformity/abnormality (as per assessment methods, Appendix F, page 9) and the four effect categories are viewed once each, and each in a different one of four fry, leaving one fry that exhibits none of the deformity/abnormality effects. Then the “average” number of normal fry across the four categories is going to be 80%, 4 out of 5 fry will be free of any particular category of deformity/abnormality even though only one of the five fry in question, or 20%, will be a fully normal fry. That’s a large bias in the direction of underestimating toxic effect (and overestimating EC-10 and EC-20 concentrations) because environmental realism dictates that any fry not fully free of deformity/abnormality is unlikely to survive in nature. The deformity data ought to be re-analyzed based on a “proportion fully normal fry” endpoint. Such an endpoint would be expressed on a 0 to 1 scale rather than a 0 to 4 scale.

When there is no causal relationship between two variables, as is the case with whole body selenium and egg selenium (dietary selenium is the causal variable for both), the proper procedure (e.g., see Gill 1987) to back translate from EC-10 and EC-20 egg selenium concentrations to associated whole body selenium concentrations in trout would be to directly calculate a new regression equation by reversing the original X and Y axes rather than by algebraically re-arranging the original regression equation to solve for

whole body selenium as a function of egg selenium as the Report does on **p. 65** of the main text and on **p. 33** of Appendix F. Accordingly, the whole body translations from EC point estimates for egg selenium presented in the Report and in Appendix F warrant re-calculation.

The species sensitivity distribution (SSD) illustrated in Figure 6-1 of the Report does not appear to be scaled properly on the Y-axis. For example at the brown trout EC-20 of 21.6 ug/g dw egg selenium 1 of 7 species is affected or a proportion of 0.143, but the brown trout data point is plotted at about 0.080 on the Y-axis. Additionally, based on EPA's current policy for tissue-based water quality criteria (i.e., Utah proposed site-specific tissue-based criterion for the Great Salt Lake and 2010 draft proposed revision to the national chronic criterion) Figure 6-1 should be re-drafted based on EC-10 values rather than EC-20 values. If it is believed that results for all the other species of fish illustrated in Figure 6-1 are valid for comparison to brown trout results from this specific study area, then typically EPA would employ such data to estimate the 5th percentile value for the SSD as an acceptably protective chronic criterion for the entire 10-species fish community in the Smoky Canyon study area (of which only two species have been tested). If it is not believed all the results illustrated in Figure 6-1 would necessarily apply on a site-specific basis to the study area then the only site-specific sensitivity conclusion that can be reached is that brown trout are more sensitive than Yellowstone cutthroat trout. Thus, either a 5th percentile value should be calculated for the SSD presented in Figure 6-1, or a safety factor should be applied to the brown trout toxicity results to acknowledge that only two of ten site-specific species were tested for toxic sensitivity to selenium.

6.0 UNADDRESSED ISSUES

Compliance with the Clean Water Act mandates water standards that will provide for the "... protection and propagation of fish, shellfish, and wildlife... (PL 92-500, Sec.101(2)). The Report is silent on all taxa other than fish and aquatic invertebrates. Recent research (Presser and Luoma 2010) reveals that fish whole body selenium concentrations are consistently about 1.1 times the selenium concentrations of co-located aquatic invertebrates. The Report asserts that the proposed site-specific fish egg criterion (21.6 ug/g dw) would translate to about 14 ug/g fish whole body selenium, which, divided by 1.1, would translate to about 12.7 ug/g dw aquatic invertebrate selenium. Based on Ohlendorf's (2003: Figure 17.1) regression of mallard egg hatchability against dietary selenium exposure of hens the EC-10 is only 4.9 ug/g dw. Mallard hens, along with the majority of other species of water birds, rely heavily on aquatic invertebrates during the breeding season in order to meet the high protein requirements of ovulation. For a dietary exposure of 12.7 ug/g dw dietary selenium, Ohlendorf's regression equation predicts a point toxicity estimate exceeding 85% failure of mallard eggs to hatch. Thus, it seems highly doubtful that the proposed site-specific criterion would comply with the Clean Water Act's mandate to protect wildlife. As the studies of Skorupa et al. (2002) and Vasterling (2003) have documented, in both riparian and wetland systems in the phosphate-mining region of southeastern Idaho, avian eggs exhibit elevated selenium

concentrations commensurate with the system wide levels of selenium contamination for species of birds across a spectrum ranging from blackbirds to mallards to geese to sandhill cranes.

Employing brown trout as a sensitive surrogate for all the untested species of fish in Crow Creek, the goal would be to keep the selenium concentrations in eggs of all species below the brown trout-based site-specific criteria of 21.6 ug/g dw (for the moment neglecting whether this particular value is defensible or not, see recalculations below). This would be straight forward to monitor and manage if compliance monitoring were going to be based on direct measures of selenium in fish eggs. But for practical reasons (difficulty of sampling ripe female fish, as for example at the reference site) it is actually whole body selenium that is likely to be monitored. This raises the question of what whole body value would generally keep egg selenium below the target value in most (if not all) local species of fish? Determining this is not as simple as merely answering what whole body selenium concentration is likely to keep brown trout eggs below the target value and then extrapolating that to all other species of fish, as is done in the Report. The Report is silent on the rationale for this. Just as species of fish vary in their toxic sensitivity to selenium exposure, there is also a great deal of interspecies variability in the partitioning of selenium between different tissues. Studying a community of Colorado River Basin fish species, Osmundson and Skorupa (2011) found that simple ratios of egg to whole body selenium vary among species from average ratios of about 1 to 8. The 75th percentile value was about 4. The Report found a ratio for brown trout, at the EC-20 target for eggs of 1.54, i.e; the eggs contained about 1.54 times as much selenium as a whole body analysis would yield. Thus, a proposed egg target of 21.6 ug/g dw “translates” to a whole body target of $(21.6)/(1.54) = 14$ ug/g dw.

However, a ratio of 1.54 is on the low end of the range of species variability for such ratios documented for 12 species of fish in the Colorado River Basin. If species variability of ratios is similar for the 10 species of fish in the Smoky Canyon study area, then a whole body target of 14 ug/g could result in most species of fish (60-70% in the Colorado River Basin data set) actually exceeding the 21.6 ug/g egg target (some species by a lot). The way to protect against this potential problem is to develop a site-specific set of tissue ratios for the 10 species of local fish (or most of them) and then select an appropriately protective translation ratio from that data set. Presumably such a local data set could be obtained within a spawning season or two of field sampling, as was achieved for the study of the Colorado River Basin fish community reported in Osmundson and Skorupa (2011). Alternatively, data from studies such as Osmundson and Skorupa (2011) could be employed to estimate a conservative ratio, for example Osmundson and Skorupa's 75th percentile value. In any case, arbitrarily extrapolating the brown trout tissue translation ratio to all other species of fish is inappropriate and could lead to substantive loss of the presumed protectiveness of a site-specific criterion.

SECTION II: Recommended Recalculations

(1.) Hatch to 15-days post swim-up fry survival endpoint:

This toxicity response endpoint for the brown trout toxicity study (Appendix F of the Final Report) is based on raw data presented in Appendix A of the brown trout toxicity study (i.e., Appendix A of Appendix F) in Tables 3-3 and 3-4. The specific endpoint is the “Total Survival %” from Table 3-4 divided by the “% Hatch” from Table 3-3. For example, for control sample SPC-006 these figures would be $[(96.0) / (99.7)]$ for a survival proportion of 0.963 from hatch until test termination at 15-days post swim-up. For reasons that this reviewer was unable to determine, 27 of the 36 reported survival proportions for this endpoint reported in Table 3-4 are incorrect. Of the 27 incorrect values, 24 are incorrect high and 3 are incorrect low. In some cases the discrepancy is quite substantial. For example, for sample LSV2C-004 the reported endpoint survival proportion is 0.662 (66.2%) even though the reported “Total Survival %” in Table 3-4 of 16.8 and reported “% Hatch” from Table 3-3 of 50.6 actually yield a “hatch to termination” endpoint survival proportion of 0.332 (33.2%; i.e., $16.8 / 50.6$). Thus, to begin with for this recommended recalculation the 27 incorrectly reported results were corrected.

Next, there were five samples (LSV2C-003, LSV2C-004, LSV2C-005, LSV2C-010, and LSV2C-21) that did not produce any swim-up fry by the time the experiment was terminated. Instead of recording a post swim-up fry survival of 0.0% for those samples, the authors of the final report chose to record the percent survival among the developmentally “dead-ended” alevins. By doing this, they are assessing a post swim-up fry endpoint for most of the samples and a developmentally completely different dead-end alevin endpoint for the five samples mentioned above. Mixing two different endpoints for exposure-response modeling fundamentally invalidates the modeling. This error is easily corrected by using only the post swim-up fry endpoint and recording a post swim-up fry survival of 0.0% for the five samples in question.

The authors excluded data from the hatchery control samples because the first set of hatchery controls performed very poorly with regard to % hatch and the second set of hatchery controls were obtained as eyed embryos and therefore weren't comparable to the starting point for all the other samples (started as undeveloped fertilized eggs). While the second set of controls yielded excellent % hatch performance, that may have been due to the advanced stage of the embryos at receipt and thus much shorter holding time to hatch (9-13 days) than for any of the other samples (which had holding times to hatch of 37-47 days). In addition to those data exclusions, there were 10 additional samples with missing test organisms because of clogged drain pipes that caused water to overflow out of the test system, apparently carrying some test organisms away (see Appendix A of Appendix F). The authors do not know how many of the carried away organisms were dead or alive, deformed or normal, or anything else about them. The authors chose to simply ignore the missing test organisms, effectively assuming the proportion of missing

fish that were live or dead and deformed or normal was exactly the same as those proportions in the fish left behind. There is no valid justification for such an assumption. Best scientific protocol dictates that the uncertain results for these 10 samples also be excluded from exposure-response modeling.

Thus, the valid data set for modeling the “hatch to 15-days post swim-up fry survival” endpoint is as follows (Table 1; log transformation of exposure data is performed on ppb exposure instead of ppm exposure to avoid negative logarithms):

Table 1. Corrected dataset for the hatch to 15-days post swim-up fry survival endpoint.

[1] Sample ID	[2] Egg Se ppm dry wgt.	[3] Log Egg Se ppb dry wgt.	[4] Total (post swim-up fry) Survival %	[5] % Hatch	[6] Hatch to 15-day post swim- up proportion survival	[7] Number hatched	[8] Expected number of surviving fry 15- days post swim-up [6] X [7]
CC-150-009	12.8	4.107	27.0	28.5	.947	171	162
CC-150-011	8.4	3.924	95.3	96.0	.993	288	286
CC-150-012	8.5	3.929	86.0	88.8	.967	311	301
CC-150-015	9.1	3.959	77.5	78.3	.990	470	465
CC-150-018	6.9	3.839	84.3	87.2	.967	523	506
CC-150-020	6.2	3.792	96.3	97.3	.990	584	578
CC-350-006	14	4.146	67.7	71.7	.944	430	406
LSV2C-003	40.3	4.605	0.0	93.5	.000	374	0
LSV2C-004	36	4.556	0.0	50.6	.000	253	0
LSV2C-005	26.8	4.428	0.0	71.3	.000	214	0
LSV2C-010	38.8	4.589	0.0	87.0	.000	87	0
LSV2C-012	13.2	4.121	95.7	98.3	.974	590	575
LSV2C-016	13.4	4.127	91.7	95.0	.965	570	550
LSV2C-021	28.1	4.449	0.0	69.3	.000	416	0

The lack of useable data points within the intermediate response range violates the TRAP program requirements for model fitting, generating an error warning message. The EC-10 and EC-20 estimates of 14.9 and 16 ppm shown above are the result of forcing a model fit and should not be viewed as reliable. Recalculation for this endpoint reveals that the useable dataset is not sufficient for reliably estimating any EC-10 or EC-20 values. The only reasonably certain information that these data yield is that the finite threshold for total response, the EC-100 threshold, occurs somewhere between an egg selenium concentration of 14 ppm and 27 ppm. Because 100% response (i.e., total failure of brown trout alevins to achieve swim-up) is already being expressed at an egg selenium concentration of 27 ppm, and because valid estimates of the EC-10 and EC-20 cannot be modeled from the useable dataset, proposing an egg selenium standard of nearly 22 ppm (as the authors do in the final report) based on an invalid EC-20 estimation (as the authors do in the Report) does not seem very defensible. Any site-specific standard proposal higher than 14 ppm egg selenium cannot be defensibly supported by the “hatch to 15-day post swim-up fry survival” endpoint.

(2.) Larval Deformity Endpoint:

The collection of larval deformity data was seriously flawed in at least two ways.

First, the larvae assessed for deformities were not a random subsample of all larvae. Alevins and post swim-up fry that died during the brown trout toxicity study mostly did not get assessed for presence or absence of deformities (see Section 2.5 of Appendix A to Appendix F). Since deformed larval fish can be expected to have a differentially high propensity to die compared to undeformed larval fish, all the measures of percent deformed larvae are likely biased low. For example, among a limited number of test groups (samples LSV2C-004, LSV2C-005, and LSV2C-021) deformity assessments are available for reasonable samples of both dead and live larval fish as follows (Table 2 from raw data presented in the first three of the un-numbered Tables in Appendix C of Appendix F):

Table 2. Comparison of percent deformed fry in samples of dead and live specimens (sample sizes in parentheses).

[1] Sample ID	[2] Type of deformity	[3] % deformed dead larval fish (n)	[4] % deformed live larval fish (n)
LSV2C-004	Craniofacial	100 (101)	1.6 (64)
LSV2C-004	Vertebral	100 (64)	68.3 (63)
LSV2C-005	Craniofacial	100 (89)	44.9 (49)
LSV2C-005	Vertebral	100 (84)	65.3 (49)
LSV2C-021	Craniofacial	100 (101)	31.9 (69)
LSV2C-021	Vertebral	100 (62)	71.0 (69)

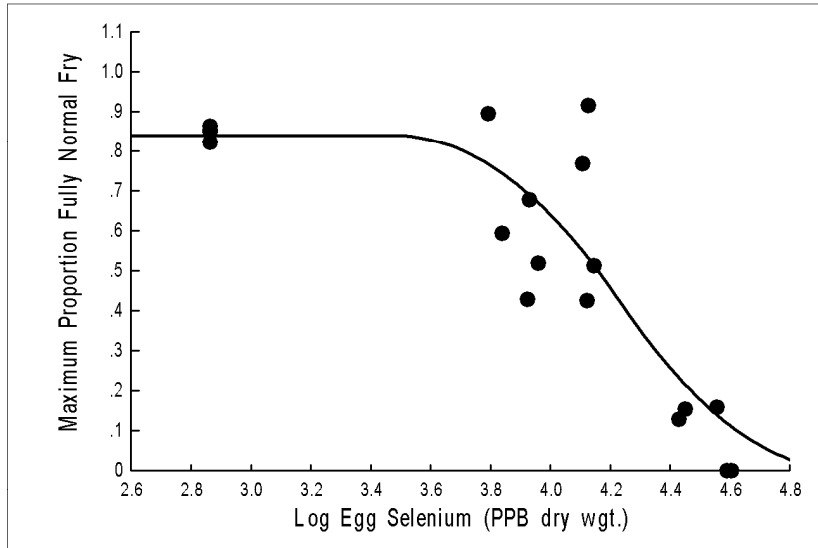
As the above comparisons show, the bias introduced by assessing mostly live (surviving) larval fish for deformities can be substantive. However, the comparisons shown above are for test samples that had high selenium exposures (27-36 ppm egg selenium) and presumably therefore would express this type of bias to a greater extent than test samples from lower exposures would. Presumably, lower exposures would not uniformly produce exclusively (100%) deformed dead larval fish. Since the authors did assess greater numbers of dead fish for the five highest exposure groups and the lower exposure groups often yielded predominantly surviving larval fish, it is possible that the overall magnitude of this bias on exposure-response modeling for larval deformities is relatively modest. Nonetheless, the main points to consider are that the exact amount of bias is unknown, and whatever the magnitude of bias is it is certain to be in only one direction, underestimation of response values (thus overestimation of toxic threshold concentrations).

Second, each of the four types of deformity examined were assessed and reported completely independently of each other (see Appendix C of Appendix F). Thus, while it is possible to determine what percent of assessed larval fish were normal for each type of deformity, it is not possible to determine what percent of assessed larval fish were completely undeformed (i.e., were fully “normal” larval fish). Unfortunately, the raw deformity assessment data were not available for review, but were cited (see p. 3-10 of Appendix A of Appendix F) as being presented in a separate document that Simplot Company refused to release for this review. The authors of the Report used an endpoint that they called “sum total normal fry” which in reality should more accurately have been called “sum partial normal fry” because it is unknown if any of the fry in this sum are simultaneously normal for all four types of deformity. It is only known that the fry in this sum are normal for at least one of the four types of deformity (craniofacial, vertebral, fin, edema). Of course the sum of partial normal fry will necessarily overestimate the sum of fully normal fry and it is only fully normal fry that can be expected in nature to be free of any deformity-related toxic impairment. A recalculation that can be done here to partially correct for the overestimation of fully normal fry is to recognize that the proportion of fully normal fish cannot have been any higher than the lowest proportion of normal fish in any of the four deformity categories reported in Appendix C of Appendix F. This will be referred to as “maximum fully normal fry”. That endpoint should be a much better approximation of percent fully normal fry than the author’s “sum partial normal fry” endpoint. Also, for this recalculation the second set of hatchery controls provide useable estimates of expected background deformity rates because the hatching rates were all acceptable control values (i.e., >90% hatch) and from hatch to 15-days post swim-up fry these controls are strictly comparable in their holding times to the field-exposed test groups. However, the results from the “accidental overflow” test samples must again be excluded from analysis. This yields the following data set (Table 3) for modeling the response variable of larval deformity (log transformation of exposure data is performed on ppb exposure instead of ppm exposure to avoid negative logarithms):

Table 3. Dataset for maximum normal fry endpoint.

[1] Sample ID	[2] Egg Se ppm dry wgt.	[3] Log Egg Se ppb dry wgt.	[4] Maximum proportion totally undeformed (normal) fry	[5] Number undeformed fry for most common of four types of deformity	[6] Number of assessed fry for most common of four types of deformity
CC-150-009	12.8	4.107	.768	109	142
CC-150-011	8.4	3.924	.429	114	266
CC-150-012	8.5	3.929	.637	191	282
CC-150-015	9.1	3.959	.519	231	445
CC-150-018	6.9	3.839	.593	288	486
CC-150-020	6.2	3.792	.894	499	558
CC-350-006	14	4.146	.513	198	386
LSV2C-003	40.3	4.605	.000	0	98
LSV2C-004	36	4.556	.158	20	127
LSV2C-005	26.8	4.428	.128	17	133
LSV2C-010	38.8	4.589	.000	0	71
LSV2C-012	13.2	4.121	.424	235	554
LSV2C-016	13.4	4.127	.915	485	530
LSV2C-021	28.1	4.449	.153	20	131
SPC-001	0.73	2.863	.863	490	568
SPC-003	0.73	2.863	.822	448	545
SPC-005	0.73	2.863	.848	476	561
SPC-006	0.73	2.863	.854	475	556

The authors of the Report employ EPA’s Toxicity Relationship Analysis Program (TRAP) for modeling exposure-response. However, TRAP offers several options with regard to statistical models. The authors chose to use the Least-Squares Nonlinear Logistic Regression model (see Appendix E of Appendix F). EPA advises in the TRAP program overview that the Least Squares Logistic Regression model is most appropriate for endpoints that are (1) measured on a continuous scale (for example weight or height) and (2) expected to have infinite tails approaching 0% and 100% response. Neither of these conditions applies to the brown trout data in question. The data in question are (1) non-continuous dichotomous (binomial) data (each hatched egg either goes on to become an undeformed fry or a deformed fry) and (2) the endpoint would be expected to have a finite “threshold” response in which there is no response until a minimum finite threshold exposure is experienced, not an infinitely gradual response tail. For dichotomous, threshold response data, EPA advises that the most appropriate TRAP model choice is the Tolerance Distribution Analysis fitted to a Triangular Distribution. Therefore, for this recalculation those are the modeling options that were chosen for TRAP exposure-response modeling with the following result (Figure 2):



Parameter Summary (Triangular Tolerance Distribution Analysis)

Parameter	Guess	FinalEst	StdError	95%LCL	95%UCL
X50	4.366	4.234	0.012	4.212	4.257
S	0.15163	0.3090	0.0145	0.2830	0.3403
Y0	0.6740	0.8395	0.0077	0.8238	0.8543

Effect Concentration Summary

%Effect	Xp Est	95%LCL	95%UCL
50.0	4.234	4.212	4.257
20.0	3.956	3.927	3.986
10.0	3.816	3.776	3.856
5.0	3.717	3.669	3.765
0.0	3.478	3.403	3.552

Figure 2. Trap output for maximum fully normal fry endpoint.

This dataset met all TRAP program requirements for a clean model fit without any error warnings. The estimated EC-10 is 6.5 ppm egg selenium and the estimated EC-20 is 9.0 ppm egg selenium. These estimates are not fully corrected for the “sum partial normal fry” bias compared to proportion fully normal fry and therefore are still overestimates of

the EC-10 and EC-20 for the larval deformity endpoint. Even so, both of these recalculated estimates are substantially lower than the site-specific standard of almost 22 ppm egg selenium proposed by the authors of the Report. This endpoint also illustrates why it is so crucial to eventually obtain valid field control data. If the hatchery control data points are omitted, and it is erroneously assumed that the CC-150 and CC-350 (upstream) sampling sites are valid field control sites then the estimates of the EC-10 and EC-20 for larval deformity respectively increase to 15.2 and 17.5 ppm egg selenium as illustrated below (Figure 3):

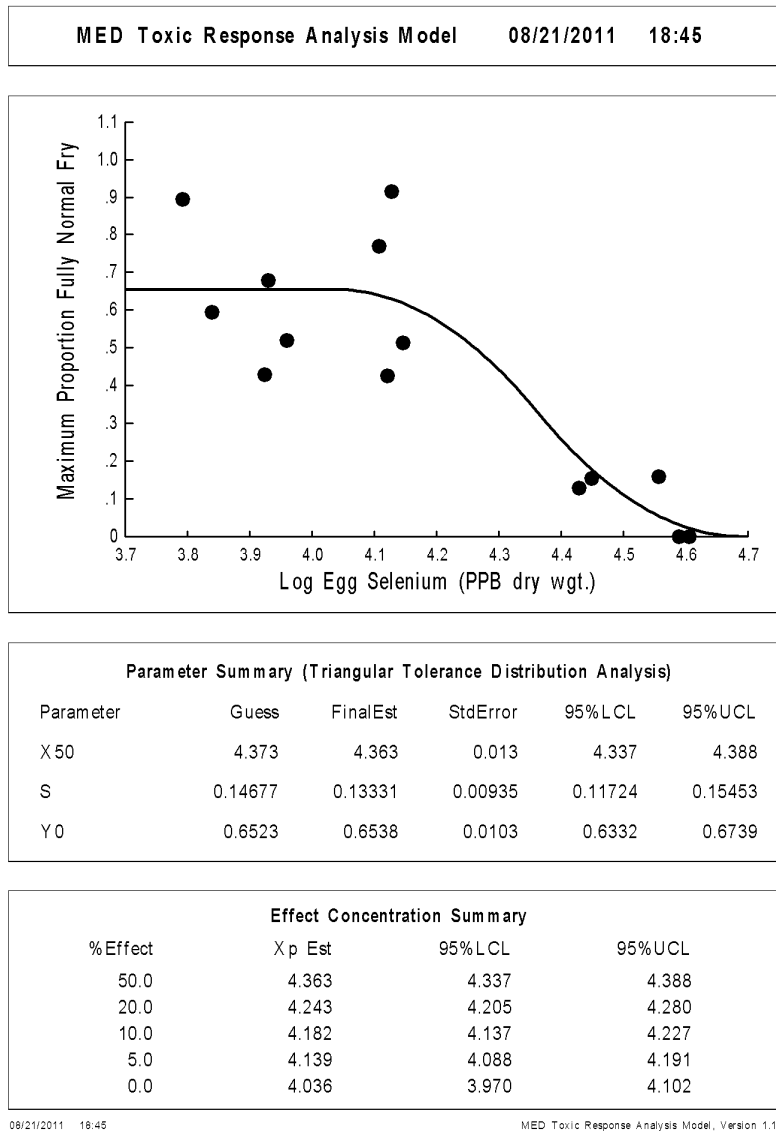


Figure 3. TRAP output for maximum fully normal fry endpoint excluding the hatchery control data points.

Obtaining valid field control results for the larval deformity endpoint should be a very high priority for future research.

(3.) Regressing X on Y for Estimation of Critical Whole Body Tissue Concentrations:

Because of the distinctly species-specific nature of selenium partitioning between tissue types within individual fish (Osmundson and Skorupa 2011) and because of the complex spatio-temporal biology of selenium deposition into brown trout eggs which can be expected to differ in a hatchery versus in the field (Estay et al. 2003), this recalculation is based only on the field data for brown trout as presented in Table 5-2 of the final report. Because tissue concentrations from polluted environments typically conform to log-normal distributions, the regression recalculated here is calculated on log-log transformed data. The recalculated regression is illustrated below (Figure 4). The regression is highly significant ($p < 0.0000001$) and the adjusted R-square value for the regression is 0.788.

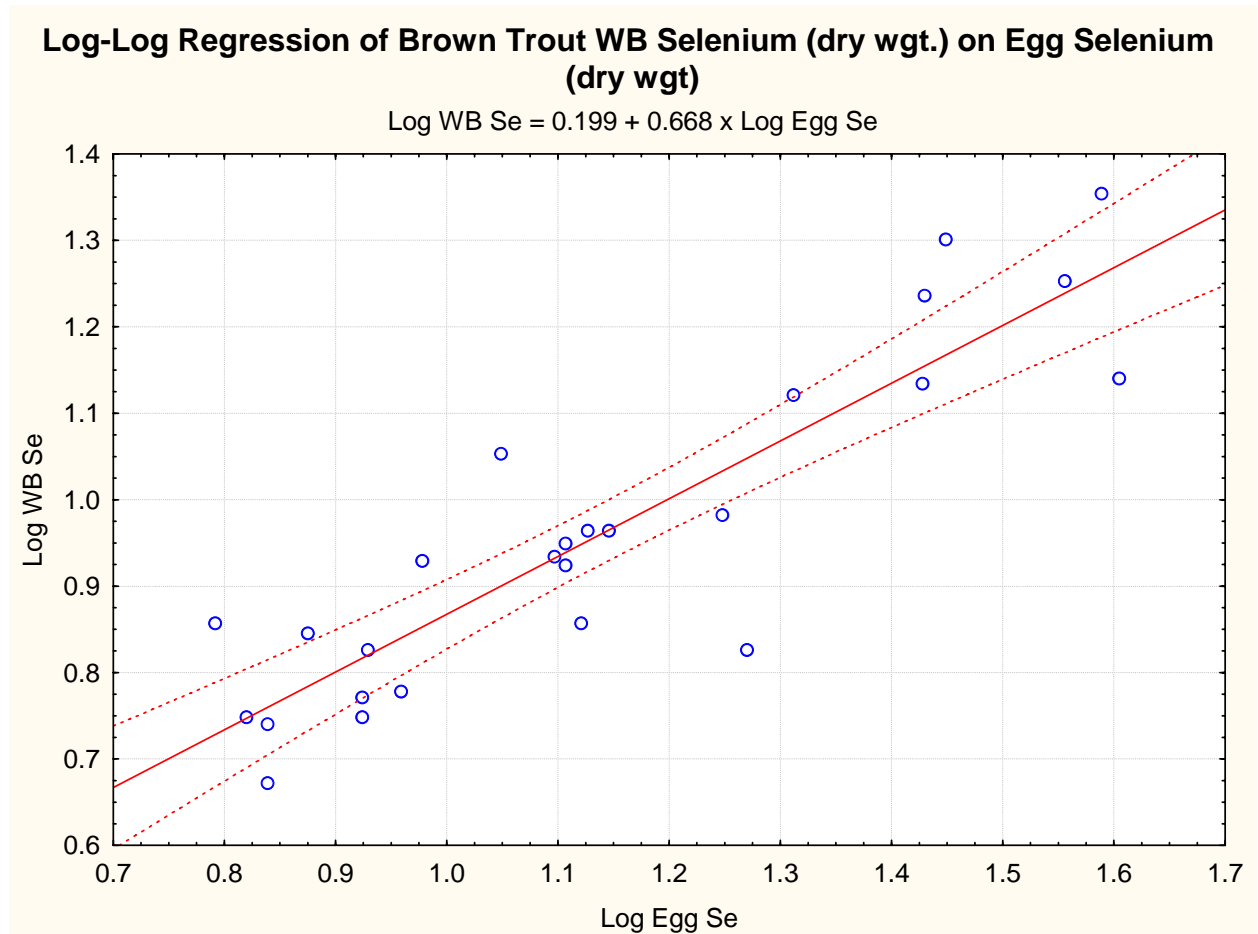


Figure 4. Regression of X on Y for back-calculating critical whole body concentrations from EC-10 and EC-20 egg concentrations.

This regression yields a critical whole body selenium concentration of 12.32 ppm at an egg selenium concentration of 21.63 ppm, a result that is slightly lower than the value of 13.35 ppm critical whole body selenium reported for brown trout data in the Report based on algebraic re-arrangement of the egg selenium on whole body selenium regression equation (see p. 65). When the recalculated regression above (Figure 4) is evaluated at the EC-10 for maximum fully normal fry (Figure 2) of 6.5 ppm egg selenium it yields an estimated critical whole body selenium concentration of 5.5 ppm.

- (4.) Test of Assumption that the 14 ppm Proposed Critical Whole Body Tissue Concentration Derived from Brown Trout Tissue Partitioning Data Would Generally Keep Egg Selenium Values Below a Critical Value of 21.63 ppm Across a Multi-species Community of Fish:

Tissue partitioning ratios from elevated selenium exposure can be expected to be dose-dependent. Therefore to examine this issue using the fish community dataset from the Colorado River Basin reported in Appendices 1 and 2 in Osmundson and Skorupa (2011), only the fish samples with egg selenium results relatively near (in the range of 15 to 30 ppm) the target value of 21.63 ppm proposed in the Report were evaluated. The results are presented below (Table 4):

Table 4. Expected egg selenium concentrations at a whole body selenium concentration of 14 ppm in a multi-species fish community (exceedances of proposed criterion in bold type).

Fish Species	Ovary/Egg Se	Whole Body Se	OE:WB Ratio	Expected Egg Se at Whole Body Se of 14 ppm
Green Sunfish	27.4	22.8	1.20	16.8
Green Sunfish	21.8	15.8	1.38	19.3
Green Sunfish	18.1	11.9	1.52	21.3
Green Sunfish	15.2	9.1	1.67	23.4
Green Sunfish	15.2	9.7	1.57	22.0
Common Carp	16.3	11.7	1.39	19.5
Common Carp	27.3	23.1	1.29	18.1
Roundtail Chub	15.2	6.44	2.36	33.0
Roundtail Chub	17.8	8.4	2.12	29.7
Roundtail Chub	18	6.6	2.73	38.2
Roundtail Chub	16.9	7.2	2.35	32.9
Black Bullhead	26.4	8.59	3.07	43.0
Black Bullhead	28.6	2.9	9.86	138
Channel Catfish	30.3	4.0	7.58	106
Channel Catfish	21.1	3.3	6.39	89.5
Channel Catfish	29.5	3.4	8.68	122
Channel Catfish	15.9	1.9	8.37	117
Channel Catfish	15.2	2.4	6.33	88.6

Thus, for the Colorado River Basin community of fish, assuming that a critical whole body concentration of selenium derived from brown trout data would be universally applicable would have allowed other species of fish to reach egg selenium concentrations as high as 138 ppm, or more than 6-times the proposed site-specific standard of 21.63 ppm. Moreover, the overall exceedance rate would be 68% (weighting equally each species exceedance rate in Table 4). In other words, for this example fish community, a whole body tissue criterion of 14 ppm (that would keep brown trout eggs under 21.63 ppm) would keep only 32% of other fish species eggs under 21.63 ppm. Consequently, in this case, assuming that all fish species partition selenium between tissues comparably to brown trout would be a very under protective assumption. This exercise does not prove that such an assumption would also be very under protective as applied to the Crow Creek fish community, but it does emphasize that making such an assumption for the Crow Creek fish community (as is done in the Report) would seem unlikely to hold true and therefore must be validated before it can be accepted for regulatory purposes.

REVIEWER'S SUMMARY

1. Comparative data for trout versus comparative data for water and periphyton from the reference site (outside the mining district) and the purported background upstream study area sites suggest that regionally appropriate background whole body tissue concentrations should be about 2-3 ug/g dw rather than the 6-9 ug/g dw measured at the purported background upstream sites.
2. The population level surveys of fish abundance throughout the study area do not account for the ability of fish to freely move into the studied populations from outside the study area.
3. Because the upstream study area sampling sites did not represent normal background conditions with respect to trout, and no spawning trout from the reference site were available for sampling, the laboratory toxicity tests for brown trout were conducted without a valid field control.
4. It is uncertain that the most sensitive endpoint was evaluated for threshold toxicity point estimates. Complete failure (EC-100) of alevin swim-up appears to have been reported at egg selenium concentrations comparable to the EC-20 for the Report's chosen endpoints.
5. One of the chosen endpoints, sum total normal fry, is formulated in an environmentally unrealistic manner that is essentially the sum of partially normal fry.
6. The Species Sensitivity Distribution in the Report should be re-formatted based on EC-10s rather than EC-20s to be consistent with current USEPA policy regarding tissue-based criteria, and analyzed for its 5th percentile value.

7. How the proposed site-specific criterion would meet the Clean Water Act mandate for protection and propagation of wildlife should be addressed.
8. It is recommended that translation of a fish egg selenium target to a fish whole body selenium target not be arbitrarily extrapolated from brown trout to all other species of fish without estimating how protective that would be for other species of fish.
9. Correction of errors in the “hatch to 15-day post swim-up fry survival” endpoint database and re-analysis of the corrected data reveal that the data are insufficient for reliably estimating EC-10 and EC-20 tissue concentrations. What can be concluded with high confidence from these data is that the EC-100 is < 27 ppm egg selenium (dw).
10. The “larval deformity” endpoint appears to be a more sensitive toxicity endpoint than the “hatch to 15-day post swim-up fry survival” endpoint. However, data collection for this endpoint suffers from lack of random sampling, and clear presentation of the percent fully undeformed (normal) fry. To the extent that these shortcomings could be partially corrected for, a re-analysis of the deformity data yielded estimated EC-10 and EC-20 values < 10 ppm egg selenium. This is substantively lower than the draft proposed site-specific criterion of 21.63 ppm.
11. The proposed critical whole body selenium tissue concentration of 14 ppm, based on tissue partitioning dynamics in brown trout, was found to be only 32% protective when applied to a full community of Colorado River fish species. It therefore seems unlikely that assuming all species of fish partition selenium between different tissues comparably to brown trout in the Crow Creek fish community will hold true.
12. At the re-calculated EC-10 concentration for larval deformity of 6.5 ppm egg selenium, the estimated critical whole body selenium concentration is 5.5 ppm, an outcome substantially lower than the draft proposed site-specific criterion of 14 ppm.

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APPENDIX 1.

Peer Reviews and Peer Reviewer Curriculum Vitae

Peer Review Scorecard

Scientific Issue	Peer 1	Peer 2	Peer 3
Premature to interpret uncompleted YCT toxicity testing study	+	+	(-)
Current EPA chronic water quality criterion for Se not based solely on aqueous exposure	+	+	+
EPA's draft 2004 chronic criterion not based on Belew's Lake data	+	++	+
Follow-up EPA sponsored winter stress study did not replicate Lemly	+	(-)	++
The literature review is distinctly uneven	+	++	0
Sulfate inhibition of selenium bioaccumulation a nonfactor	0	+	+
Invalid assumption that upstream sample sites represent background	(+)	+	++
Brown trout vitellogenesis chronology not properly considered	+	++	0
Fish densities and age class distributions invalid indicators of effects	(+)	++	++
Absence of true field controls for toxicity testing	(+)	+	+
Swim-up failure EC-100 not properly noted	+	+	+
Frequency of fully normal fry is the proper deformity endpoint	+	+	+
X on Y regression preferable to algebraic rearrangement of Y on X for back calculating from egg Se to whole-body Se	+	0	0
SSD curve should be based on EC-10s not EC-20s	0	(+)	0
Wildlife risk assessment required, but not conducted	+	+	0
Inappropriate extrapolation of Brown Trout tissue partitioning ratio	++	(-)	++
Correction required for 27 hatch to post swim-up survival data points	??	+	??
Swim-up failure beakers should have been counted as 0% survival	+	+	+
Replicates w/ missing organisms should be excluded from analyses	+	+	++
Inappropriate TRAP option selected for modeling	+	(+)	(+)
15-day post swim-up survival endpoint results unsuitable for modeling	(+)	+	0
Survivor bias in larval deformity assessments	(+)	+	+
"Maximum fully normal fry" adjustment warranted	+	+	0
EC-10 & EC-20 estimates substantively lower than reported	+	??	0

Key: ++ = concurrence & expand point beyond FWS critique; + = concurrence; (+) = conditional concurrence; 0 = no judgment; (-) = conditional non concurrence; - = non concurrence ; ?? = peer reviewer too confused by data in draft report to make judgment

I (Mark Rigby, Ph.D.) was asked by Atkins Global to review USFWS comments on the “Interpretive Findings for Field and Laboratory Studies and Literature Review in Support of a Site-Specific Selenium Criterion, Smoky Canyon Mine.” The Interpretive Report represents a substantial body of research and effort. USFWS has reviewed the report and provide thoughtful comments with which I generally concur.

I have been performing human health and ecological risk assessments in the United States since 2000. Those risk assessments have mainly been on behalf of the military bases. With respect to selenium, I have performed risk assessments for fishes and aquatic birds on behalf of the Salton Sea Authority for solar evaporation ponds and other surface water bodies in the Salton Sea Area. I also worked on the North San Francisco selenium TMDL while I was employed at Tetra Tech. Recently, I have been working with Dennis Lemly of USFWS on re-evaluating several published papers on the toxicity of selenium to various fishes.

My evaluation of USFWS’ comments follows.

Responses to Comments from USFWS.

1. Introduction

- A. Interpretive findings for Yellowstone cutthroat trout (YCT) are brought into question by the disclaimer presented in this section stating that this “... Interpretive Report precedes the completion of the YCT studies reporting...” If the YCT toxicity studies are not yet completed, then, by normal scientific protocol, it would be premature to attempt to interpret those studies. [p. 3]

Evaluation: Concur. The interpretive report should be based on the final results of the YCT toxicity testing. Although Appendix H is labeled as “draft,” Appendix G appears to be final.

- B. The Report exhibits some confusion regarding the basis for EPA’s current national water quality chronic criterion for selenium of 5 parts per billion (ug/L). It states that it is “...based on aqueous exposure only and includes no dietary component of exposure...” which is erroneous. The Report authors correctly state that the criterion is based on field data from Belews Lake in North Carolina, but they fail to link that with the fact that fish at Belews Lake experienced both aqueous and dietary exposure to selenium as is typical of a field study at any selenium-polluted lake. [p. 5]

Evaluation: Concur.

- C. The Report again exhibits a degree of confusion with regard to the basis for EPA’s draft chronic criterion revision of 2004 stating that it also was based on data from Belew’s Lake in North Carolina. It was not; it was based on a laboratory experiment (Lemly 1993). To add to this confusion, the authors then ask the reader to compare GMCVs (genus mean chronic values) for trout from EPA’s 2004 draft document to the lower bluegill-based draft criterion proposal while failing to mention that the bluegill result is for an entirely different, and more sensitive, toxicity test (multiple stressors of Se and temperature) than the trout GMCVs (single stressor, Se only). Thus, the implication that trout are more tolerant than bluegill is based on a false comparison. [p. 6]

Evaluation: Concur. As written, the text is incorrect. The Interpretive Report should provide a table comparing chronic toxicity values for trout and bluegill from the literature to

illustrate their point. However, at first glance, the studies reviewed by USEPA (2010) do not appear to support this point.

- D. It is correctly noted in the Report that USEPA commissioned a follow-up study meant to replicate and extend the work of Lemly (1993), but the Report fails to note that the follow-up study did not replicate Lemly's work. Lemly's work focused on the interactive effects of selenium exposure and winter-stress syndrome (a physiological response to declining water temperature and decreasing photo period expressed as lipid loss). The follow-up study did not decrease photo period and only lowered water temperature, which failed to induce lipid loss (i.e., failed to induce winter-stress syndrome). Because the follow-up study did not mimic "winter", unlike Lemly's work, it does not refute or replace Lemly's original work; furthermore this limited study is of unknown applicability to free-ranging fish. [p. 6]

Evaluation: Concur. While I do not disagree, it should be noted photoperiod and the applicability of USEPA (2008) to field populations was not discussed by USEPA (2008). Therefore, this comment is based on USFWS' review and interpretation of the USEPA (2008) study. Nonetheless, USFWS is correct when they state that USEPA (2008) did not manipulate photoperiod and, therefore, the results may not be applicable to fish outside of the laboratory. The Interpretive Report should be revised to include USFWS' critique of USEPA (2008).

2. Site setting

- A. The field sampling design is predicated on an assumption that sampling locations upstream of selenium discharges to Crow Creek will represent water quality conditions "...unimpacted by mining activities." However, as will be discussed below, the biological data contradict that assumption, which has important implications for the laboratory trout toxicity studies (also discussed below). [p. 11]

Evaluation: see response to Comment 4.B.

3. Literature review

- A. The literature review is distinctly uneven. For example, Besser et al.'s (2008) reporting of a low toxicity threshold for desert pupfish is dismissed based on an unpublished memo (Canton and DeForest 2009) prepared on behalf of the Mining Association and Rio Tinto (a mining corporation); a memo that is not easily accessible to the scientific community, as no URL link to a public posting of this privately held memo is provided in the References section of the report [p. 22 and p. 74]. Yet, results that support a high toxicity threshold reported by Kennedy et al. (2000) are accepted uncritically with no mention of a peer-reviewed, published critique of Kennedy et al. (2000) by Hamilton and Palace (2001) [p. 24 and p. 25]. Hamilton and Palace argued, for example, that the study was compromised by unexplained high mortality in eggs obtained from the reference site and by such a narrow exposure interval among most non-reference eggs that there would be "... little hope of deciphering [statistically] significant differences in reproductive success." Issues also could be taken with many other specifics of the literature review, but ultimately the Report's derivation of a proposed site-specific selenium criterion is not based substantively on the literature review, so the limited time available for this review will be allocated to higher priority comments.

Evaluation:

- i. The Interpretive Report states that its literature review is focused on “relevant studies of cold water species”, which is entirely appropriate. However, the Interpretive Report should define what “relevant” species means. I assume that this refers to those species present in the study area.
 - ii. USFWS is correct that the review by Canton and DeForest (2009) is not available online and is not in a peer-reviewed journal. However, if the literature review is focused on species that occur in the study area, then the discussion of selenium toxicity to desert pupfish should be removed, as they do not occur in the study area. If Besser et al. (2008) and Canton and DeForest (2009) are going to be discussed in the literature review, the Interpretive Report should provide both in their entirety as an Appendix. Further, all unpublished studies (e.g., the reports by GEI Consultants) should be included in their entirety in an Appendix.
 - iii. A number of the studies reviewed in the Interpretive Report are rather problematic in experimental design, data analysis, and interpretation. Therefore, without careful review and re-analysis of the original data, the findings of many of the studies included in the literature review may be of limited utility. A critique and evaluation of all of the studies included in the literature review would be very lengthy and time consuming, as indicated by USFWS. However, USEPA (2010) has done some of that work and USEPA’s (2010) review of the literature should probably be included in the Interpretive Report. It should also be noted that the study by Golder from 2009 on Dolly Varden char was published in 2010 (McDonald et al. 2010. *Environmental Toxicology and Chemistry* 29:2800–2805) and the findings of the published study should be presented instead. The report by Elphick et al. on the toxicity of selenium to westslope cutthroat trout (see <https://circle.ubc.ca/handle/2429/24644>) should also be included in the literature review.
 - iv. The statement that “Observations of fish deformities revealed that most locations had few or no fish with any observable deformities” is rather misleading. Selenium induced developmental deformities occur in the larval stages and those larvae with significant deformities are unlikely to survive to adulthood. Thus, the absence of deformed adult fish does not necessarily mean that selenium is not causing developmental deformities. This statement should be removed from the summary of the GEI 2007 report. However, the other findings about fish diversity and density are relevant and instructive.
4. FIELD MONITORING – EXPOSURE CHARACTERISTICS
- A. It is stated in the Report that, “Sulfate has been shown to potentially reduce selenium bioaccumulation and resulting toxicity.” Presumably this is deemed significant because sulfate concentrations were found to positively co-vary with selenium concentrations in the study area and national selenium standards are not based on studies from high sulfate waters. However, it is not noted in the Report that sulfate inhibition of selenate uptake, while demonstrated in the laboratory for environmentally unrealistic conditions (very short-term exposures without other chemical species of selenium present), has never been demonstrated to meaningfully alter bioaccumulation of selenium into aquatic organisms under field conditions, even in selenate-dominated waters (e.g., Birkner 1978). The authors of lab studies demonstrating sulfate reduction of selenium uptake in aquatic organisms clearly recognized the strict limits of their work for extrapolation to natural waters. For example Hansen et al. (1993:77) wrote, “Thus, at this time, it does not appear that we have sufficient evidence to justify the consideration of sulfate as a factor in the regulation of Se in aquatic environments.” Williams et al. (1994:452) wrote, “At present there is little information available that allows us to assess how relevant this

study's conclusions will be in natural waters containing a complex assemblage of selenium [chemical] species." Finally, Ogle and Knight (1996:278) reported that for water concentrations of selenium near the national criterion of 5 ppb, "... the differences [in selenium bioaccumulation and toxicity] between extremely different sulfate concentrations are not significant..." In short, there is not a reasonable scientific basis to expect the sulfate concentrations in the study area to make any difference with regard to a site-specific selenium criterion. [p. 33]

Evaluation: This is outside my area of expertise. However, USEPA (2010) states "The antagonistic relationship between selenium and sulfur, which inhibits selenium bioaccumulation, is well defined, as sulfate competes with selenate in their uptake into aquatic organisms (Ogle and Knight 1996; Riedel and Sanders 1996; Bailey et al. 1995; Hansen et al. 1993; Appendix B)." USEPA (2010) applied a sulfate "correction" to the acute toxicity of selenate.

- B. Because of the hydrological connectivity between the upstream putative background sampling sites and the selenium source waters the SSSC Workgroup collectively agreed that a reference site completely outside the phosphate mining district was also needed. The chosen reference site was on the South Fork Tincup Creek [p. 14]. The Report later states, "With few exceptions, concentrations of selenium in both trout and sculpins [sic] tissue from upstream Crow Creek and Deer Creek locations ([putative] background) are not significantly different from the reference location tissue concentrations." [p. 34]. However, that statement is contradicted by data presented in Figure 4-8, Table 5-2, Appendix A: Table 4 and the discussion of statistical results on page 6 of Appendix A. Statistical test results presented in Appendix A: Table 4, show that trout whole body tissue selenium at the reference site is statistically significantly lower than at the upstream purported background sampling sites on Crow Creek and Deer Creek. This is also visually evident in Figure 4-8. Table 5-2 reports a trout tissue selenium value of 2.56 ug/g dw at the regionally appropriate reference site outside the mining district, which is typical for fish from waters unpolluted with selenium (see Walsh et al. 1977; Lowe et al. 1985 and summary in Skorupa 1998: Table 1). By comparison, fish tissue values in upstream Crow Creek and Deer Creek, the purported background streams, averaged from about 6-9 ug/g dw which is well above the unpolluted fish tissue norms established by national monitoring programs. Trout movement up and downstream into and out of the exposed stream segments and the purported background segments constitute the most plausible explanation for these data. Accordingly, the assumption that the upstream sampling sites represent background conditions uninfluenced by mining seems unlikely to be valid, especially given the absence of barriers to fish movement in the study system.

Evaluation: Partially concur. The text on page 34 is incorrect and should be updated to match Appendix A. The statement that Crow Creek upstream of the confluence with Sage Creek is unimpacted by mining activities should be clarified to state that surface water and sediments are unimpacted and that some of the biota there is also unimpacted; i.e., sculpins (see Appendix A, Table 8), invertebrates (Figure 4-13, assuming Deer Creek is representative of unimpacted conditions), and periphyton (Figure 4-14, assuming Deer Creek is representative of unimpacted conditions). The same statistical comparisons that were performed for fish should be performed for these other environmental media; i.e., the ANOVA/Kruskall-wallis test to evaluate whether there are differences in concentrations by "location" to support these statements. The interpretive report should acknowledge that trout in Crow Creek appear to moving back and forth among the "locations" and trout

caught upstream of the confluence with Sage Creek may have spent some of their time downstream of the confluence. Thus, trout upstream of the confluence may also be affected by the selenium downstream of the confluence and even in Sage Creek. It may be better to focus future work on sculpins, which are probably less vagile, or to install fish barriers prior to fish sampling.

- C. It should be noted in the Report that brown trout are known to undergo vitellogenesis, the stage of egg formation when selenium is incorporated into fish eggs, for 6-7 months prior to spawning (Estay et al. 2003). This extended period allows ample time for long distance movement of fish up and downstream. This interpretation of the fish tissue data is further supported by the results illustrated for periphyton in Figure 4-14. Periphyton is capable of only passive movement with water currents and therefore cannot move upstream. In contrast to the mobile trout, the tissue selenium of periphyton is not significantly different (see also Appendix A, Table 14) between the regionally appropriate reference site on South Fork Tincup Creek and the upstream Crow Creek and Deer Creek sites (see also Appendix A, Table 14).

Evaluation: Concur. The reference cited indicates that the formation of eggs in trout takes at least six months. Combined with the apparent movement of trout in Crow Creek, this allows an extended period over which egg formation of trout upstream of the confluence of the Crow and Sage Creeks could be affected by selenium in Sage Creek.

- D. In summary, Figure 4-1 shows tight concordance of water selenium concentrations (< 1-2 ppb) for the reference site and the purported background upstream sites, Figure 4-14 shows tight concordance of periphyton selenium concentrations (2-3 ppm) for the reference site and the upstream sites, yet the trout selenium concentrations are distinctly discordant at the reference site versus the purported background upstream sites. The much higher trout selenium at the upstream sites relative to the regionally appropriate reference site would be the unexpected result, not the low reference site trout selenium, according to current models of selenium transfer from a periphyton food web base up to fish (see Presser and Luoma 2010). The fact that the fish sampled at the purported background locations appear to have been influenced by mining has important implications for the brown trout toxicity study in light of the fact that no spawning brown trout that could have served as valid field controls were collected from South Fork Tincup Creek (discussed further below).

Evaluation: please see response to comments 4.B. and 4.E.

- E. The Report indirectly highlights the fundamental problem with population level evaluations of trout when it is stated, "For this study, trout populations are being evaluated to assess if there are any obvious [underlining mine] negative impacts to those fish populations due to selenium." [p. 40] Clearly, "...obvious negative impacts.." are rarely evident at the threshold levels of toxicity that are the appropriate focus for establishing water quality criteria; especially in demographically open populations that can be maintained by immigration. Using fish density measurements or age-class profiles as the baseline criteria to establish an effect is indefensible due to the high baseline variability of such coarse metrics (see review of this topic in Janz et al. 2010: Section 6.7.3). By relying on such coarse metrics the Report fails to clearly distinguish between measures of habitat attractiveness and habitat quality. What these investigators should be looking at in the field are specific demographic parameters that would allow demographic modeling. For example, Vasterling (2003) took that approach to studying riparian birds in the Idaho phosphate district and found that even though the species she studied were

highly abundant, they were not self-replacing and that their persistence (at least during the study years) depended on immigration. She also found evidence for localized selenium toxicity, despite documenting abundant populations. Low quality polluted habitats that poison fish and birds can, nonetheless, be highly attractive. For example, Kesterson Reservoir in California was a site that caused catastrophic levels of reproductive toxicity among birds including complete (100%) reproductive failure, yet year after year very high densities of breeding water birds could be found at Kesterson Reservoir, with these demographically open populations being maintained by immigration of new breeders from outside the system. Yet, if researchers had gone no further than documenting the high densities of breeding birds at the site they would have erroneously concluded that no adverse effects on birds were occurring at Kesterson when in fact this reservoir was a highly dangerous “attractive nuisance” and regionally attracted waterbirds from other “source” populations into a demographic “sink” population. The trout populations in the Crow Creek watershed, just like the bird populations at Kesterson, are open populations with no barriers for movement of individuals into and out of the population. Accordingly, all of the investigative effort spent and space used in the Report to document trout densities and draw toxic risk conclusions from those densities [summarized on pp. 46-47] is misleading. These data simply show that trout are in the stream just as simple counts of breeding birds at Kesterson showed birds there were abundant. In neither instance would these data address the issue of whether the water quality was impaired, or not, from selenium or any other chemical. This comment also applies to the portion of the literature review that was devoted to reviewing the many other fish count studies that also erroneously conclude that high habitat occupancy, simply one measure of habitat attractiveness, equates to high habitat quality (i.e., low toxic risk). These studies fail to account for the mobility of the species and the confounding effect of immigration to data interpretation. Studies of that type, as opposed to the type represented by Vasterling (2003), are simply not designed to rule out the possibility that they are dealing with contaminant-induced demographic sink populations that would collapse in the absence of continual immigration from outside the site. Janz et al. (2010:191) recount a good case example of this issue. The measured whole body selenium concentrations in Thompson Creek trout were 4-14 ug/g dw and fish in this creek persisted across about 20 years of standardized monitoring. In a nearby tributary and adjacent pond the selenium concentration in trout averaged 13 ug/g dw whole body, yet the trout in these water bodies collapsed and disappeared within the first three years of monitoring. The primary difference between Thompson Creek and the nearby water bodies was Thompson Creek was open to fish movement into and out of the resident population from other areas, while the other nearby waters were not.

Evaluation:

- i. Page 36 states “If selenium is having a toxic effect, even on a chronic basis, monitoring over a period would be expected to indicate some relative reduction in fishery potential, when other factors are considered.” As stated by USFWS, immigration may maintain populations in locations where fitness is reduced due to pollutants. Therefore, the statement on page 36 should be amended to take this into account; e.g., “If selenium is having a toxic effect, even on a chronic basis, fish density/biomass may be reduced, as long as the effect of selenium toxicity is not masked by immigration from other areas.”
- ii. On a scientific basis, I agree with the statement by USFWS that evaluating populations may not reveal individual level adverse effects when the population evaluated is maintained by immigration (see, for example Larison et al. 2000). However, it should be

noted that none of the fishes in Crow or Sage Creeks are covered by the Endangered Species Act and that IDFG (2007) manages Yellowstone cutthroat trout (YCT) on the population (and drainage) level. Further, the Clean Water Act and the Consent Order do not appear to contain a no incidental take provision. IDFG (2007) states that their goal for YCT in Crow Creek is to “ensure that mine-generated selenium is not having population level effects on cutthroat trout.” Thus, evaluating the effects of selenium on the fishes in Sage and Crow Creeks on a population level appears to be in agreement with the precedent set by IDFG and within the applicable laws. In contrast, the death of birds from selenium poisoning at the Kesterson National Wildlife Refuge was a violation of the Migratory Bird Treaty Act (see Benson et al. 1993).

- iii. In order to evaluate whether selenium has had an adverse effect on the fish populations in Sage and Crow Creeks, additional statistics should be performed, as follows:
 - a. If there is sufficient historical data, statistical tests should be performed to determine whether the populations are increasing or decreasing in density
 - b. The regressions in Figures 4-24a-d should be run as either a principal component analysis (or other ordination technique) or a step-wise multiple regression. When performed that way, the analyses will tell us the relative contribution to the pattern in fish density and diversity due to selenium concentrations, flow rate, summer water temperature, stream gradient, and any other parameters that the authors wish to evaluate (e.g., benthic macroinvertebrate density/diversity, etc). Please also provide p values for all regressions in the figures. An r^2 by itself is not meaningful.

5. LABORATORY STUDIES – EFFECTS CHARACTERISTICS

- A. There are two fundamental concerns with the interpretation of the brown trout toxicity study:
 - i. The first concern is the lack of any true field control fish from which to calibrate point estimates of effects. The authors themselves state, “For the brown trout studies, the response observed at [hatchery] controls was adjusted to the response observed at background since true controls for this study were not practical.” [p. 50] This is problematic given the evidence (discussed above) that the upstream Crow Creek and Deer Creek fish didn’t represent normal or background exposures to selenium, but probably something on the order of about 2-times to 4-times normal background exposures of 2-3 ug/g dw whole body fish tissue as found at the appropriate reference site. Until spawning fish can be collected from South Fork Tin Cup Creek or a comparably mining-independent reference site, with whole body tissue selenium < 3 ug/g dw and egg selenium < 4 ug/g dw (see Table 5-2 for South Fork Tin Cup Creek data), the point estimates of toxic effects cannot be confirmed to have been based on proper controls. If the fish sampled at upstream study area sites are compromised controls, then toxicity point estimates such as EC-10s or EC-20s will systematically overestimate the tissue concentration of selenium that causes a 10% or 20% toxic effect. To check for this potential source of systematic bias, comparison toxicity response curves could be calculated based on the hatchery controls where the data for hatchery fish seem suitable or on theoretical full performance values where the hatchery control data are problematic.

Evaluation: USFWS states that the lack of a “true field control” for the brown trout reproduction study is problematic. However, it should be noted that there are three different factors that could be controlled: 1) background selenium exposure levels, 2) maternal fish condition, and 3) parental genetic effects. The fish from the

hatchery used in the brown trout experiment are appropriate controls in terms of selenium exposures as the selenium concentration in eggs from the hatchery fish was 0.73 mg/kg-dw (see Table 6 in Appendix F). Unfortunately, the figures in Appendix F are unclear as to whether the length (Figure 3), weight (Figure 4), and number of eggs (Figure 8) shown for the “hatchery fish” are from the fish that were used to calculate the ECs (i.e., SPC) as opposed to those hatchery fish that were not used to calculate the ECs (i.e., SC). However, what figures are presented in Appendix F do show that there is a difference in fish condition between the wild caught fish and the “hatchery fish” that might have affected the results. With respect to parental genetic effects, IDFG (2007) and Cegelski et al. (2006) show that the creeks that are genetically closest to Crow Creek for YCT (which I assume would be similar for brown trout) are Horse and West Pine, which would be the most appropriate sources for field controls, with the exception of a location somewhere on Crow Creek. South Fork Tin Cup Creek is somewhat removed genetically, but still may be a viable field control in terms of parental genetics, although that question would best be addressed by the geneticists at IDFG. Overall, I concur with USFWS that the lack of a true field control for the brown trout study (Appendix F) adds some uncertainty to the ECs derived for brown trout and that it would be preferable to use either fish a) only from the field or b) only from hatcheries. Mixing the two adds an additional variable that cannot be controlled without further replication.

- ii. The second concern is that the calculated point estimates of toxic effects are not based on the most sensitive reproductive endpoint. The point estimates are based on the endpoints of alevin survival to 15 days post-swim-up and sum total normal fry across four categories of deformity/abnormality. The EC-20s for those two endpoints are 21.6 ug/g and 21.7 ug/g egg selenium respectively (Appendix F: Table 8), yet Appendix F, Figures 12 and 13, show that the EC-100 for swim-up occurs somewhere between about 20 ug/g and 27 ug/g egg selenium. Although many of the brown trout alevins that had failed to swim-up by the time this study was terminated may have still been living (2.4-33%), in nature total failure of swim-up would be equivalent to total reproductive failure. Analysis of the swim-up endpoint for less severe point estimates of effect than the EC-100 should be explored.

Evaluation: USFWS states that “the EC-100 for swim-up occurs somewhere between about 20 ug/g and 27 ug/g egg selenium” and that the analysis of the swim-up end point should be explored. I used ImageJ to evaluate Figure 13 and calculated the following: the first data point for total failure of swim-up was approximately 26.7 mg/kg-dw and the last data point before total failure was approximately 20.5 mg/kg-dw. Thus, the EC10 for failure to reach swim up would probably be around 20 mg/kg-dw, which exceeds the lowest EC10 of 17.68 (Table 5-1).

- iii. Related to the reported observation that “... yolk fry never swam up...” (Appendix F: page 17) for the five sets of eggs with the highest selenium exposures (also recorded as 0 “% Swim-up” in Table 5-2 of the Report’s main body and illustrated that way in Figure 12 of Appendix F), it is very perplexing as to how the 15-day Post Swim-up feeding trials produced any data for those same sets of eggs (reported in Appendix F, Figure 37)? Clearly, before a 15-day Post Swim-up Feeding Trial can be conducted alevins have to

actually swim-up. If "... yolk fry never swam up...", how is Post swim-up data presented in Figure 37 generated?

Evaluation: Concur. Table 5-2 shows that the following fish had 0% swim-up but were assessed in the post swim-up survival test: LSV2C-003, LSV2C-004, LSV2C-005, LSV2C-010, LSV2C-021. This agrees with the number and approximate concentrations of eggs that did not achieve swim-up shown in Appendix F Figure 13.

- iv. The formulation of the "sum total normal fry" endpoint across four deformity/abnormality categories is not an environmentally realistic way to formulate that data [Appendix F, page 30 and Figure 44]. For example suppose five fry are each assessed for the four categories of deformity/abnormality (as per assessment methods, Appendix F, page 9) and the four effect categories are viewed once each, and each in a different one of four fry, leaving one fry that exhibits none of the deformity/abnormality effects. Then the "average" number of normal fry across the four categories is going to be 80%, 4 out of 5 fry will be free of any particular category of deformity/abnormality even though only one of the five fry in question, or 20%, will be a fully normal fry. That's a large bias in the direction of underestimating toxic effect (and overestimating EC-10 and EC-20 concentrations) because environmental realism dictates that any fry not fully free of deformity/abnormality is unlikely to survive in nature. The deformity data ought to be re-analyzed based on a "proportion fully normal fry" endpoint. Such an endpoint would be expressed on a 0 to 1 scale rather than a 0 to 4 scale.

Evaluation: Concur. The proportion of deformed fry should be calculated on a per fry basis, with each fry assigned a 0 for normal and a 1 for deformed. Thus, the "fraction normal" should range from 0 to 1.

- v. When there is no causal relationship between two variables, as is the case with whole body selenium and egg selenium (dietary selenium is the causal variable for both), the proper procedure (e.g., see Gill 1987) to back translate from EC-10 and EC-20 egg selenium concentrations to associated whole body selenium concentrations in trout would be to directly calculate a new regression equation by reversing the original X and Y axes rather than by algebraically re-arranging the original regression equation to solve for whole body selenium as a function of egg selenium as the Report does on p. 65 of the main text and on p. 33 of Appendix F. Accordingly, the whole body translations from EC point estimates for egg selenium presented in the Report and in Appendix F warrant re-calculation.

Evaluation: Concur. As long as the whole body selenium concentration data is available, the ECs should be calculated directly (e.g., in a regression of whole body concentration vs. fraction normal fry). This improves the accuracy of the calculated result (i.e., unless the r^2 for all equations is 1.0, there is some loss of accuracy).

- vi. The species sensitivity distribution (SSD) illustrated in Figure 6-1 of the Report does not appear to be scaled properly on the Y-axis. For example at the brown trout EC-20 of 21.6 ug/g dw egg selenium 1 of 7 species is affected or a proportion of 0.143, but the brown trout data point is plotted at about 0.080 on the Y-axis. Additionally, based on EPA's

current policy for tissue-based water quality criteria (i.e., Utah proposed site-specific tissue-based criterion for the Great Salt Lake and 2010 draft proposed revision to the national chronic criterion) Figure 6-1 should be re-drafted based on EC-10 values rather than EC-20 values. If it is believed that results for all the other species of fish illustrated in Figure 6-1 are valid for comparison to brown trout results from this specific study area, then typically EPA would employ such data to estimate the 5th percentile value for the SSD as an acceptably protective chronic criterion for the entire 10-species fish community in the Smoky Canyon study area (of which only two species have been tested). If it is not believed all the results illustrated in Figure 6-1 would necessarily apply on a site-specific basis to the study area then the only site-specific sensitivity conclusion that can be reached is that brown trout are more sensitive than Yellowstone cutthroat trout. Thus, either a 5th percentile value should be calculated for the SSD presented in Figure 6-1, or a safety factor should be applied to the brown trout toxicity results to acknowledge that only two of ten site-specific species were tested for toxic sensitivity to selenium.

Evaluation: this is outside my area of expertise.

6. UNADDRESSED ISSUES

- A. Compliance with the Clean Water Act mandates water standards that will provide for the "... protection and propagation of fish, shellfish, and wildlife... (PL 92-500, Sec.101(2)). The Report is silent on all taxa other than fish and aquatic invertebrates. Recent research (Presser and Luoma 2010) reveals that fish whole body selenium concentrations are consistently about 1.1 times the selenium concentrations of co-located aquatic invertebrates. The Report asserts that the proposed site-specific fish egg criterion (21.6 ug/g dw) would translate to about 14 ug/g fish whole body selenium, which, divided by 1.1, would translate to about 12.7 ug/g dw aquatic invertebrate selenium. Based on Ohlendorf's (2003: Figure 17.1) regression of mallard egg hatchability against dietary selenium exposure of hens the EC-10 is only 4.9 ug/g dw. Mallard hens, along with the majority of other species of water birds, rely heavily on aquatic invertebrates during the breeding season in order to meet the high protein requirements of ovulation. For a dietary exposure of 12.7 ug/g dw dietary selenium, Ohlendorf's regression equation predicts a point toxicity estimate exceeding 85% failure of mallard eggs to hatch. Thus, it seems highly doubtful that the proposed site-specific criterion would comply with the Clean Water Act's mandate to protect wildlife. As the studies of Skorupa et al. (2002) and Vasterling (2003) have documented, in both riparian and wetland systems in the phosphate-mining region of southeastern Idaho, avian eggs exhibit elevated selenium concentrations commensurate with the system wide levels of selenium contamination for species of birds across a spectrum ranging from blackbirds to mallards to geese to sandhill cranes.

Evaluation: Concur. Presser and Luoma (2010) also conclude that "based on typical TTFs [trophic transfer factors] for Se, USEPA's proposed whole-body fish tissue criterion of 7.9mg/g dw (USEPA 2004) would also allow Se concentrations in aquatic invertebrates that, when eaten by breeding waterbirds, would pose a substantively higher hazard (see, e.g., Ohlendorf 2003; EC50) for avian toxicity than the designed level of protection for fish..."

- B. Employing brown trout as a sensitive surrogate for all the untested species of fish in Crow Creek, the goal would be to keep the selenium concentrations in eggs of all species below

the brown trout-based site-specific criteria of 21.6 ug/g dw (for the moment neglecting whether this particular value is defensible or not, see recalculations below). This would be straight forward to monitor and manage if compliance monitoring were going to be based on direct measures of selenium in fish eggs. But for practical reasons (difficulty of sampling ripe female fish, as for example at the reference site) it is actually whole body selenium that is likely to be monitored. This raises the question of what whole body value would generally keep egg selenium below the target value in most (if not all) local species of fish? Determining this is not as simple as merely answering what whole body selenium concentration is likely to keep brown trout eggs below the target value and then extrapolating that to all other species of fish, as is done in the Report. The Report is silent on the rationale for this. Just as species of fish vary in their toxic sensitivity to selenium exposure, there is also a great deal of interspecies variability in the partitioning of selenium between different tissues. Studying a community of Colorado River Basin fish species, Osmundson and Skorupa (2011) found that simple ratios of egg to whole body selenium vary among species from average ratios of about 1 to 8. The 75th percentile value was about 4. The Report found a ratio for brown trout, at the EC-20 target for eggs of 1.54, i.e; the eggs contained about 1.54 times as much selenium as a whole body analysis would yield. Thus, a proposed egg target of 21.6 ug/g dw “translates” to a whole body target of $(21.6)/(1.54) = 14$ ug/g dw. However, a ratio of 1.54 is on the low end of the range of species variability for such ratios documented for 12 species of fish in the Colorado River Basin. If species variability of ratios is similar for the 10 species of fish in the Smoky Canyon study area, then a whole body target of 14 ug/g could result in most species of fish (60-70% in the Colorado River Basin data set) actually exceeding the 21.6 ug/g egg target (some species by a lot). The way to protect against this potential problem is to develop a site-specific set of tissue ratios for the 10 species of local fish (or most of them) and then select an appropriately protective translation ratio from that data set. Presumably such a local data set could be obtained within a spawning season or two of field sampling, as was achieved for the study of the Colorado River Basin fish community reported in Osmundson and Skorupa (2011). Alternatively, data from studies such as Osmundson and Skorupa (2011) could be employed to estimate a conservative ratio, for example Osmundson and Skorupa’s 75th percentile value. In any case, arbitrarily extrapolating the brown trout tissue translation ratio to all other species of fish is inappropriate and could lead to substantive loss of the presumed protectiveness of a site-specific criterion.

Evaluation: Concur. However, if using ratios from fishes other than those at the site, it would be more protective to use the 95th percentile, rather than the 75th.

7. RECOMMENDED RECALCULATIONS

- I. Hatch to 15-days post swim-up fry survival endpoint
 - a) This toxicity response endpoint for the brown trout toxicity study (Appendix F of the Final Report) is based on raw data presented in Appendix A of the brown trout toxicity study (i.e., Appendix A of Appendix F) in Tables 3-3 and 3-4. The specific endpoint is the “Total Survival %” from Table 3-4 divided by the “% Hatch” from Table 3-3. For example, for control sample SPC-006 these figures would be $[(96.0) / (99.7)]$ for a survival proportion of 0.963 from hatch until test termination at 15-days post swim-up. For reasons that this reviewer was unable to determine, 27 of the 36 reported survival proportions for this endpoint reported in Table 3-4 are incorrect. Of the 27 incorrect values, 24 are incorrect high and 3 are incorrect low. In some cases the discrepancy is

quite substantial. For example, for sample LSV2C-004 the reported endpoint survival proportion is 0.662 (66.2%) even though the reported “Total Survival %” in Table 3-4 of 16.8 and reported “% Hatch” from Table 3-3 of 50.6 actually yield a “hatch to termination” endpoint survival proportion of 0.332 (33.2%; i.e., 16.8 / 50.6). Thus, to begin with for this recommended recalculation the 27 incorrectly reported results were corrected.

Evaluation: The tables in the Interpretive Report and its appendices have headings that do not accurately reflect the data in those tables. For example, one of the column headings is “%hatch,” however, this is defined in the text of Appendix A of Appendix F page 2-10 “as the number of live fish and alevins at day of first hatch compared to the number of eggs at test initiation.” Thus, the actual number of eggs that hatched should be higher than the %hatch shown in the tables. As defined this way, I am unsure of the utility of “%hatch.” Further, if additional eggs hatched after the day of first hatch, USFWS’ re-calculation is invalid and the data cannot be reliably evaluated until all endpoints are clearly defined and the actual raw data is presented in terms of the number of individuals surviving at each stage.

Below, I list the endpoints from Tables 3-1, 3-3 and 3-4 and indicate which endpoints are unclearly defined in the Interpretive Report:

1. #Eggs placed in study: self-explanatory
2. Day of first hatch: self-explanatory
3. %hatch
 - a. Defined as “Percent hatch was determined as the number of live fish and alevins at day of first hatch compared to the number of eggs at test initiation.” Therefore, this column in the tables should be labeled something like “%hatch on 1st day”
 - b. a second column should be added showing the total number of individuals that hatched and labeled “%hatch”
4. Day of swim up: please define more completely. Is this the day that the first individual reached swim-up or the day that some % reached swim-up?
5. % Swim-up: the number of individuals that reached the swim-up stage
6. Survival (%) at Swim-up Stage: this appears to be defined as the number of individuals alive when some % of those individuals have reached swim-up, including individuals that have not reached swim-up. However, I do not think that this variable is useful and should be deleted.
7. Survival (%) in 15-d Post swim-up stage:
 - a. This variable is self-explanatory
 - b. However, in order to be transparent, the tables need another column to explain this variable; i.e., “# in 15-d Post swim-up test”
8. Total Survival (%): it is unclear how this variable has been defined. Using SC-001 as an example, if there were 600 eggs and 22.8% reached swim-up, then 137 individuals reached swim-up. If 100 of those individuals were then placed in the 15-d post swim-up test and 97 survived, to determine the number that are assumed to have survived to 15-d after swim-up, one would multiply 97% by 137, to get 133. However, 133/600 is 22.2% and not the 22.7% shown in Table 3-4. Please define how this was calculated.

Please also note that this is an estimated number and not an actual observation.

9. Survival (%) from Hatch until test term.: self explanatory. But, it should also be noted that this is an estimated number and not an actual observation.

I have translated these variables into number of individuals on the following page for SC-001 and CC-150-009 to show what the raw data tables should look like. Then, the percentages can be calculated from this in a manner that is completely transparent to the reviewers. Note that because some of the data was not provided, there are some “?” in my example tables.

Suggest raw data format:

Variable	1	2	3a	3b	4	5	7b	7a	8*
Treatment	#Eggs placed in study	Day of 1 st hatch	#hatch on 1 st day	#eggs hatched	Day of swim up	# reached swim-up	# entered into post swim-up test	# alive at 15d post swim-up	Estimated total survival (#)
SC-001	600	42	143	?	69	137	100	97	133
CC-150-009	600	39	171	?	72	163	100	99	161

Notes: * calculated as $5 \times (7a/7b)$

Suggested table format for presenting percentages

Calculation	3a/1	3b/1	5/1	7a/7b	8/1	8/3b
Treatment	%hatch on 1 st day	%eggs hatched	% reached swim-up	Survival (%) in 15-d Post swim-up stage	Estimated total survival (%)	Survival (%) from Hatch until test term.
SC-001	23.8	?	22.8	97	22.2	?
CC-150-009	28.5	?	27.2	99	26.8	?

- b) Next, there were five samples (LSV2C-003, LSV2C-004, LSV2C-005, LSV2C-010, and LSV2C-21) that did not produce any swim-up fry by the time the experiment was terminated. Instead of recording a post swim-up fry survival of 0.0% for those samples, the authors of the final report chose to record the percent survival among the developmentally “dead-ended” alevins. By doing this, they are assessing a post swim-up fry endpoint for most of the samples and a developmentally completely different dead-end alevin endpoint for the five samples mentioned above. Mixing two different endpoints for exposure-response modeling fundamentally invalidates the modeling. This error is easily corrected by using only the post swim-up fry endpoint and recording a post swim-up fry survival of 0.0% for the five samples in question.

Evaluation: Concur.

- c) The authors excluded data from the hatchery control samples because the first set of hatchery controls performed very poorly with regard to % hatch and the second set of hatchery controls were obtained as eyed embryos and therefore weren't comparable to the starting point for all the other samples (started as undeveloped fertilized eggs). While the second set of controls yielded excellent % hatch performance, that may have been due to the advanced stage of the embryos at receipt and thus much shorter holding time to hatch (9-13 days) than for any of the other samples (which had holding times to hatch of 37-47 days). In addition to those data exclusions, there were 10 additional samples with missing test organisms because of clogged drain pipes that caused water to overflow out of the test system, apparently carrying some test organisms away (see Appendix A of Appendix F). The authors do not know how many of the carried away organisms were dead or alive, deformed or normal, or anything else about them. The authors chose to simply ignore the missing test organisms, effectively assuming the proportion of missing fish that were live or dead and deformed or normal was exactly the same as those proportions in the fish left behind. There is no valid justification for such an assumption. Best scientific protocol dictates that the uncertain results for these 10 samples also be excluded from exposure-response modeling.

Evaluation: Concur.

- d) Thus, the valid data set for modeling the “hatch to 15-days post swim-up fry survival” endpoint is as follows (Table 1; log transformation of exposure data is performed on ppb exposure instead of ppm exposure to avoid negative logarithms): [table not shown here]

Evaluation: as presented in above, I think that USFWS is assuming that the definition of “%hatch” is the total number of eggs that hatched, instead of the number of eggs that hatched on the first day of hatching (which was used in the Interpretive Report). Therefore, I do not think that the calculated numbers presented by USFWS are correct. The authors of the Interpretive Report should weigh in on this and provide tables as I have suggested above for re-evaluation by USFWS.

- e) The authors of the Report employ EPA's Toxicity Relationship Analysis Program (TRAP) for modeling exposure-response. However, TRAP offers several options with regard to statistical models. The authors chose to use the Least-Squares Nonlinear Logistic

Regression model (see Appendix E of Appendix F). EPA advises in the TRAP program overview that the Least Squares Logistic Regression model is most appropriate for endpoints that are (1) measured on a continuous scale (for example weight or height) and (2) expected to have infinite tails approaching 0% and 100% response. Neither of these conditions applies to the brown trout data in question. The data in question are (1) non-continuous dichotomous (binomial) data (each hatched egg either goes on to become a 15-day post swim-up fry or does not) and (2) the endpoint would be expected to have a finite “threshold” response in which there is no response until a minimum finite threshold exposure is experienced, not an infinitely gradual response tail. For dichotomous, threshold response data, EPA advises that the most appropriate TRAP model choice is the Tolerance Distribution Analysis fitted to a Triangular Distribution. Therefore, for this recalculation those are the modeling options that were chosen for TRAP exposure-response modeling with the following result (Figure 1): [figure not shown here]

Evaluation: Concur, TRAP does recommend a triangular distribution for dichotomous data. However, I would suggest that USEPA’s BMDS (see <http://www.epa.gov/ncea/bmnds/>) or Cetus (<https://www.tidepool-scientific.com/index.htm>) be used instead. These other programs have several more dichotomous models than TRAP which will allow the authors to determine which model best fits the data instead of assuming that a logistic model is the best fit.

- f) The lack of useable data points within the intermediate response range violates the TRAP program requirements for model fitting, generating an error warning message. The EC-10 and EC-20 estimates of 14.9 and 16 ppm shown above are the result of forcing a model fit and should not be viewed as reliable. Recalculation for this endpoint reveals that the useable dataset is not sufficient for reliably estimating any EC-10 or EC-20 values. The only reasonably certain information that these data yield is that the finite threshold for total response, the EC-100 threshold, occurs somewhere between an egg selenium concentration of 14 ppm and 27 ppm. Because 100% response (i.e., total failure of brown trout alevins to achieve swim-up) is already being expressed at an egg selenium concentration of 27 ppm, and because valid estimates of the EC-10 and EC-20 cannot be modeled from the useable dataset, proposing an egg selenium standard of nearly 22 ppm (as the authors do in the final report) based on an invalid EC-20 estimation (as the authors do in the Report) does not seem very defensible. Any site-specific standard proposal higher than 14 ppm egg selenium cannot be defensibly supported by the “hatch to 15-day post swim-up fry survival” endpoint.

Evaluation: As indicated above, I think that the authors have defined “%hatch” in a different manner than assumed by USFWS and, therefore, any recalculations based on the % or # of individuals that hatched should wait until the authors clarify their endpoints and provide the raw (i.e., number of individuals) data. However, if USFWS is correct in their calculations, there is a data gap in that there are no data points between almost total survival and almost total mortality. In that case, almost all dichotomous models should fit the data and the EC10 or EC20 will vary depending on what model is chosen to fit the data.

II. Larval Deformity Endpoint

The collection of larval deformity data was seriously flawed in at least two ways.

- a) First, the larvae assessed for deformities were not a random subsample of all larvae. Alevins and post swim-up fry that died during the brown trout toxicity study mostly did not get assessed for presence or absence of deformities (see Section 2.5 of Appendix A to Appendix F). Since deformed larval fish can be expected to have a differentially high propensity to die compared to undeformed larval fish, all the measures of percent deformed larvae are likely biased low. For example, among a limited number of test groups (samples LSV2C-004, LSV2C-005, and LSV2C-021) deformity assessments are available for reasonable samples of both dead and live larval fish as follows (Table 2 from raw data presented in the first three of the un-numbered Tables in Appendix C of Appendix F): [table not shown here] As the above comparisons show, the bias introduced by assessing mostly live (surviving) larval fish for deformities can be substantive. However, the comparisons shown above are for test samples that had high selenium exposures (27-36 ppm egg selenium) and presumably therefore would express this type of bias to a greater extent than test samples from lower exposures would. Presumably, lower exposures would not uniformly produce exclusively (100%) deformed dead larval fish. Since the authors did assess greater numbers of dead fish for the five highest exposure groups and the lower exposure groups often yielded predominantly surviving larval fish, it is possible that the overall magnitude of this bias on exposure-response modeling for larval deformities is relatively modest. Nonetheless, the main points to consider are that the exact amount of bias is unknown, and whatever the magnitude of bias is it is certain to be in only one direction, underestimation of response values (thus overestimation of toxic threshold concentrations).

Evaluation: the table provided by USFWS shows the percentage of deformed live and dead fry. The number of deformed dead fry was taken from the first un-numbered table in Appendix C of Appendix F. However, the number of deformed live fry, as presented in the table generated by USFWS, is not presented in Appendix C of Appendix F. USFWS appears to have generated this number by subtracting the number of deformed dead fry in the first table in Appendix C of Appendix F from the number of fish from the second table in Appendix C of Appendix F. The second table does not indicate whether it is displaying the results for live, dead, or live and dead fry. Some values in the second table are also underlined and some sample IDs are highlighted, which are not explained. If USFWS is correct in their assumption that the second un-numbered table is for both live and dead fry, then there were no craniofacial deformities in the live fry for LSV2C-004; i.e., the number of deformed fry in the second table is $6+80+15=101$, which is the same as is shown in the first table. Nonetheless, if USFWS assumption is correct that the second table shows both live and dead fry, then dead fry may be expected to have higher deformity rates than live fry and the loss of some dead fry due to decomposition etc. can be expected to lead to an under-estimate of deformity rates for those samples that lost dead fish due to decomposition.

- b) Second, each of the four types of deformity examined were assessed and reported completely independently of each other (see Appendix C of Appendix F). Thus, while it is possible to determine what percent of assessed larval fish were normal for each type of deformity, it is not possible to determine what percent of assessed larval fish were

completely undeformed (i.e., were fully “normal” larval fish). Unfortunately, the raw deformity assessment data were not available for review, but were cited (see p. 3-10 of Appendix A of Appendix F) as being presented in a separate document (never made available to this reviewer). The authors of the Report used an endpoint that they called “sum total normal fry” which in reality should more accurately have been called “sum partial normal fry” because it is unknown if any of the fry in this sum are simultaneously normal for all four types of deformity. It is only known that the fry in this sum are normal for at least one of the four types of deformity (craniofacial, vertebral, fin, edema). Of course the sum of partial normal fry will necessarily overestimate the sum of fully normal fry and it is only fully normal fry that can be expected in nature to be free of any deformity-related toxic impairment. A recalculation that can be done here to partially correct for the overestimation of fully normal fry is to recognize that the proportion of fully normal fish cannot have been any higher than the lowest proportion of normal fish in any of the four deformity categories reported in Appendix C of Appendix F. This will be referred to as “maximum fully normal fry”. That endpoint should be a much better approximation of percent fully normal fry than the author’s “sum partial normal fry” endpoint. Also, for this recalculation the second set of hatchery controls provide useable estimates of expected background deformity rates because the hatching rates were all acceptable control values (i.e., >90% hatch) and from hatch to 15-days post swim-up fry these controls are strictly comparable in their holding times to the field-exposed test groups. However, the results from the “accidental overflow” test samples must again be excluded from analysis. This yields the following data set (Table 3) for modeling the response variable of larval deformity (log transformation of exposure data is performed on ppb exposure instead of ppm exposure to avoid negative logarithms): [table not shown here]

Evaluation: Concur. I checked the first two entries on the table generated by USFWS and found them to be correct.

- c) This is a repeat of Comment 7.1.e above. Please see response above.
- d) This dataset met all TRAP program requirements for a clean model fit without any error warnings. The estimated EC-10 is 6.5 ppm egg selenium and the estimated EC-20 is 9.0 ppm egg selenium. These estimates are not fully corrected for the “sum partial normal fry” bias compared to proportion fully normal fry and therefore are still overestimates of the EC-10 and EC-20 for the larval deformity endpoint. Even so, both of these recalculated estimates are substantially lower than the site-specific standard of almost 22 ppm egg selenium proposed by the authors of the Report. This endpoint also illustrates why it is so crucial to eventually obtain valid field control data. If the hatchery control data points are omitted, and it is erroneously assumed that the CC-150 and CC-350 (upstream) sampling sites are valid field control sites then the estimates of the EC-10 and EC-20 for larval deformity respectively increase to 15.2 and 17.5 ppm egg selenium as illustrated below (Figure 3): [figure not shown here] Obtaining valid field control results for the larval deformity endpoint should be a very high priority for future research.

Evaluation: Concur. IDFG should also be consulted to determine which Creeks have the most genetically similar fish to avoid potential population-specific differences in the responses to selenium.

- III. Regressing X on Y for Estimation of Critical Whole Body Tissue Concentrations. Because of the distinctly species-specific nature of selenium partitioning between tissue types within individual fish (Osmundson and Skorupa 2011) and because of the complex spatio-temporal biology of selenium deposition into brown trout eggs which can be expected to differ in a hatchery versus in the field (Estay et al. 2003), this recalculation is based only on the field data for brown trout as presented in Table 5-2 of the final report. Because tissue concentrations from polluted environments typically conform to lognormal distributions, the regression recalculated here is calculated on log-log transformed data. The recalculated regression is illustrated below (Figure 4). The regression is highly significant ($p < 0.0000001$) and the adjusted R-square value for the regression is 0.788. This regression yields a critical whole body selenium concentration of 12.32 ppm at an egg selenium concentration of 21.63 ppm, a result that is slightly lower than the value of 13.35 ppm critical whole body selenium reported for brown trout data in the Report based on algebraic re-arrangement of the egg selenium on whole body selenium regression equation (see p. 65). When the recalculated regression above (Figure 4) is evaluated at the EC-10 for maximum fully normal fry (Figure 2) of 6.5 ppm egg selenium it yields an estimated critical whole body selenium concentration of 5.5 ppm.

Evaluation: I tested the distributions of the brown trout whole body and selenium concentration data collected from Sage and Crow Creeks shown in Table 5-2 using USEPA's ProUCL v4.1.0.1 (<http://www.epa.gov/osp/hstl/tsc/software.htm>). Neither data set fit a normal distribution, but both fit a log-normal distribution, as assumed by USFWS. I also re-calculated the regression presented by USFWS and got the same result. Using this equation, 6.5 ppm selenium in eggs does indeed convert to 5.5 ppm in whole body. Converting the proposed site-specific criterion (see Page 71) of 21.63 mg/kg-dw in eggs using the equation derived by USFWS yields 12.33 mg/kg-dw whole body whereas converting the site-specific criterion using the equation on page 65 yields 14.0 mg/kg-dw whole body. Overall, I concur that it is better to re-calculate the regression from the raw data rather than to algebraically re-arrange the equation.

- IV. Test of Assumption that the 14 ppm Proposed Critical Whole Body Tissue Concentration Derived from Brown Trout Tissue Partitioning Data Would Generally Keep Egg Selenium Values Below a Critical Value of 21.63 ppm Across a Multi-species Community of Fish. Tissue partitioning ratios from elevated selenium exposure can be expected to be dose-dependent. Therefore to examine this issue using the fish community dataset from the Colorado River Basin reported in Appendices 1 and 2 in Osmundson and Skorupa (2011), only the fish samples with egg selenium results relatively near (in the range of 15 to 30 ppm) the target value of 21.63 ppm proposed in the Report were evaluated. The results are presented below (Table 4): [table not shown here]. Thus, for the Colorado River Basin community of fish, assuming that a critical whole body concentration of selenium derived from brown trout data would be universally applicable would have allowed other species of fish to reach egg selenium concentrations as high as 138 ppm, or more than 6-times the proposed site-specific standard of 21.63 ppm. Moreover, the overall exceedance rate would be 68% (weighting equally each species exceedance rate in Table 4). In other words, instead of the 80% protection afforded to brown trout by an EC-20

standard, the protection afforded to the rest of a multi-species fish community in this case would be only 32%. Consequently, in this case, assuming that all fish species partition selenium between tissues comparably to brown trout would be a very under protective assumption. This exercise does not prove that such an assumption would also be very under protective as applied to the Crow Creek fish community, but it does emphasize that making such an assumption for the Crow Creek fish community (as is done in the Report) would seem unlikely to hold true and therefore must be validated before it can be accepted for regulatory purposes.

Evaluation: On page 71, the Interpretive Report proposes a site-specific criterion of 21.63 mg/kg dw egg selenium based on brown trout. A whole-body criterion is not proposed. Using the equation proposed in the Interpretive Report on page 65, this would convert to a whole body concentration of 14.0 mg/kg while using the equation derived by USFWS, this would convert to a concentration of 12.33. Thus, USFWS is correct in comparing expected whole body concentrations in fish from the Colorado River to 14 mg/kg-dw. I also checked USFWS calculations in the table they provided. There was some rounding in the table presented by USFWS that was not present in the original report but I matched all but one of the expected whole body concentrations using USFWS numbers. Thus, USFWS is correct that an egg concentration of 21.63 mg/kg-dw may allow some species to have whole body concentrations over 14 mg/kg-dw. Overall, I concur that a selenium criterion of 21.63 mg/kg-dw in eggs may not be protective of the other species of fish in Crow and Sage Creeks.

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**Peer Review of the FWS Technical Review Document based on the report entitled:
*Interpretive Findings for Field and Laboratory Studies and Literature Review in Support of a Site-Specific Selenium Criterion, Smoky Canyon Mine (August 2010)***

Peer reviewer: David M. Janz, Ph.D

Date: October 25, 2011

Preamble: I am a Professor of Veterinary Biomedical Sciences and Acting Director of the Toxicology Centre, University of Saskatchewan, Canada. I hold a PhD in Pharmacology and Toxicology, a MSc in Environmental Resource Studies and a BSc in Ecology. My main area of research is aquatic ecotoxicology, with a focus on developmental and reproductive toxicities in fish. I have conducted field and laboratory research with over 20 species of fish, including four salmonid species, over the past 25 years. I have published 76 peer-reviewed scientific articles, including 22 on the aquatic toxicology of selenium in fish. I should also state that since 2003 I have been Principal Investigator on three large grants investigating the aquatic ecotoxicology of selenium that were sponsored by the mining industry; thus I am cognizant of the regulatory issues and “sensitivities” associated with this trace metalloid. Overall, I feel very well qualified to provide this peer review on the FWS Technical Document. My goal from the outset is to provide a balanced review based on the principles of sound science. In addition, I should state that I have no conflict of interest in any way related to this review.

The Smoky Canyon Mine report provides extensive documentation in support of their application to establish an interim site-specific Se criterion to protect aquatic-dependent life downgradient of the mine, specifically Se-laden groundwater discharge into a series of creeks that support fish communities. Significant financial resources have been devoted to the field and laboratory research conducted to develop this report, and as a scientist who does similar work I acknowledge the effort required to produce this report.

Selenium is known to cause a spectrum of adverse developmental effects in early life stages of fish, which have the potential to impair recruitment into fish populations and thus sustainability of natural fish populations and communities. The experimental studies found within the Smoky Canyon report are thus focused on these toxic responses in two resident salmonid fishes, brown trout and cutthroat trout. Furthermore, the FWS Technical Document is focused on the scientific soundness of these experiments and the resulting data analyses. My peer review will proceed through the FWS Technical Document and provide an assessment of the validity, clarity and fairness of the criticisms found within, as well as other comments related to the document and report. Throughout my review, I will refer to the Smoky Canyon Mine report as the “report” and the FWS review as the “document”.

1.0 Introduction

Paragraph 1: I agree with this comment and find it strange that only a preliminary analysis of the YCT data are provided given the extensive nature of the report. Perhaps there was a deadline and these analyses could not be finalized by that time? Given the length of time

between report submission, FWS commentary and this peer review, one would think that by now the YCT dataset would be finalized and included. Nevertheless, the FWS document is directed solely on the brown trout dataset and thus my comments will focus on that aspect.

Paragraph 2: I agree with this comment, although a criterion based on aqueous Se concentration implies aqueous exposure as the primary route of uptake in fish, as is true for most other trace elements but not Se. Thus the flaw with an aqueous Se criterion for Se. I agree that the report neglects to acknowledge that dietary exposure at Belew's Lake was the major route of exposure, and that this was "integrated" into the 1987 USEPA criterion of 5 µg Se/L. This can easily be clarified in the report.

Paragraph 3: I agree with the first statement, and will expand this to say that in my opinion the literature review provided in the report is superficial in many respects and indicates a relative lack of knowledge of the current state of understanding with respect to the aquatic ecotoxicology of Se. The report should explicitly acknowledge the differences in experimental design between the bluegill and trout studies.

Paragraph 4: I do not completely agree with this comment. Although it was an unfortunate oversight in the follow-up study with bluegill that photoperiod was not appropriately controlled, water temperature is the major driver of physiological responses in poikilotherms such as fish. In my opinion the follow-up study is not irrelevant and in fact certain aspects of Lemly's (1993) study were consistent (i.e., reduced survival in fish at 4°C compared to 9°C) even though growth and lipid stores did not differ between temperature treatments. In addition, since Lemly (1993) was also a laboratory study, what applicability does it have to free-ranging fish? I would suggest "softening" this comment to address the uncertainties in both the McIntyre (2008) and Lemly (1993) studies.

2.0 Site Setting

My comments concerning appropriateness of the upstream "background" sampling sites can be found below under section 4.0.

3.0 Literature Review

I agree with the FWS reviewer that certain aspects of this section were "uneven", and the example given (Besser and Kennedy studies, and associated critiques) is a good indication of this. I also found the literature review to be very superficial, demonstrating limited knowledge of Se biogeochemistry and toxicity. I could provide many detailed comments here, but since the FWS document does not focus on this, then neither will I.

4.0 Field Monitoring

Paragraph 1: Although the sulfate argument has often been used to downplay the potential bioavailability of selenate to food webs, I agree with the reviewer's comments in the document that its relevance in field situations is not established. The only ecologically (field) relevant competition between sulfate and selenate would potentially occur during uptake into algae (and possibly other primary producers such as bacteria and fungi), although this is not clearly

established. If true, sulfate may reduce the bioavailability of selenate to algae, and thus the enrichment of Se in aquatic food webs. However this seems to me a moot point since it is clear from the near field study sites that sufficient Se is being assimilated and biotransformed by primary producers to cause diagnostic features of toxicosis in resident fish (terata). I agree that the presence of elevated sulfate at the study sites has no bearing on developing a site-specific criterion.

Selenate can theoretically be taken up across the intestine of fish *via* a sulfate transporter; however this is certainly not relevant for freshwater fish as they do not drink appreciable volumes of water (unlike marine teleosts). Furthermore, sulfate transporters have not been identified in fish gills, so there is no basis for competition between the sulfate and selenate anions at the gill-water interface, as determined for certain cations using biotic ligand modeling approaches. Very little is known in invertebrates. The most relevant study cited by the FWS reviewer is Williams et al. (1994) since it was focused on algae. The Hansen et al. (1993) study is of questionable significance since it was conducted in benthic and pelagic invertebrates, which, like fish, will obtain the majority of their Se body burden *via* diet.

Paragraphs 2-5: I agree with the reviewer that there are significant (statistically and toxicologically) differences in whole body Se concentrations between the “reference” and “background” (i.e., upstream) sites. Indeed the data in the report show this, as indicated by the reviewer. The 2.56 µg/g dw value for Tincup Creek fish can be considered “normal”, while the 6-9 µg/g dw range for Crow Creek and Deer Creek sites is greater than what could be considered even “high normal” and in fact is approaching or at a toxicity threshold value for certain fish species.

The paragraph discussing the extended period of brown trout vitellogenesis and lack of differences in periphyton Se concentrations between reference and background sites is key to this entire section and the argument that brown trout are likely migrating between the exposure and background sites within the same open watershed/drainage. I strongly agree with these assessments made by the FWS reviewer. The reviewer may also consider including mention of recent work by Vince Palace’s group using laser ablation ICP-MS in otoliths to record Se exposure chronology in free-ranging trout inhabiting lotic systems in a coal mining region of Alberta (Palace et al. 2007).

In reading the report, I was not able to find any acknowledgement of even the possibility of trout migrating between exposure and background sites. This should be added to the report.

In this section, and throughout the FWS review document, I recommend explicitly stating “whole body Se” when referring to the report data to avoid confusion (instead of, for example “trout tissue selenium value”). I also recommend not using ppm and ppb as units of concentration; use µg/g and µg/L, respectively.

Paragraphs 6-9: I strongly agree with the statements provided in this section. The mere presence of fish in an aquatic system does not necessarily constitute stable, sustainable

population dynamics. Reduced fish abundance at an impacted site can reduce competition for resources (abiotic and biotic) and thereby act as an attractive “sink” for immigration. It should also be noted that using electrofishing to obtain CPUE data is problematic among sites with differing conductivity, since conductivity has a major influence on the ability to stun fish. A combination of classic mark-recapture studies at these sites, ideally in combination with otolith microchemistry (LA-ICP-MS, mentioned above) would provide a much more robust way to evaluate any “obvious negative impacts to fish populations”.

In section 4.6 page 46 of the report it is stated that benthic invertebrate abundance and richness did not differ among sites. It should be noted that this is commonly observed in such field studies and is not an indication of “no impact” of selenium, since benthic invertebrates are tolerant of high Se exposure. The key question is not what the abundance/diversity of invertebrates is, but what the Se concentrations are in these benthic invertebrates (see Table 13, Appendix A), since they provide the majority of trout dietary items.

5.0 Laboratory Studies

This is an important section of the document and I have read Appendix F and Appendix A of Appendix F very carefully.

I will say from the start that I am concerned about the very high and variable embryo mortality across all sampling sites and the first hatchery collection. I assume this was due to fungal growth as is common when aseptic technique is not followed closely. Of concern is the documented use of NaCl and especially formalin to control fungal growth, and how this might influence endpoints. Overall, the highly variable mortalities would appear at first glance to hamper the ability to determine robust concentration-response relationships. In addition, the study suffers from the problem of having most data in the “normal” range (although the upstream “background” sites cannot be considered “normal”) with relatively few data describing the threshold for adverse effects (similar to Holm et al. 2005). However, these are the data that were collected, and this is field work after all.

I strongly agree with the first concern brought forth by the reviewer, that using responses obtained from upstream “background” site trout, with their relatively high Se body burdens, will likely overestimate EC10/EC20 values determined for exposure site trout. I agree that hatchery data should be used for comparative purposes, and perhaps a composite of the hatchery and upstream site data could be used if possible. Are the hatchery trout of the same strain as the wild trout? This needs to be considered before using hatchery data. Ideally, obtaining spawning brown trout from a true reference site in this region is the best approach. Looking at the maps supplied in the report, there doesn’t seem to be a paucity of potential locations to collect such fish. Although not ideal, conducting a study in a separate year using a proper reference site would seem to be a good approach, perhaps in combination with additional sampling of brown trout from the near field exposure sites to increase the sample size within the critical (“threshold”) portion of the concentration-response relationships.

With respect to the second concern (page 7 of document), I agree in part but have some comments to consider.

I have a major problem with use of the endpoint “survival to 15 days post-swim-up”, purported to be a measure of the ability to transition from endogenous to exogenous feeding. I do not doubt the ecological relevance of switching to exogenous food as an important life history trait in oviparous fish species. However, to my knowledge this has not been demonstrated in any previous studies to be a sensitive endpoint associated with elevated Se exposure. Just because the data for this endpoint provide a good “fit” using the dose-response software doesn’t necessarily mean that they are toxicologically meaningful. My reasoning for this is found below.

Elevated Se has been shown to alter intermediary metabolism related to lipid and carbohydrate homeostasis in mammals (Mueller et al. 2008), which has recently been at least partially confirmed in fish (Thomas and Janz 2011; Wiseman et al. 2011). Thus, it is distinctly possible that fish with higher Se exposure during development may have altered appetite in response to this metabolic phenomenon, such as the fry fed for 15 days following swim-up. If true, this would confound the results obtained, since fish with greater Se exposure may eat more readily compared to controls. In addition, I could find no details on the methodology for feeding fish post-swim-up (e.g., type of food, feeding ration, feeding frequency, removal of uneaten food, etc). Feeding studies must be very carefully controlled in order to obtain meaningful data. The most relevant type of feeding study would employ live prey (e.g., zooplankton) as food items to mimic natural conditions.

I am confused with the reviewer’s assessment of “sum total normal fry” vs. “fraction normal fry”. From what I can see the report does appear to use fraction normal, not sum. However after spending much time reading through the report, it was not clear to me how exactly this was calculated. The reviewer makes a strong case (paragraph 3, page 7, starting with “The formulation of the “sum total normal fry”...) for the apparent problems with the manner which the deformities were expressed. I agree with this assessment, and it appears that the report may not be presenting the data in the proper way. My own rationale for this can be found in the following paragraph (which could possibly just be a different way of saying what the reviewer states in this section of the review). I think the wording in this section of the FWS review could be worked on for clarity.

In my opinion, the analyses for deformities should be performed as follows. The various abnormality categories (skeletal, fin, craniofacial and edema) do not usually occur simultaneously in fish; usually just one or two is evident except at very high exposures. It can be assumed that the presence of just one deformity category will have negative consequences on fish survival in the wild. As stated by the reviewer, “environmental realism dictates that any fry not fully free of deformity/abnormality is unlikely to survive in nature”. I strongly agree with this statement. Thus, in my opinion data should be evaluated as simple presence or absence (frequency) of any specific deformity; if any deformity was present, that individual fry would be assessed as not normal. As far as edema, the report states that this can be reversible and may not influence survival, as stated in Appendix C of Appendix F (“Edema was not originally

scheduled for assessment because it was thought sometimes not a teratogenic effect and may be transitory as fish develop”). I personally tend to disagree with this given the severity of edema often observed; see photographs in Appendix C of Appendix F. Given the uncertainty surrounding edema as a relevant endpoint, the data could be analyzed both excluding and including edema as a response category for deformities. I doubt that the end result in terms of EC10 would differ substantially whether edema was included or not, but this would be worth evaluating.

In paragraph 2, page 7, (“yolk fry never swam up”), I am equally confused on this point and spent considerable time trying to make sense of it. The group LSV-2C had an original sample size of n=14 fish. Did the report authors remove the data from n=5 of these fish, where no hatched fry successfully swam up, in Figure 37 of Appendix F? (These data are also shown in Figure 3-1 on page 3-8 of Appendix A of Appendix F). This needs to be clarified in the report. It is important to note here that these five groups of embryos that did not swim up represent very toxicologically meaningful data, since the toxicity of maternally transferred Se occurs mainly during yolk resorption. Were these five groups of embryos used for deformity frequency assessments? If not they should be. This was not clear to me from the report.

Final paragraph of section 5.0 (species sensitivity distribution): I agree with the reviewer’s comments and suggested reworking of Figure 6-1. As I am not familiar with the current USEPA policy on using EC20 vs. EC10 values, nor the use of the 5th percentile values as protective, I cannot make comment on these points. Obviously this is a very important point since it would appear to reduce the proposed site-specific Se criterion for the mine.

A few comments on Appendix F of the report:

Appendix F, page 9 top: “As these non-selenium stressors for wild fish can also affect the test endpoints...”. This may be true, but without providing references to support this important claim it is meaningless. In the next paragraph “larval fish survival, growth, and deformities when no selenium exposure has occurred”. There is always Se exposure, as it is an essential ultra trace element. No Se exposure would have dire consequences. In the case of Se, it is the dose that defines the poison in both directions from “normal”!

Throughout the report, e.g. Figure 10 of Appendix F: “Egg mortality” should be changed to “Embryo mortality”. Eggs are gametes, they cannot die. Once the sperm fertilizes the egg, it is an embryo.

Appendix F, Figure 25: it was unclear to me how egg Se and whole body Se were determined in the same fish to produce this relationship. I searched throughout the report and could find no methodological explanation. I assume that the mass of eggs removed for Se analysis was accounted for when measuring total Se in the remaining carcass?

6.0 Unaddressed Issues

Paragraph 1: I agree that there may be risks to aquatic bird species feeding in this area based on the comments of the reviewer and on the invertebrate Se concentrations shown in Table 13,

Appendix A. Whether or not this has bearing on establishing a site-specific chronic Se criterion for the protection of fish I cannot say, as I am a biologist, not a regulator.

Paragraph 2-3: I somewhat disagree with these comments. Even if a comprehensive dataset of egg:whole body Se ratios were determined for the ten fish species inhabiting this area, we would still not know the relative sensitivities of those species to maternal Se exposure. I may be missing something here, but to me resources would be better spent conducting similar larval toxicity experiments in other fish species representing different taxa, e.g. cyprinids.

7.0 Recommended Recalculations

(I) Paragraph 1: I agree with the reviewer's reassessment of the primary data and applaud her/him for the fine attention to detail with respect to "mining" out these discrepancies in the calculations.

Paragraph 2: I strongly agree with this assessment, and as I stated above, consider the results obtained from these five batches of embryos to be biologically meaningful and essential to be included properly in the data analysis.

Paragraph 3: I agree that the results from these 10 samples should be excluded from the dose-response analysis.

Paragraph 5 (top of page 12): From the outset I must state that I am not familiar with TRAP and its use in dose-response modeling, including the USEPA recommendations regarding its use. Thus I will assume the reviewer's comments re: recommended curve fitting models for continuous vs. quantal data are true. Nonetheless, after viewing Figure 1 (and before reading the next paragraph on page 13) I observed that there are no data between 100% and 0% survival, making this relationship tenuous at best. After subsequently reading the first paragraph on page 13, I agree with the reviewer's assessment that a site-specific criterion based on this endpoint appears to fall "somewhere" between 14 and 27 $\mu\text{g/g dw}$, and that assigning a proposed criterion at a value greater than 14 $\mu\text{g/g}$ would not be scientifically sound based on this endpoint.

(II) Overall, I agree with the assessment made by the reviewer for this first point. Table 2 in the document provides good evidence that, at least for larvae exposed to higher Se levels, not including deformed dead larvae will reduce the actual percentages of deformed larvae. However I agree with the report when they state that many of these larvae that died prior to swim-up would be difficult to assess. From my experience conducting similar research, this would be due to rapid fungal growth, decomposition and that dead larvae were likely also being fed on by live larvae. The authors of the report state that they sent approximately 100 "extra" larvae (from each of the high exposure fish LSV-003, 004, 005, 010 and 021) that died prior to swim-up to the contract lab that performed the deformity assessments. I agree with the reviewer that (a) this potential bias would not likely be as great for the fish with lower Se exposures, (b) that the bias may not greatly influence EC10/EC20 values, but (c) that the bias can only be in one direction: derivation of a less conservative criterion based on this endpoint.

For the second point in this section (starting middle of page 14), as I stated earlier in my review (page 5, above), I strongly agree with the reviewer's comments. From what I can see in the report, I agree with the reviewer that each deformity category was assessed independently. In my opinion, which is in agreement with the reviewer, calculations should be based on the simple frequencies of fully normal vs. larvae exhibiting at least one deformity (i.e., not normal).

For the data analysis made using the 2nd batch of hatchery trout (Table 3 and Figure 2), I am not in agreement with using those trout due to uncertainties related to husbandry, water temperature, nutrition, and other factors (possibly genetics as well) that differ between hatchery and wild environments. A proper field control is essential as stated earlier. With respect to the curve-fitting options and resulting output (described bottom of page 15), I will state again that I am not familiar with the use of TRAP and the appropriate model to use for such analysis. I would hazard a guess that use of either Least Squares Nonlinear Logistic Regression vs. Tolerance Distribution Analysis as curve-fitting models for this dataset would not appreciably change the resulting estimates of EC10 (6.5 µg/g egg dw) and EC20 (9.0 µg/g egg dw) values (i.e. both models would likely produce ECx values within 95% confidence intervals of each other).

The subsequent analysis (Figure 3), which excluded the hatchery trout but included the data from upstream sites (consisting of trout with elevated Se exposure), produced EC10 and EC20 values of 15.2 and 17.5 µg/g egg dw, respectively. In the report (which also excluded hatchery trout and included upstream trout), EC10 and EC20 values were calculated as 17.7 and 21.6 µg/g egg dw, respectively. Thus, estimated EC10 values from the report and FWS review vary by less than three-fold, ranging from 6.5 to 17.7 µg/g egg dw. **Whether the EC10 of 6.5 µg/g egg dw is overly conservative or whether the EC10 of 17.7 µg/g egg dw is not protective for the sustainability of fish populations in this aquatic ecosystem cannot be ascertained until the dataset is expanded to include deformity frequencies from trout inhabiting a proper, regionally appropriate reference site (or sites).** If this is undertaken, it is recommended that additional trout are collected from near field sites with "moderate" to "high" Se exposures to generate additional data for the "lower end" of the dose-response relationship for deformity frequency (i.e., between 10 to 40% normal fry).

Worth mentioning here is that a recently published article (DeForest et al. 2011) compiled available data for ten coldwater fish species that inhabit Canadian freshwater systems with the goal of creating a SSD for egg/ovary selenium. As suggested by the FWS reviewer in the document, this paper used mainly EC10 values for larval deformities and/or mortality, and the SSD was defined as the 5th percentile. The proposed guideline (Canadian regulatory term for criterion) using this approach was 20 µg/g egg dw. It should be noted that the EC10 values for brown trout and Yellowstone cutthroat trout that were derived in the present report were included as two of the ten species in DeForest et al. (2011). It also appears that the EC10 value for YCT in the report (35 µg/g egg) differs from the EC10 value shown in DeForest et al. (2011) (25 µg/g egg).

(III) Determining species-specific relationships between egg/ovary and whole body Se concentrations are important, since previous studies (e.g., Holm et al. 2005) and the regionally relevant and comprehensive report included in my review package (Osmundson and Skorupa 2011) document that there are significant differences among fish species in Se partitioning. This is not surprising given the diversity in life histories of female fish with respect to the pattern of oogenesis (e.g., synchronous vs. asynchronous [batch] spawners), length of the vitellogenic phase of oogenesis, amount of yolk in eggs, and even the large differences in amino acid sequence of vitellogenin and its catabolic products among fish species (specifically the number of methionine residues normally present in vitellogenin, which will be substituted for selenomethionine randomly and dose-dependently with increasing Se body burden). There is therefore much uncertainty involved in using generic equations (e.g., those provided by the USEPA in their 2004 draft Se criterion document) to predict whole body Se from egg/ovary Se in a given species.

In this section, when comparing Figure 4 of the FWS review to Figure 5-33 of the report it appears that the x and y axes were switched so that whole body Se is the dependent variable, and that only the brown trout Se relationship is shown. The R^2 value for the brown trout regression changed from 0.81 in the report to 0.79 in the FWS review. I don't have an opinion as to which way is correct, except to say that if whole body Se is to be predicted from egg Se, then the FWS review regression is more appropriate.

In the review the p value for the regression is given as " $p < 0.0000001$ ", which seems unusual. Once p is less than 0.001 it indicates a highly significant result, and I'm not sure how much more confident one can be that the slope of this line is not horizontal with these extra zeroes included.

Rearrangement of the regression axes resulted in a slightly lower predicted critical whole body Se concentration compared to the report (12.32 vs. 13.35 $\mu\text{g/g}$ egg dw), based on the EC20 value of 21.6 $\mu\text{g/g}$ egg dw calculated in the report. Based on the EC10 value determined from Figure 2 in the FWS review (6.5 $\mu\text{g/g}$ egg dw), the estimated critical whole body Se concentration was 5.5 $\mu\text{g/g}$ egg dw. In my opinion, this whole body value appears overly conservative based on the literature and weight of evidence (see my comments in Summary below). If this is to be included in the FWS review, it is suggested that back-calculation of the whole body values from the EC10 derived from Figure 3 of the FWS document is also included in this section, to provide some balance.

(IV) The first sentence of this section ("Tissue partitioning ratios...") requires literature reference(s), since it is central to the argument presented. What evidence is there that Se partitioning to specific tissues such as the ovary changes with increasing exposure? I'm not disagreeing with this statement, but feel that it should be substantiated with evidence from the literature.

In this section the FWS reviewer makes a strong case that the use of brown trout Se partitioning among tissues to predict egg/ovary to whole body Se ratios for the other fish species making up

the Crow Creek fish community may not be valid. Obviously there might even be more exceedances if a more conservative whole body criterion was used. It appears that sampling of the other fish species in this system, with collections occurring during mid to late vitellogenesis, to derive species-specific egg/ovary to whole body Se ratios would be appropriate from a regulatory standpoint.

Final Comments

Overall, I feel that the FWS reviewer provided accurate and fair comments based on sound scientific principles and statistical methodologies.

Although certain of the recommended recalculations produced only slight changes to derived values, which may seem semantic, they are based on sound science and should be considered by the authors of the report.

It is of utmost importance for an appropriate reference site(s), under no influence of anthropogenic activities that alter regionally normal aquatic selenium levels, be employed to obtain proper background frequencies of larval deformities in brown trout. I've done similar studies and had to go do an extra field season. It's probably worth it.

The differences in opinion between the FWS reviewer and authors of the report represent common industry vs. government differences in conservatism with respect to conservation issues. What is appropriate criterion? In my opinion it is likely somewhere between the 6.5 µg Se/g egg dw EC10 value proposed by the FWS reviewer and the 17.7 µg Se/g egg dw EC10 value proposed in the report. Looking on the bright side, we are only talking a less than three-fold difference here; comparing this discrepancy to differences in protective criteria for the vast majority of inorganic and organic environmental contaminants in both ecological and human health risk assessments, it seems to me that a solution can be reached fairly easily with some additional resources devoted to this dataset. My goal in providing this review was hopefully to provide some balance to the argument from both perspectives (as a conservationist and a researcher who works in collaboration with industry).

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Mr Hoffman,

I have completed my review and analysis of “*Selected Technical Review Comments*” by the US Fish and Wildlife Service (hereafter referred to as the **FWS Commentary**) relevant to a report entitled “*Interpretive Findings for Field and Laboratory Studies and Literature Review in Support of a Site-Specific Selenium Criterion, Smoky Canyon Mine*” (August 2010) (hereafter referred to as the **Interpretive Report**) prepared by Formation Environmental and HabiTech for the J.R. Simplot Company in Pocatello, Idaho. In addition to reviewing the material contained within those documents, Appendices A,C and E in the *Interpretive Findings Report*, my review also included material presented in a report from the FY11 Environmental Contaminants Program Off-Refuge Investigations Sub-Activity (Project FFS ID: 6F50) entitled “*CO-Selenium in Fish Tissue: Prediction Equations for Conversion between Whole Body, Muscle, and Eggs*” (Osmundson and Skorupa 2011). I also reviewed several citations from the primary scientific literature to verify the accuracy of statements made in the FWS Commentary and the Interpretive Report.

In my capacity as a research scientist with the Department of Fisheries and Oceans, Canada I have been examining the potential impacts of selenium on freshwater fish for more than 12 years. I have a working knowledge of the statistics of dose-response modeling, and biology of salmonid fishes that are relevant to this area of study. While I bring this experience and working knowledge of the scientific literature, I must be clear that this review was performed outside of my responsibilities and working hours and, therefore, reflects my personal opinions and not necessarily those of the Department of Fisheries and Oceans.

I have focused my review to address the following statement of work outlined in your message of October 4, 2011:

“The reviewers will consider, but not be limited to, whether the authors’ conclusions presented in the FWS technical comment document accurately and fairly reflect the content of the reviewed documents and are scientifically valid and fair criticisms of the reviewed documents. If any of the criticisms presented in the FWS technical comment document appear to be based on misunderstanding or unfair rendering of the reviewed documents or are so unclear as to be undecipherable they should be identified and included in the reviewers comments on the FWS technical comment document. In

addition the reviewers should consider: Are the specific critiques presented in the FWS technical comment document coherently presented, logically sound, technically valid, and nontrivial? In addition, the reviewers should consider: How could the FWS technical comment document be improved?

The points of contention provided in the FWS Commentary are discussed in order of their appearance in that document.

i) FWS Commentary

Section 1.0 Introduction – The FWS Commentary notes that the Yellowstone Cutthroat Trout (YCT) studies are portrayed as preliminary in the Introduction of the Interpretive Report and therefore, that interpretation is premature. However, it appears that analysis of the data from the YCT Adult Reproduction in the Interpretive Report are more than preliminary. Specifically, relationships between Se in YCT eggs and whole body Se, survival, growth and deformities appear to have been evaluated sufficiently to provide model analysis and Effects Concentration levels. I am unclear what analysis remain pending, such that the Introduction needs to portray the results as “preliminary”.

I concur with the FWS Commentary that the Interpretive Report has misinterpreted the USEPA’s process for establishing a national water criterion (page 5, Interpretive Report). While only an aqueous exposure metric is nationally prescribed, this value was established with consideration that dietary exposure also occurred at the modeled Belews Lake field setting. Furthermore, the FWS Commentary correctly notes that USEPA’s proposed whole body tissue criterion (<http://water.epa.gov/scitech/swguidance/standards/criteria/aqlife/pollutants/selenium/upload/complete.pdf>) was established primarily using data from a laboratory study in which bluegill were exposed to dietary selenomethionine (Lemly, A.D. 1993. Metabolic stress during winter increases the toxicity of selenium to fish. *Aquat. Toxicol.*, 27:133-158.) and not from a sole analysis of Belews Lake fish data.

As the FWS Commentary contends, the 7.91 mg/kg dw whole body draft criterion established by USEPA was based on responses in bluegill exposed to temperature stress in addition to Se exposure. Therefore, comparison of genus mean chronic values (GMCV) from studies in which GMCV can be derived for trout, but for which no temperature stress is also included, is inappropriate. Specifically, the sensitivity of trout cannot be said to be less than bluegill based on the existing data in USEPA’s 2004 Draft Criterion.

The Interpretive Report identifies that the USEPA commissioned the study by McIntyre et al. (2008) to re-examine Lemly’s winter stress syndrome paper of 1993, but as noted in the FWS Commentary, there are important differences in the two studies that preclude their direct comparison. Among these are potentially different strains of fish (wild caught fish were used in Lemly (1993) while McIntyre used fish from a commercial source (Osage Catfisheries, Osage

Beach, MO), differences in acclimation periods and temperature reduction, and different light regimes. The latter is noted in the FWS Commentary and is especially important, because light cues are important for reproductive timing and energy metabolism shifts. The FWS Commentary also notes that lipid depletion determined in Lemly (1993) was not reported by McIntyre et al. (2008). Whether or not this was due entirely to different light regimes is uncertain, but direct comparisons of effects concentrations from the two studies certainly must consider these differences.

Section 3.0 Literature Review

Notwithstanding the dismissal of Besser et al. (2008) noted by in the FWS Commentary, other recent studies in which Se toxicity thresholds were developed were also omitted from the Interpretive Report. These omissions are important, because while the FWS Commentary contends that the “derivation of a proposed site-specific selenium criterion is not based substantively on the literature review”, the site specific thresholds that are developed in the Interpretive Report are considered in the context of literature values later in the document (eg. Section 6.2, Pages 68-69).

Further to the reliance on Kennedy et al. (2000) for providing a toxicity threshold, it should be noted that while those authors reported a NOEC of > 81 mg SE/kg dw in eggs, two later studies ***using the same species*** reported effects thresholds of 16-20 mg SE/kg dw (Rudoph et al. 2008; Nautilus 2009). These values are more in line with the rest of the salmonid literature and do not suffer from some of the methodological issues we identified with Kennedy et al. in Palace and Hamilton (2001). I am in agreement with the statement in the Interpretive report, that egg survival is a highly variable endpoint and not particularly useful as a study endpoint for Se effects studies.

4.0 Field Monitoring

It is my understanding that there is a well established negative effect of sulphate on selenium uptake by aquatic macrophytes and lower trophic organisms and that competition for transport sites on the permease enzyme system is the accepted mechanism for this effect. However, a brief review of the literature indicates that, indeed, as stated in the FWS Commentary, there are few field validations of these laboratory results that would allow adjustment of a waterborne Se criterion based on elevated sulphate levels at field locations.

The contention in the FWS Commentary that South Fork Tincup external reference site fish are significantly different from both upstream Crow Creek and Deer Creek reference sites is accurate. Specifically, Table 5.2 of the Interpretive Report shows that whole body Se in cutthroat trout from South Fork Tincup was 2.56 mg/kg dw, while concentrations in the upstream reference sites range from ca. 8 to 25 mg/kg dw. References to support comparable Se concentrations in fish from other reference sites are provided in the FWS Commentary. In brown

trout, reference fish from the Saratoga Hatchery contained whole body Se concentrations ranging from ca. 2.5 to 4.5 mg/kg dw, while the Crow Creek fish contained from 4.7 to 9.2 mg/kg dw (Table 5-2. Interpretive Report). What these comparisons of reference fish point out, is that fish from the “in field” reference sites (Upstream Crow Creek and Deer Creek) have higher than expected concentrations of Se. As contended in the FWS Commentary, this is not necessarily a surprising result. Fish in open systems are free to move and we have also shown evidence of salmonid fish movement into, and out of, Se impacted streams from reference locations (Palace et al. 2007, Environ. Sci. Technol. 41: 3679-3683). That periphyton more closely reflect Se exposure conditions at the chosen reference sites (Upstream Crow Creek and Deer Creeks) than reference fish captured from these locations, emphasizes the concept that fish movement is the likely source of higher than expected Se in fish at the reference sites locations.

As the FWS Commentary contends, the use of fish densities in potentially Se-affected streams is not an appropriate assessment endpoint for the effects of Se (summary statement on Page 40 of the Interpretive Report). One reason for this is the potential for fish movement. In other words, streams with elevated Se can still contain fish assemblages even though effects on recruitment are likely to be present. Extending on the example from our research in Alberta that was cited above (Palace et al. 2007), spawning rainbow trout were still identified in Se-impacted streams, even though significant reproductive effects were identified (Holm et al. 2005, Environ. Toxicol. Chem. 24:2373-2381). The interpretation is that while recruitment may be affected, adult fish that are not adversely affected by elevated Se, continue to use the affected habitat for foraging and spawning (citation is, again, Palace et al. 2007).

The FWS Commentary contends that demographic parameters such as those described in Vasterling (2003) would provide more appropriate metrics to identify potential effects of Se in affected streams. While I agree in principle, even these measures may provide responses that respond too slowly to provide meaningful “alarm bells” before population level impacts are evident. Continuing with our Alberta example above, population monitoring based on fish densities and size classes took more than 2 decades to show declining rainbow trout populations in the Se-affected Luscar Creek (G. Sterling, Alberta Sustainable Development, person. Commun. and VP Palace, GS Sterling, P Siwik, RE Evans, N Halden, KG Wautier, J Holm. 2006. Limitations of non-lethal sampling for determining spatiotemporal exposure to selenium in fish from mine impacted sites. 27th Annual Society of Toxicology and Chemistry Meeting, Montreal, QC. Nov. 5-9). The timeframe for response variables in this type of demographic assessment is relevant when one considers the time to reproductive maturity (generally 3-5 years) and long duration of active reproduction among cutthroat and brown trout (13+ years, Avery et al. (1985) [Eds] *Sexual maturity and fecundity of brown trout in central and northern Wisconsin streams*. Tech. bull.. Wisconsin Dept. of Natural Resources, No. 154, 12pp.). In simple terms, failures of several years

classes would have to have occurred before demographic sampling would yield significant results because adult populations have extended reproductive lifespans. This is exacerbated in open systems where recruitment from adjacent unaffected systems can occur. Therefore, the most sensitive measures of potential Se impacts remain reproductive bioassays of relevant species under controlled conditions (Janz et al. 2010, Selenium Toxicity to Aquatic Organisms, Chapter 6 IN, Ecological Assessment of Selenium in the Aquatic Environment. Chapman et al. [Eds]. CRC Press, Boca Raton, FL.).

5.0 Laboratory Studies

As noted in the FWS Commentary, the adjustment of hatchery control responses to background (Upstream Crow and Deer Creeks) is inappropriate given the apparently elevated Se concentrations in these reference fish. Establishing a valid regression relationship is dependent on the inclusion of fish with a relevant range of Se concentrations. Specifically, it is fish at the lower end of the exposures that are most important for accurately deriving no effects concentrations (NOEC). Again, the FWS Commentary correctly points out that without these data points at the lower end of the exposures, overestimates of effects levels are inevitable. In my opinion, it is preferable to include the hatchery data uncorrected to strengthen the predictive capability of the regressions.

The use of survival among 15 day post-swim up fry is not expected to be a sensitive endpoint for Se effects in a laboratory setting where rearing conditions are optimized. In contrast, the sum total of normal fry is appropriate as long as it does not include fry that did not swim up as part of the group characterized as "normal". As noted in the FWS Commentary, failure to swim up at all is tantamount to death among wild fry. The fact that failure to swim-up occurred in a high proportion of eggs that contained high concentrations of Se (>25mg/kg dw, Figure 13 of the Interpretive Report) is an important result, albeit limited as a diagnostic indicator. Furthermore, 15 day feeding trials using the fry with the 5 highest concentrations of Se, and that failed to swim-up, is environmentally unrealistic and of no ecological value as noted in the FWS Commentary.

As noted in my comment from the paragraph above, the sum total of normal fry is an appropriate diagnostic indicator for examining potential Se reproductive effects in fish. However, this is true only if records are maintained that allow the analyst to link deformity analyses for each fry across all 4 of the assessed categories and not simply a sum of the proportions of normal fry for each deformity category separately. The analysis description in the Interpretive Report, however, stipulates that:

"deformities were evaluated as the sum fraction of normal fish (sum of normal fish/ total number of fish) for each deformity" (Appendix F, Page 11).

This indicates a de-coupling of the deformity analysis for each individual fry. As illustrated in the FWS Commentary, this practice allows for inflation of the total

number of normal fry where multiple deformities do not exist. I view this point as being of utmost in importance for the derivation of reliable effects concentrations from the studies described in the Interpretive Report. In fact, as discussed below, I feel strongly enough about this shortcoming that I recommend a re-analysis of deformities from fry in these studies.

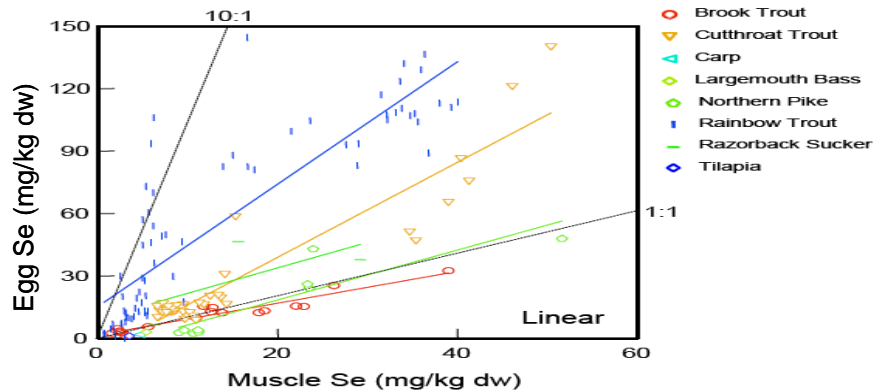
I am fairly certain that re-plotting the regression of whole body Se and egg Se, with the X and Y axis reversed, would not significantly affect the derived whole body translations.

The FWS Commentary contends that the sensitivity of brown trout is incorrectly illustrated in Figure 6-1 of the Interpretive Report because an EC20 is shown and not the preferred EC10 based on USEPA protocol. While this may be an important consideration, in my opinion it is more important to note that the data presented in the Interpretive Report are not comparable to the other maternal transfer studies. Again, this arises from the flawed deformity analysis that summed proportions of deformities independently.

6.0 Unaddressed Issues

I have limited expertise in the area of aquatic waterfowl biology and the potential for Se reproductive effects and, therefore, cannot comment on the first issue raised in the FWS Commentary.

The Interpretive Report advocates the use of brown trout as an ecologically relevant species to monitor for potential Se effects. Summary statements in the Interpretive Report illustrate that it is the intent of the authors to use Se in whole body of brown trout to calculate potential Se concentrations in eggs of other species as suggested by the FWS Commentary. And so, it is important to address comments noted on page 9 of the FWS Commentary, regarding variability in the ratios of Se concentrations in whole body (or muscle) and those found in eggs among different fish species that have been investigated thus far. This is important when the relationships from one species are assumed to reflect the partitioning kinetics between tissues of other species within the same habitats and when that species is used as a sentinel species to determine protective effects levels.



Consider data in the figure above showing relationships between concentrations of Se in eggs and those in muscle for 8 freshwater species. (Source: *Selenium Tissue Thresholds 2008*. GEI Consultants, Golder Assoc., Parametrix, U of Sask.).

The figure shows the regression plots for 8 species as well as 1:1 and 10:1 regression lines. The Interpretive Report stipulates that for brown trout from this study, the ratio for egg to muscle Se is 1.54, meaning this species is near the bottom end of known regressions in terms of its slope. The FWS Commentary notes that this is also true when brown trout are considered among the 12 documented species of fish in the Colorado River Basin for which these regressions have been documented. What this means is that, relative to most other species, brown trout accumulate higher concentrations of Se in muscle or whole body while maintaining lower Se concentrations in their eggs. Thus, if brown trout are used as the lone sentinel species for an aquatic system, an underestimate of potential reproductive effects from Se would result for some other fish species.

7.0 Recommended Calculations

(I.) Hatch to 15-days post swim-up fry survival endpoint:

I am uncertain why the endpoint “Hatch to 15 days post swim up fry survival” was calculated in the FWS Commentary by dividing %Total Survival by % Hatch. I have re-calculated the measure as follows, which seems to more closely agree with the data presented in the Interpretive Report:

$$\text{Hatch to 15 days post swim up survival} = 100 - (\% \text{Hatch} - \% \text{total survival})$$

Using this equation to calculate the measure, there are still several miscalculations contained in Table 3-4 of Appendix A of the Interpretive Report. The following Table of revised values shows that 12 measures appear to be incorrect. These are highlighted in yellow and, in addition, there is one value (CC-150-016) for which total survival appears to be higher than the initial percent hatch (highlighted in red). While this is not possible (i.e. fish that did not hatch cannot survive to 15 days post hatch) the Interpretive Report provides a footnote explaining that “missing test organisms at test termination were not included in

calculation” for the Hatch to 15 days post swim up survival. If this is the explanation for the total survival number being elevated above the initial hatch, this needs to be corrected before any regression analysis can be performed.

TABLE OF RECALCULATED HATCH TO 15 DAY SURVIVAL VALUES

Sample ID	% Hatch	%Total Survival	Hatch to 15 days survival
SC-001	23.8	22.7	98.9
SC-002	22.8	22.3	99.5
SC-003	56.7	54.7	98
SC-004	31.1	27.3	96.2
SC-005	11.7	10.7	99
SC-006	93.2	92.7	99.5
SC-007	30.5	26.3	95.8
SC-008	66	65	99
SPC-001	99.3	98	98.7
SPC-002	100	100	100
SPC-003	97.5	94.2	96.7
SPC-004	100	100	100
SPC-005	98.8	96.8	98
SPC-006	99.7	96	96.3
CC-150-009	28.5	27	98.5
CC-150-011	96	95.3	99.3
CC-150-012	88.8	86	97.2
CC-150-013	66.7	57.5	90.8
CC-150-015	78.3	77.5	99.2
CC-150-016	14.8	15.4	100.6
CC-150-017	89.2	84.3	95.1
CC-150-018	87.2	84.3	97.1
CC-150-020	97.3	96.3	99
CC-350-006	71.7	67.7	96
CC-350-007	29.7	26	96.3
CC-350-008	67.7	62.6	94.9
LSV2C-002	99	96.6	97.6
LSV2C-003	93.5	2.25	8.75
LSV2C-004	50.6	16.8	66.2
LSV2C-005	71.3	23	51.7
LSV2C-006	0	-	-
LSV2C-007	0	-	-
LSV2C-008	88.3	84.2	95.9
LSV2C-010	87	11	24
LSV2C-012	98.3	95.7	97.4
LSV2C-016	95	91.7	96.7
LSV2C-017	71.3	60.5	89.2
LSV2C-019	94.2	88.9	94.7
LSV2C-020	89.2	86.8	97.6
LSV2C-021	69.3	14.8	45.5

The FWS Commentary is absolutely correct in noting that the 5 samples that did not produce swim-up fry at the end of the experimental period cannot be portrayed as anything other than 0% for legitimate regressions to be performed. Fry that fail to swim-up would be mortalities in the wild and portraying them in the same manner as fry which did swim-up from other clutches is completely inappropriate.

The FWS notes several methodological errors and exclusions of data from missing organisms in the last paragraph on page 10. With regard to the hatchery control datasets, in my opinion, the exclusion of the first set of hatchery fish (SC) based on poor % hatch is appropriate. As noted in the Interpretive Report the

poor performance of these eggs/fry may have been a result of different fertilization techniques or due to fungal infections. The second set of hatchery fish (SPC) is also problematic in terms of making comparisons to wild caught eggs because they were received as a later stage of development (eyed eggs) and not as fertilized eggs. Therefore, as the FWS Commentary points out, the percent hatch and potentially other parameters are not strictly comparable with other clutches of eggs treated differently. Finally, the FWS Commentary raises the issue of missing organisms from 10 groups of eggs (12 groups are actually noted as having missing organisms, but 2 of these are from the SC group which were excluded from further modelling). It is an unsubstantiated assumption that lost fry would have the same proportion of deformities and/or survival. Specifically, the Interpretive Report portrays these animals as having been lost "due to food clogging the drain pipe, resulting in an overflow of the test chamber." In my opinion, it is more likely that dead or weakly swimming organisms would have been lost from the overflow at the top of the incubation container, because at this stage of development, fry are negatively phototropic and would seek refuge near the bottom. Therefore, as suggested by the FWS Commentary, it is more appropriate to exclude these tests from the developed models. This is also more consistent with the methodologies employed by the authors of the Interpretive Report in earlier stages of their studies. Specifically, they describe on page 3-6 of Appendix A that "*Any organisms that were not found during the test were considered dead*". This refers to the period of the test from fertilization to hatch, but the authors continue the statement to provide an alternative treatment of lost organisms in the second part of the studies (after swim-up) as: "...*except during the 15-d swim-up study. For this phase of the study, any missing organisms were considered missing and were excluded from survival analysis.*"

With regard to the model chosen (Tolerance Distribution Analysis) that the FWS Commentary suggests for analyzing exposure-responses, I cannot provide input as I am unfamiliar with the USEPA's TRAP software program and its conditions of operation and statistical assumptions, and requirements. I can, however, concur that the data generated for response variables from the brown trout study were not continuous, nor would they be expected to have infinite tails approaching 0 or 100%. In this sense, the description of the type of data generated by the studies is as described by the FWS Commentary on page 12, paragraph 1 (i.e. non-continuous with a finite threshold).

(II.) Larval Deformity Endpoint:

With regard to the collection of larval deformities, I concur with the FWS Commentary that exclusion of alevins and post swim-up fry that died before termination of the study is inappropriate and likely influenced deformity analysis by excluding potentially higher rates of deformed fry (dead fry are more likely to have been deformed) from further analysis. However, it should also be noted that many of the studies in the published literature have also excluded pre-swim up fry from analysis. In most cases, this exclusion is likely to have originated in an

interest to maintain comparable developmental stages from each batch of eggs for assessment and comparison purposes.

I have already discussed my views of the independent reporting of the 4 categories of deformities for groups of fry rather than a summation of deformities for each individual fry (see the 3rd paragraph on Page 5 of this Report Under the heading 5.0 Laboratory Studies). It is worth noting that a recent publication (McDonald and Chapman 2009. *Integ. Environ. Asses. Mngmt.* 5:470–475) prescribes QA/QC procedures in which

“A nonsequential labeling system is also recommended but will vary depending on the number of larval fish included in the deformity assessment. Individual vials are recommended for studies with small numbers of larval fish per adult female”.

This type of re-analysis is, fortunately, possible where preserved specimens are available, as is the case for the studies described in the Interpretive Report. ***I would strongly favour such a re-analysis rather than the expression of “maximum fully normal fry” proposed by the authors of the FWS Commentary*** (page 14, 2nd paragraph). Should the authors of the Interpretive Report pursue such a re-analysis of the preserved specimens, they should consult the aforementioned publication for additional QA/QC measures (eg. Non-sequential labelling of fry, external deformity analysis checks) to ensure the most defensible data can be collected.

Again, without familiarity regarding the operational requirements of the TRAP software, I am unable to comment on the model derivation offered by the authors of the FWS Commentary at the bottom of Page 15 and continuing on Page 16. However, as noted above, I am confident that the lack of a measure truly accounting for “fully normal fry” could certainly bias the EC10 and EC20 estimates generated by the model toward the conservative. This is why, in my opinion, it is so critical to undertake a re-analysis of the preserved fry. Certain aspects of the studies described in the Interpretive Report cannot be re-examined (eg. Lost embryos during the 15 day swimup phase), but a reanalysis of the deformities is possible and should be the highest priority for this project. The issue of appropriate field control data has already been addressed on Page 3 of this report, last paragraph).

(III.) *Regressing X on Y for Estimation of Critical Whole Body Tissue Concentrations*

As discussed earlier in my report there are significant species differences in distribution of Se between whole body or muscle and eggs. A review of Osmundson and Skorupa (2011) shows that deposition of Se into eggs from muscle or whole body can be expected to differ between wild fish and those raised in the laboratory. Similar data were also already discussed above in this report. While reasons for this difference are not fully known, it has been suggested

that differential binding strengths of Se to the egg yolk precursor, vitellogenin, may account for some of the variability (Janz et al. 2010, Selenium Toxicity to Aquatic Organisms, Chapter 6 IN, Ecological Assessment of Selenium in the Aquatic Environment. Chapman et al. [Eds]. CRC Press, Boca Raton, FL.). Secondly, there are marked differences in vitellogenin contents among different fish species' eggs (Osmundson and Skorupa 2011). The regression plot and derivation of whole body Se from Se in eggs of brown trout that are given in the FWS Commentary (last paragraph of page 18 and first paragraph of page 19) are accurate. However, I contend, again, that a re-analysis of deformities in preserved fry and evaluation of the data from those analyses are far more preferable to expression of an EC10 based on the "fully normal fry" measure proposed in the FWS Commentary. Any derivation using these criteria are destined to be conservative in deriving an effect level, for reasons already discussed.

(IV.) Test of Assumption that the 14 ppm Proposed Critical Whole Body Tissue Concentration Derived from Brown Trout Tissue Partitioning Data Would Generally Keep Egg Selenium Values Below a Critical Value of 21.63 ppm Across a Multi-species Community of Fish:

Osmundson and Skorupa (2011) provide a data set illustrating significant differences among fish species in how they partition Se to their eggs/ovaries from muscle or whole body. The same report also shows differential partitioning to eggs/ovaries dependent on relative loads of Se among two species of fish (white sucker and green sunfish). For example, white sucker accumulate relatively low Se concentrations relative to those achieved by black bullhead. Moreover, even within white sucker, relatively less Se is deposited to their eggs from an increasing exposure dose during pre-spawning periods, relative to post spawning periods. These species and seasonal specific slopes for relating concentrations of Se in eggs to expected values in muscle or whole body become extremely important when whole body or muscle concentrations of Se are intended to provide monitoring data that will identify potential reproductive issues arising from Se exposure in the future. As identified in the FWS Commentary, the choice of brown trout as a (SSSC) monitoring species to derive a Site Specific criterion threshold value has great implications for risk management decisions for other species. Without a detailed accounting of complete fish assemblages in the study area described in the Interpretive Report, a judgement on the protectiveness of the brown trout SSSC is highly questionable. This is especially true given the lack of precision in deriving the brown trout EC10.

Thank you for the opportunity to comment on both the Interpretive Report and the FWS Commentary. I am keenly interested in selenium and its potential to act as a reproductive toxicant and I would be pleased to offer additional opinions regarding re-analysis of deformities, should the stakeholders in this study deem that course of action to be appropriate. Should you have any questions regarding

any of the opinions offered within this report, please do not hesitate to contact me.

Respectfully submitted,

Vince P. Palace, Ph.D.

Curriculum Vitae for Dr. Mark Rigby

EDUCATION/EXPERIENCE

- 6/1/2008- Senior Risk Assessor, Parsons, South Jordan, USA.
2001- Assistant Researcher at the University of California, Santa Barbara, CA, USA.
6/2000-5/2008. Staff Scientist for Tetra Tech, Inc., Lafayette, CA, USA.
2000. Lecturer (Parasitology) at the University of California, Santa Barbara, CA, USA.
1999. Ph.D. Swiss Federal Institute of Technology, Zürich, Switzerland.
1996. M.Sc. University of Alberta, Edmonton, Canada.
1993. B.A. (distinction). University of California, Santa Barbara, USA.

PUBLICATIONS IN REFEREED JOURNALS - ENVIRONMENTAL

1. Rigby MC, Deng X, Grieb TM, Teh SJ, Hung SSO. 2009. Effect threshold for selenium toxicity in juvenile Splittail, *Pogonichthys macrolepidotus* A. Bulletin of Environmental Contamination and Toxicology **84**:76–79.
2. Hartman B, Schumacher BA, Zimmerman JH, Springer DS, Elliot RJ, and **Rigby MC**. 2007. Results from EPA Funded Research Programs on the Importance of Purge Volume, Sample Volume, Sample Flow Rate and Temporal Variations on Soil Gas Concentrations. Proceedings of the AWMA Conference Vapor Intrusion: Learning from the Challenges (Sep 26-28, 2007; Providence, RI)
3. Mills WB, Liu S, **Rigby MC**, Brenner D. 2007. Time-variable simulation of soil vapor intrusion into a building with a combined crawl space and basement. Environmental Science and Technology 41:4993-5001.

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1. Costa G, Freitas N, Pinheiro de Carvalho MAA, Melo-Moreira E, Rigby MC. 2009. *Oncophora melanocephala* (Nematoda, Camallanidae) from the chub mackerel, *Scomber japonicus* (Teleostei, Scombridae), caught off Madeira Island (Portugal). Acta Parasitologica 54:253-256.
2. Vignon M, Sasal P, Rigby MC, Galzin R. 2009. Multiple parasite introduction and host management plan: case study of lutjanid fish in the Hawaiian Archipelago. Diseases of Aquatic Organisms 85:133–145.
3. **Rigby MC**, Sharma RSK, Hechinger RF, Platt TR, Weaver JC. 2008. Two new species of *Camallanus* (Nematoda: Camallanidae) from freshwater turtles in Queensland, Australia. Journal of Parasitology 94:1364-1370.
4. Moravec F, Justine J-L, Rigby MC. 2006. Some camallanid nematodes from marine perciform fishes off New Caledonia. Folia Parasitologica **53**:223–239.
5. Sharma R.S.K., **Rigby M.C.**, Sumita S., Sani R.A., Vidyadaran M.K., and Jasni S. 2002. Redescription of *Serpinema octorugatus* (Nematoda: Camallanidae) from the Malayan box turtle *Cuora amboinensis* (Chelonia: Bataguridae). Systematic Parasitology **53**: 19-28.
6. **Rigby M C**, Hechinger, RF, Stevens L. 2002. Why should parasite resistance be costly? Trends in Parasitology **18**:116-120.
7. **Rigby, M. C.**, and W. F. Font. 2001. Statistical re-analysis of the distinction between *Spirocamallanus istiblenni* and *S. monotaxis* (Nematoda: Camallanidae). Journal of Parasitology **87**: 1210-1213.

8. Cribb TH, Pichelin S, Dufour V, Bray RA, Chauvet C, Faliex E, Galzin R, Lo CM, Lo-Yat A, Morand S, **Rigby MC**, Sasal P. 2000. Parasites of recruiting coral reef fish larvae in New Caledonia. *International Journal for Parasitology* **30**: 1445-1451.
9. Font, W. F. and **Rigby, M. C.** 2000. Implications of a new Hawaiian host record from blue-lined snappers *Lutjanus kasmira*: is the nematode *Spirocamallanus istiblenni* native or introduced? *Hawaii Biological Survey* **64**: 53-56.
10. Morand S, T. H. Cribb, M. Kulbicki, **M. C. Rigby**, C. Chauvet, V. Dufour, E. Faliex, R. Galzin, C.M. Lo, A. Lo-Yat, S. Pichelin, and P. Sasal. 2000. Endoparasite species richness of New Caledonian Butterfly fishes: host density and diet matter. *Parasitology* **121**: 65-73.
11. Jokela, J., Schmid-Hempel, P. and **Rigby, M. C.** 2000. Dr. Pangloss restrained by the Red Queen - steps towards an unified theory of defence. *Oikos* **89**: 267-275.
12. **Rigby, M. C.** and Jokela, J. 2000. Predator avoidance and immune defence: costs and trade-offs in snails. *Proceedings of the Royal Society, Series B Biological Sciences* **267**: 171-176.
13. **Mark C. Rigby**, Cédrik M. Lo, Thomas H. Cribb, Louis Euzet, Elisabeth Faliex, René Galzin, John C. Holmes, and Serge Morand. 1999. The role of parasites in coral reef fish communities – considerations and an example from French Polynesia with a parasite checklist. *Cybio* **23**: 273-284.
14. Morand, S., and **Rigby, M. C.** 1998. Cucullanin nematodes from coral reef fishes of French Polynesia, with a description of *Cucullanus faliexae* n. sp. (Nematoda: Chitwoodchabaudiidae). *Journal of Parasitology* **84**: 1213-1217.
15. **Rigby, M. C.**, Adamson, M. L., and Deardorff, T. L. 1998. *Camallanus carangis* Olsen, 1954 (Nematoda: Camallanidae) reported from French Polynesia and Hawai'i with a redescription of the species. *Journal of Parasitology* **84**: 158-162.
16. **Rigby, M. C.**, Holmes, J. C., Cribb, T. H., and Morand, S. 1997. Patterns of species diversity in the gastrointestinal helminths of a coral reef fish, *Epinephelus merra* (Serranidae), from French Polynesia and the South Pacific Ocean. *Canadian Journal of Zoology* **75**: 1818-1827.
17. **Rigby, M. C.**, Font, W. F., and Deardorff, T. L. 1997. Redescription of *Camallanus cotti* Fujita, 1927 from Hawai'i. *Journal of Parasitology* **83**: 1161-1164.
18. **Rigby, M. C.**, and W. F. Font. 1997. Redescription and range extension of *Spirocamallanus istiblenni* Noble, 1966 (Nematoda: Camallanidae) from coral reef fishes in the Pacific. *Journal of the Helminthological Society of Washington* **64**: 227-233.
19. **Rigby, M. C.**, and Adamson, M. L. 1997. *Spirocamallanus* species of French Polynesian coral reef fishes. *Canadian Journal of Zoology* **75**: 1270-1279.
20. **Rigby, M. C.**, and V. Dufour. 1996. Parasites of coral reef fish recruits, *Epinephelus merra* (Serranidae), in French Polynesia. *Journal of Parasitology* **82**: 405-408.
21. **Rigby, M. C.** 1996a. The epibionts of beach hoppers (Crustacea: Talitridae) of the North American Pacific coast. *Journal of Natural History* **30**: 1329-1336.

22. **Rigby, M. C.** 1996b. Association of a phoretic juvenile mite (Acari: Uropodidae) with beach hoppers (Crustacea: Talitridae). *Journal of Natural History* **30**: 1617-1624.
23. Adamson, M. L., and **M. C. Rigby.** 1996. *Rhabditis (Crustorhabditis) stasileonovi* (Belogurov) from beach hoppers (Amphipoda: Talitridae) from the Pacific coast of North America. *Fundamental and Applied Nematology* **19**: 579-584.

NON-REFEREED PUBLICATIONS

1. Stephen J. Cornell, Yves Desdevises, and Mark C. Rigby. 1999. Evolutionary biology of host-parasite relationships: reality meets models. *Trends in Ecology and Evolution* **14**: 423-425.

BOOK CHAPTERS

1. Rigby, M. C., and Y. Moret. 2000. Life history trade-offs with immune defenses. *In Evolutionary Biology of Host-Parasite Relationships*, edited by R. Poulin, S. Morand and A. Skorping, eds., Amsterdam: Elsevier, pp 129-142.
2. Rigby, M. C. 1999. The Camallanidae (Nematoda) of Indo-Pacific fishes: taxonomy, ecology, and host-parasite coevolution. *In Proceedings of the 5th Indo-Pacific Fish Conference (Nouméa, 3-8 November 1997)*, edited by B. Séret and J.Y. Sire. Paris: Société Française d'Ichtyologie & Institut de Recherche pour le Développement, pp 633-644.

PRESENTATIONS – VAPOR INTRUSION

1. Hartman B., Schumacher B.A., Zimmerman J.H., Springer D.S., Elliot R.J., and **Rigby M.C.** 2007. Results from EPA Funded Research Programs on the Importance of Purge Volume, Sample Volume, Sample Flow Rate and Temporal Variations on Soil Gas Concentrations. AWMA Conference Vapor Intrusion: Learning from the Challenges (Sep 26-28, 2007; Providence, RI).
2. Rigby, M.C., S. Liu, B. Mills, D. Brenner, S. Olliges, and T. H. Anderson. 2005. New models for predicting indoor vapor concentrations for buildings with crawl spaces & basements: a case study from Moffet Field. Tri-Service Ecological Risk Assessment Work Group, January 24, 2006, Port Hueneme, CA.
3. Rigby, M.C., B. Mills, S. Liu, K. Johnson, D. Brenner, S. Olliges, and T. H. Anderson. 2005. New models for predicting indoor vapor concentrations for buildings with crawl spaces & basements: a case study from Moffet Field. Society for Risk Analysis, December 5, 2005, Orlando, FL.
4. Rigby, M.C., S. Liu, B. Mills, D. Brenner, S. Olliges, and T. H. Anderson. 2005. Predicting indoor vapor concentrations for a building with a crawl space at Moffet Field. Groundwater Resources Association, May 25, 2005, San Jose, CA.
5. C.B. Liban, K. Hancock, S. Liu, K. Johnson, M. Rigby. 2003. Calculating vapor intrusion to indoor air from groundwater and soil: an evolving science. PSAAPG and WRSPE, May 22, 2003, Long Beach, CA.
6. K.M. Johnson and M.C. Rigby. 2003. Predicted and measured indoor vapour concentrations in housing over a VOC plume. Society for Risk Analysis, December 7-10, 2003, Baltimore, MD.

7. K.M. Johnson and M.C. Rigby. 2003. Predicted and measured indoor vapour concentrations in housing over a VOC plume at the Alameda Naval Air Station, California. Toxicology and Risk Assessment Conference, April 28 – May 1, 2003, Fairborn, OH.
8. K.M. Johnson and M.C. Rigby. 2003. Predicted and measured indoor vapor concentrations in housing over a VOC plume at the Alameda Naval Air Station, California. Toxicology and Risk Assessment Conference, April 28 – May 1, 2003, Fairborn, OH.

PRESENTATIONS – TOXICOLOGY

1. Torres, K.C., B.L. Kephart, D.F. Clark, M.F. Shibata, M.C. Rigby, T.E. Donn, and K.L. Waitman. 2002. Bioaccumulation of metals in terrestrial plants and invertebrates at Vandenberg Air Force Base, California. SETAC Annual Meeting, November 2002, Salt Lake City, UT.
2. D.F. Clark, B.L. Kephart, Torres, K.C., M.F. Shibata, M.C. Rigby, M. McElligott, and K.L. Waitman. 2003. Bioaccumulation of PCBs in terrestrial plants and invertebrates at Vandenberg Air Force Base, California. Toxicology and Risk Assessment Conference, April 28 – May 1, 2003, Fairborn, OH.
3. K.M. Johnson and M.C. Rigby. 2004. Dicyclopentadiene toxicity assessment. Poster presentation at the Society for Risk Analysis, December 5-8, 2004, Palm Springs, CA.

PRESENTATIONS – PARASITES

1. Rigby, M. C. and Jokela, J. Costs of Predator avoidance and Environmental Stress in snails. Annual meeting SSE, ASN, SSB in Madison June 20-24, 1999.
2. M. C. Rigby and Y. Moret. Life history trade-offs and immune defense. Evolutionary biology of host-parasite relationship: reality meets models. Laboratoire Arago, Banyuls-sur-Mer, France, 3-7 May 1999.
3. Rigby, M. C. Cantieni, M., and Jokela, J. Costs and interactions among defenses in snails. IIIrd cycle meeting: Life-history, immunocompetence and resistance against parasites. University of Neuchâtel, Neuchâtel, Switzerland, 11-12 March 1999.
4. Rigby, M. C. Cantieni, M., and Jokela, J. Costs and interactions among defenses in snails. Zoologia et Botanica, Swiss Society of Zoologists, Zürich, Switzerland, 17-19 February 1997.
5. Rigby, M. C. and Jokela, J. Costs of Predator avoidance and Environmental Stress in snails. Annual meeting SSE, ASN, SSB in Vancouver June 20-24, 1998.
6. Rigby, M. C. Camallanid nematodes of Indo-Pacific coral reef fishes. Presented at the 5th Indo-Pacific Fish Conference, Nouméa, New Caledonia, 3-8th November 1997.
7. Font, W. F., and Rigby, M. C. Occurrence of *Spirocamallanus istiblenni* in tide pool and stream fishes of Hawai'i. Abstract of paper presented at the annual meeting of the Southeastern Society of Parasitologists, Greenville, South Carolina, April 17, 1997.

8. Rigby, M. C. and Holmes, J. C. Nematode parasites of fishes in the eastern south Pacific. Presented to the American Society of Parasitologists in June 1996. (Title doesn't say much, but it is about the possibility of multiple invasions of the nematode genus *Spirocamallanus* to French Polynesia.)

REVIEWER FOR THE FOLLOWING JOURNALS/ORGANIZATIONS:

Folia Parasitologica

The Journal of Parasitology

Grant Agency of the Czech Republic

Evolution

Ecology Letters

Marine Ecology Progress Series

PROFESSIONAL AFFILIATIONS

American Society of Parasitologists (1995 –)

Helminthological Society of Washington (1996 -)

American Institute of Biological Sciences (2000-)

Society for Risk Analysis (2004-)

WESTERN COLLEGE OF VETERINARY MEDICINE

CURRICULUM VITAE

FOR

JANZ, David Michael

Department of Veterinary Biomedical Sciences

1. **PERSONAL:**



2. **ACADEMIC CREDENTIALS:**

Degree	University	Year	Program
Ph.D.	University of British Columbia, Vancouver, BC	1995	Pharmacology and Toxicology
M.Sc.	Trent University, Peterborough, ON	1991	Watershed Ecosystems
B.Sc.	Simon Fraser University, Burnaby, BC	1987	Ecology Major; Environmental Toxicology Minor

3. **OTHER CREDENTIALS:** None

4. **APPOINTMENTS AND PROMOTIONS AT U OF S:**

Classification	Status	Year(s)	Department
Acting Director, Toxicology Centre		2010-present	
Professor	With Tenure	2008 - present	Veterinary Biomedical Sciences
Associate Professor	With Tenure	2007 - 2008	Veterinary Biomedical Sciences
Associate Professor	Tenure Track	2002 - 2007	Veterinary Biomedical Sciences

5. **ASSOCIATE MEMBERSHIPS:**

Society of Toxicology	2007 – present
Canadian Society of Zoologists	2007 – present
Society of Environmental Toxicology and Chemistry	1989 - present
Canadian Geographical Society	1991 - present
Society for Integrative and Comparative Biology	1997 – 2005
Society of Toxicology of Canada	1992 - 1997
Canadian Federation of Biological Societies	1992 - 1997

6. **LEAVES:**

Sabbatical leave, January 1 to June 30, 2010

7. HONOURS (MEDALS, FELLOWSHIPS, PRIZES):

7.1 PERSONAL:

Pfizer Animal Health Award for Research Excellence	2006
Watkins Young Investigator Award, Wichita State University	1998
SETAC Student Travel Award	1994
Dr. W.W. Simpson Memorial Award, UBC	1994
UBC Graduate Student Travel Award	1994
UBC Graduate Fellowship	1993 (Declined)
Eco-Research Doctoral Fellowship	1993, 1994
NSERC Postgraduate Scholarship	1991, 1992
Governor General's Gold Medal	1991
Ontario Graduate Scholarship	1988, 1989
Burt Bursary, Simon Fraser University	1986

7.2 HONOURS TO SUPERVISED STUDENTS:

- Hauck, D.W. 2010. Third place, Best Student Platform Presentation, SETAC North America 31st Annual Meeting, Portland, OR, November 7, 2010.
- Driedger, K. 2010. Highlighted article in journal *Ecotoxicology and Environmental Safety*.
- Macbeth, B.J. 2009. 1st place, Best Poster Award (Animal Sciences category), University of Saskatchewan Life and Health Sciences Research Conference.
- Thomas, J.K. 2009. 1st place, Best Poster Award (Environmental Sciences category), University of Saskatchewan Life and Health Sciences Research Conference.
- Kelly, J.M. 2008. Dr. Richard Playle Award for Best Master's Thesis in Aquatic Toxicology, 35th Annual Aquatic Toxicity Workshop, Saskatoon, SK.
- Kelly, J.M. 2008. Highlighted article in journal *Ecotoxicology and Environmental Safety*.
- Bennett, P.M. 2007. Highlighted article in journal *Ecotoxicology and Environmental Safety*.
- Muscattello, J.R. 2006. 2nd place, Best Student Poster Award, 33rd Annual Aquatic Toxicity Workshop, Jasper, AB.
- Muscattello, J.R. 2006. 2nd place, Best Poster Award (Environmental Sciences category), University of Saskatchewan Life and Health Sciences Research Conference.
- Lin, L. 2005. Runner-up, Best Student Poster Award, 32nd Annual Aquatic Toxicity Workshop, Waterloo, ON.
- Brasfield, S.M. 2002. SETAC Europe Best Publication Award in Environmental Research.
- Porter, C.M. 2001. Best Student Poster Award, SETAC Regional Meeting, Stillwater, OK.
- Savabieasfahani, M. 2000. Best Student Poster Award, SETAC Regional Meeting, Leavenworth, KS.
- Johnson, J.L. 2000. Best Student Platform Award, SETAC Regional Meeting, Leavenworth, KS.
- Savabieasfahani, M. 1999. Best Student Poster Award, SETAC Regional Meeting, Carbondale, IL.

8. PREVIOUS POSITIONS RELEVANT TO U OF S EMPLOYMENT:

Position	Institution	Year(s)
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Assistant Professor (Tenure Track)	Department of Zoology, Oklahoma State University, Stillwater, OK USA	1997 - 2002
Postdoctoral Researcher	Department of Zoology, University of Guelph, Guelph, ON	1995 - 1997
Teaching Assistant	Environmental and Resource Studies, Trent University, Peterborough, ON	1988 - 1991
Research Technician	Department of Biological Sciences, Simon Fraser University, Burnaby, BC	1987 - 1988
Research Technician	Fisheries and Oceans Canada, West Vancouver, BC	1987
Research Assistant	Department of Biological Sciences, Simon Fraser University, Burnaby, BC	1985 - 1986

9. TEACHING RECORD:

9.1 SCHEDULED INSTRUCTIONAL ACTIVITY:

Year	Course Description	Instruction Type	Enrollment	Yearly Instr. Hours
2010-2011	TOX 300.3 General Principles of Toxicology	Lec	56	33
	VBMS 333.6 Basic Principles of Pharmacology	Lec	76	8
	TOX 301.3 Environmental Toxicology	Lec	54	1.5
2009-2010	TOX 412.3 Toxicology of Industrial Pollutants	Lec	23	1
	TOX 300.3 General Principles of Toxicology	Lec	51	33
	VBMS 333.6 Basic Principles of Pharmacology	Lec	76	8
	TOX 480.3 Research Toxicology	Lab	1	39
2008-2009	TOX 300.3 General Principles of Toxicology	Lec	50	33
	VBMS 333.6 Basic Principles of Pharmacology	Lec	76	8
	TOX 412.3 Toxicology of Industrial Pollutants	Lec	23	32
	TOX 480.3 Research Toxicology	Lab	1	39
	TOX 301.3 Environmental Toxicology	Lec	52	1.5
2007-2008	TOX 300.3 General Principles of Toxicology	Lec	56	35
	VBMS 323.6 Basic Principles of Pharmacology	Lec	72	8
	TOX 412.3	Lec	24	20

	Toxicology of Industrial Pollutants			
	TOX 842.3 Biochemical Toxicology	Lec	8	37
	TOX 301.3 Environmental Toxicology	Lec	52	1.5
2006-2007	TOX 300.3 General Principles of Toxicology	Lec	49	35
	VBMS 323.6 Basic Principles of Pharmacology	Lec	70	8
	TOX 412.3 Toxicology of Industrial Pollutants	Lec	22	23
	TOX 301.3 Environmental Toxicology	Lec	55	1.5
2005-2006	TOX 300.3 General Principles of Toxicology	Lec	54	35
	VBMS 323.6 Basic Principles of Pharmacology	Lec	70	8
	TOX 412.3 Toxicology of Industrial Pollutants	Lec	20	20
	TOX 480.3 Research Toxicology	Lab	1	39
	TOX 898.3 Biochemical Toxicology	Lec	7	39
	TOX 301.3 Environmental Toxicology	Lec	58	1.5
2004-2005	TOX 300.3 General Principles of Toxicology	Lec	44	35
	VBMS 323.6 Basic Principles of Pharmacology	Lec	70	9
	TOX 412.3 Toxicology of Industrial Pollutants	Lec	11	19
	TOX 481.6 Toxicology Research	Lab	1	78
	VBMS 836.5 General Toxicology I	Lec	12	7.5
2003-2004	TOX 300.3 General Principles of Toxicology	Lec	21	37
	VBMS 323.6 Basic Principles of Pharmacology	Lec	70	9
	TOX 412.3 Toxicology of Industrial Pollutants	Lec	9	12
	TOX 480.3 Toxicology Research	Lab	1	39
	TOX 898.3 Biochemical Toxicology	Lec	9	39
	VBMS 836.5 General Toxicology I	Lec	12	7.5

	TOX 301.3 Environmental Toxicology	Lec	50	1.5
2002-2003	VBMS 300.3 General Principles of Toxicology	Lec	9	39
	VBMS 323.6 Basic Principles of Pharmacology	Lec	70	8
	TOX 301.3 Environmental Toxicology	Lec	22	1.5

9.2 UNSCHEDULED INSTRUCTIONAL ACTIVITY:

Participated in Problem-Based Learning exercise with first year veterinary students
(Physiology, 2003, 2004 and 2005)

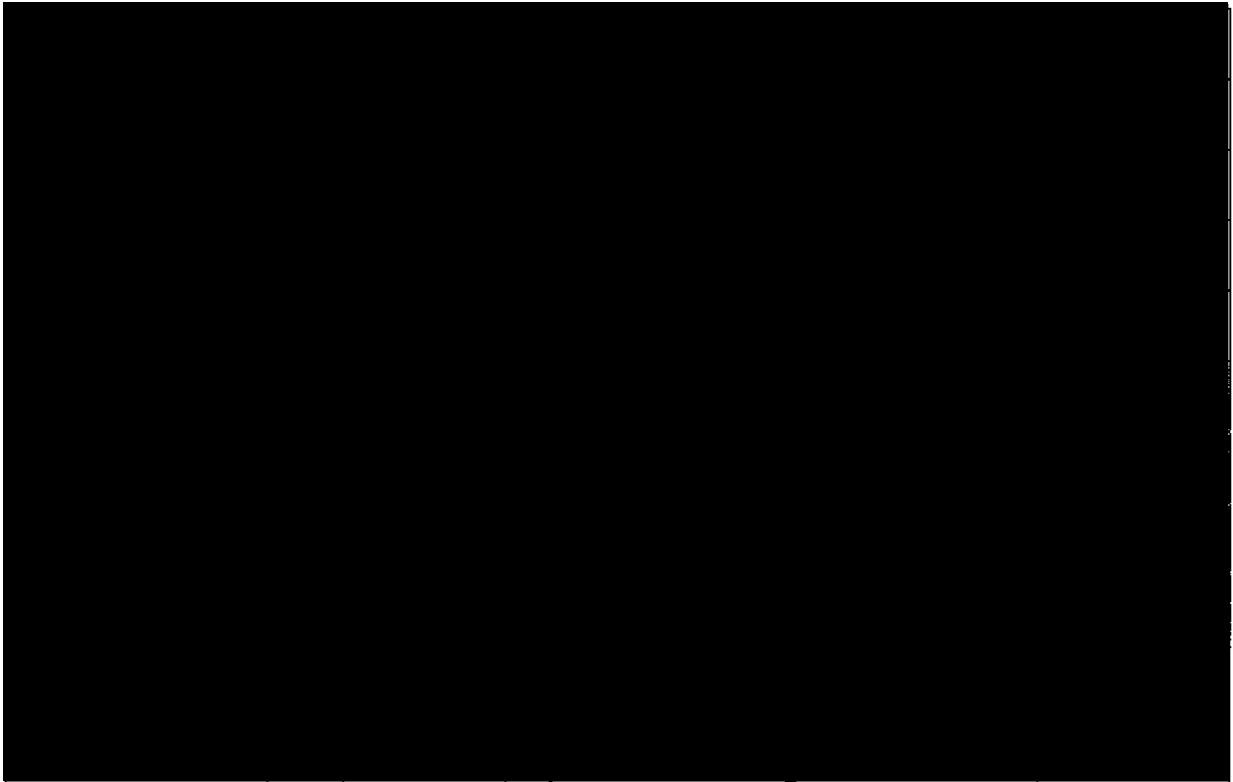
9.2.1 UNIVERSITY COURSES TAUGHT OUTSIDE U of S:

Oklahoma State University:

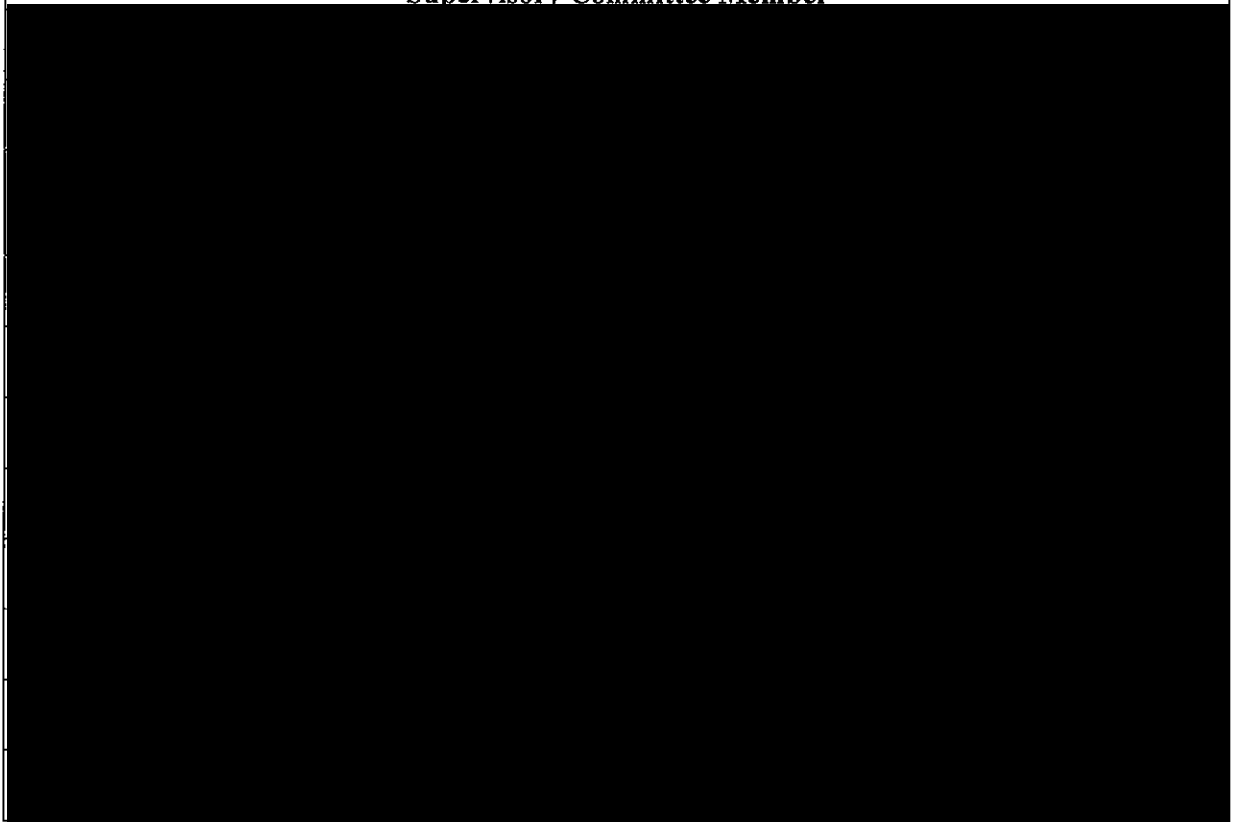
ZOOL 4303.3 Environmental Toxicology (2002)
 ZOOL 5010.2 Comparative Endocrinology (Graduate seminar) (2000)
 ZOOL 4243.3 Pharmacology (1999 and 2001)
 ZOOL 4283.3 Endocrinology (1999 and 2001)
 ZOOL 5323.3 Cellular and Molecular Toxicology (1998 and 2000)
 BIOL 1304.3 Principles of Biology (1998)
 ZOOL 5010.2 Advances in Environmental Toxicology (Graduate seminar) (1998)
 BIOL 1114.3 Introductory Biology: Populations and Ecosystems (1997)

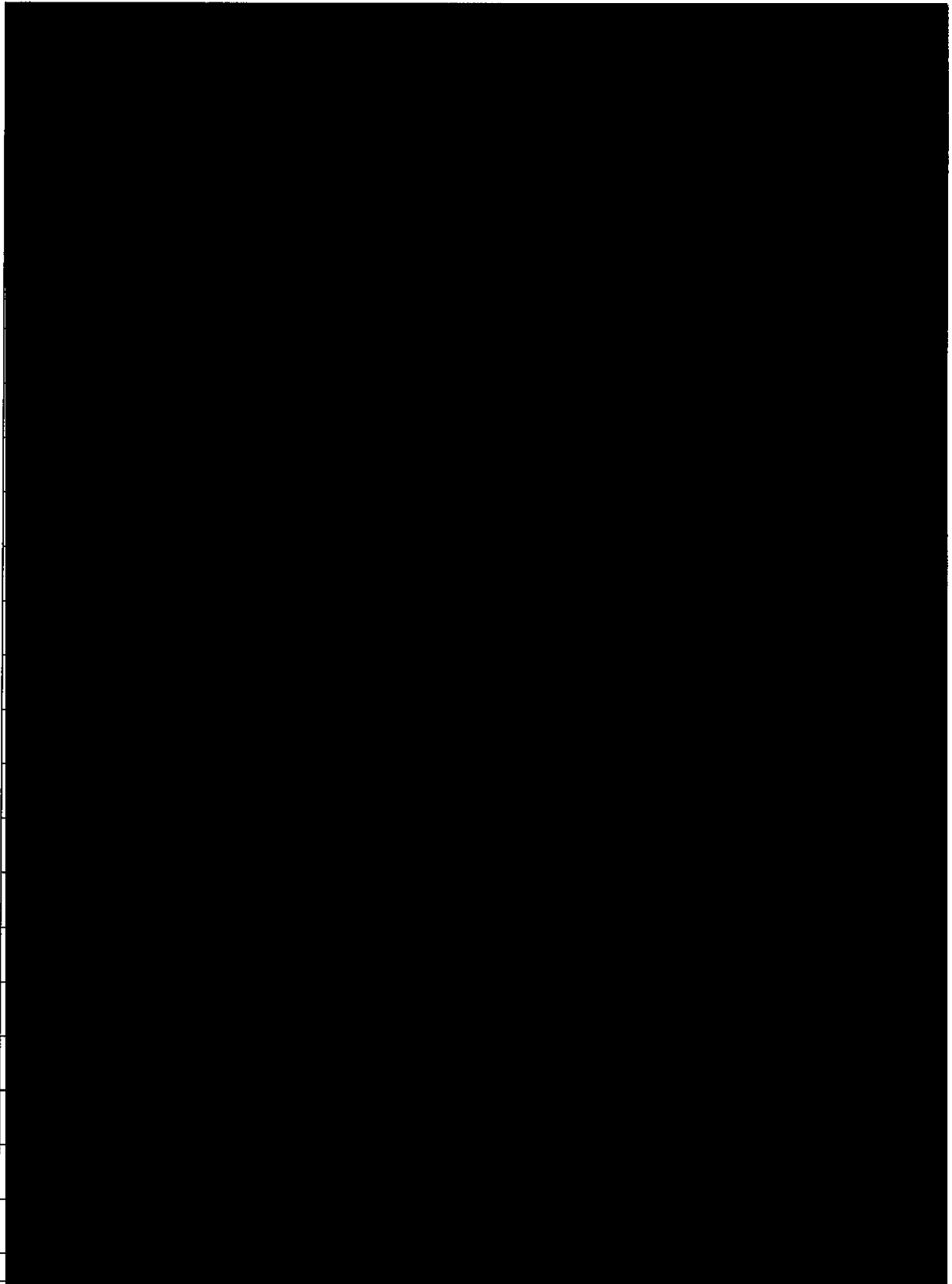
9.3 POSTGRADUATE STUDENTS SUPERVISED OR ON THEIR COMMITTEE:

Name	Degree Sought (Program)	Thesis Subject Area	Year
Supervisor or *Co-supervisor			



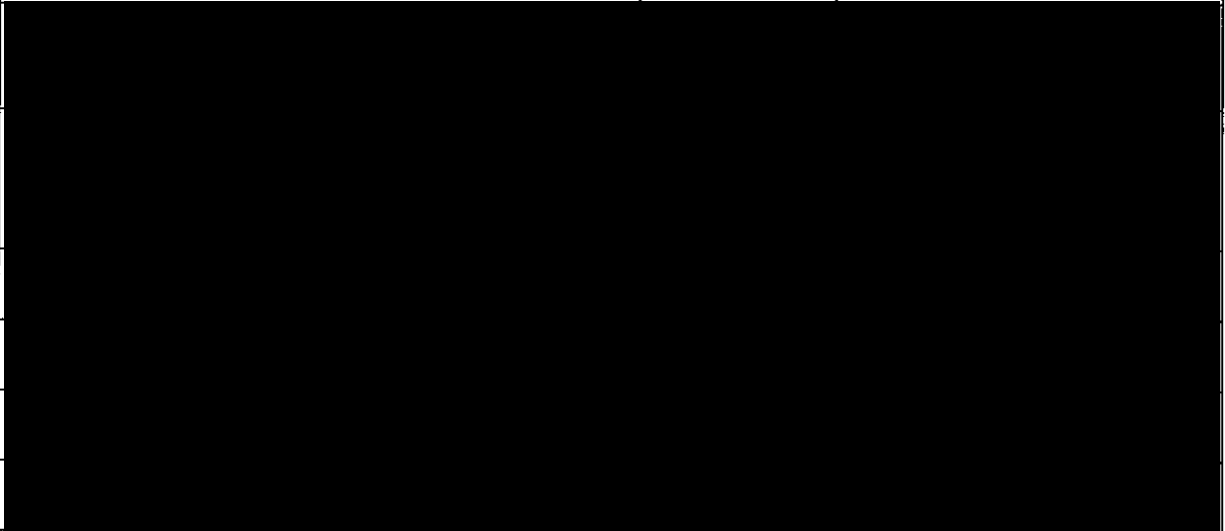
Supervisory Committee Member



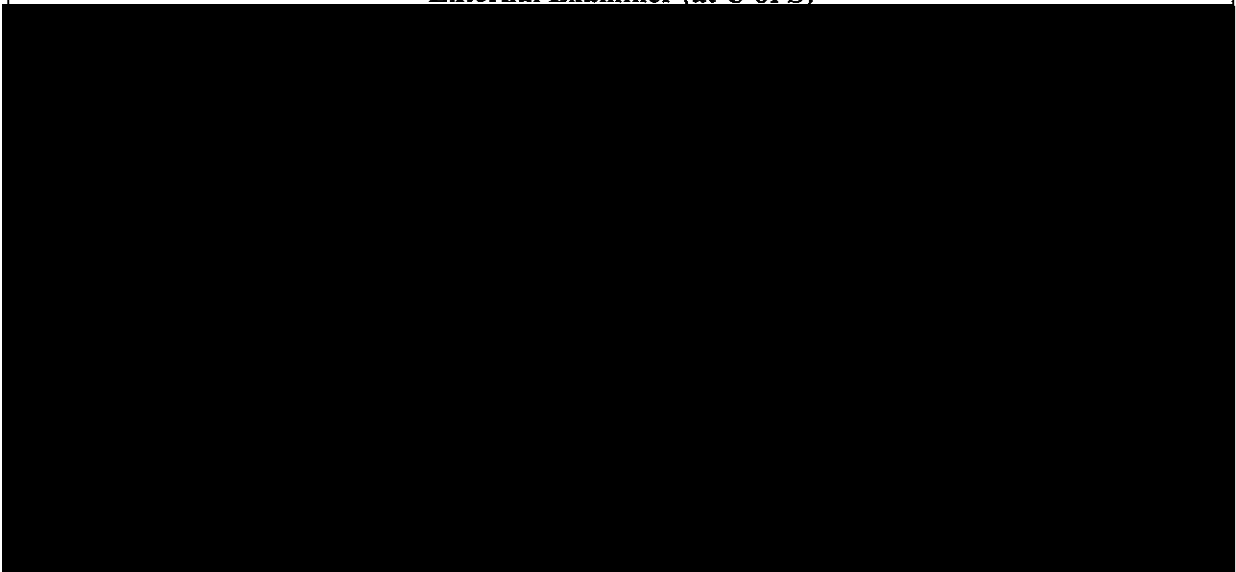




External Examiner (Outside U of S)

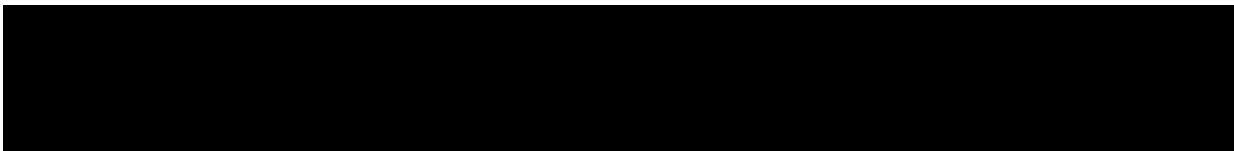


External Examiner (at U of S)

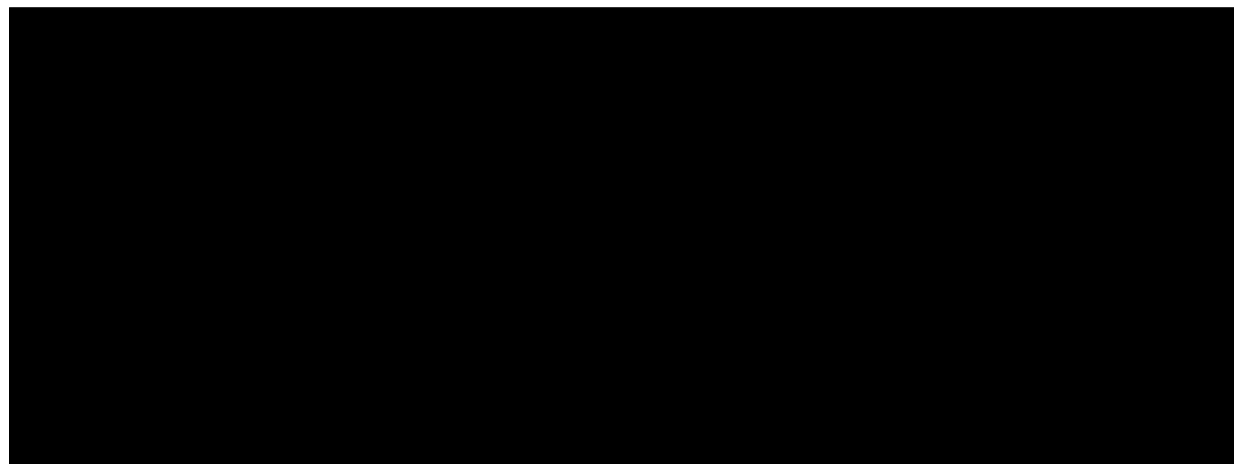


Chair of Defence

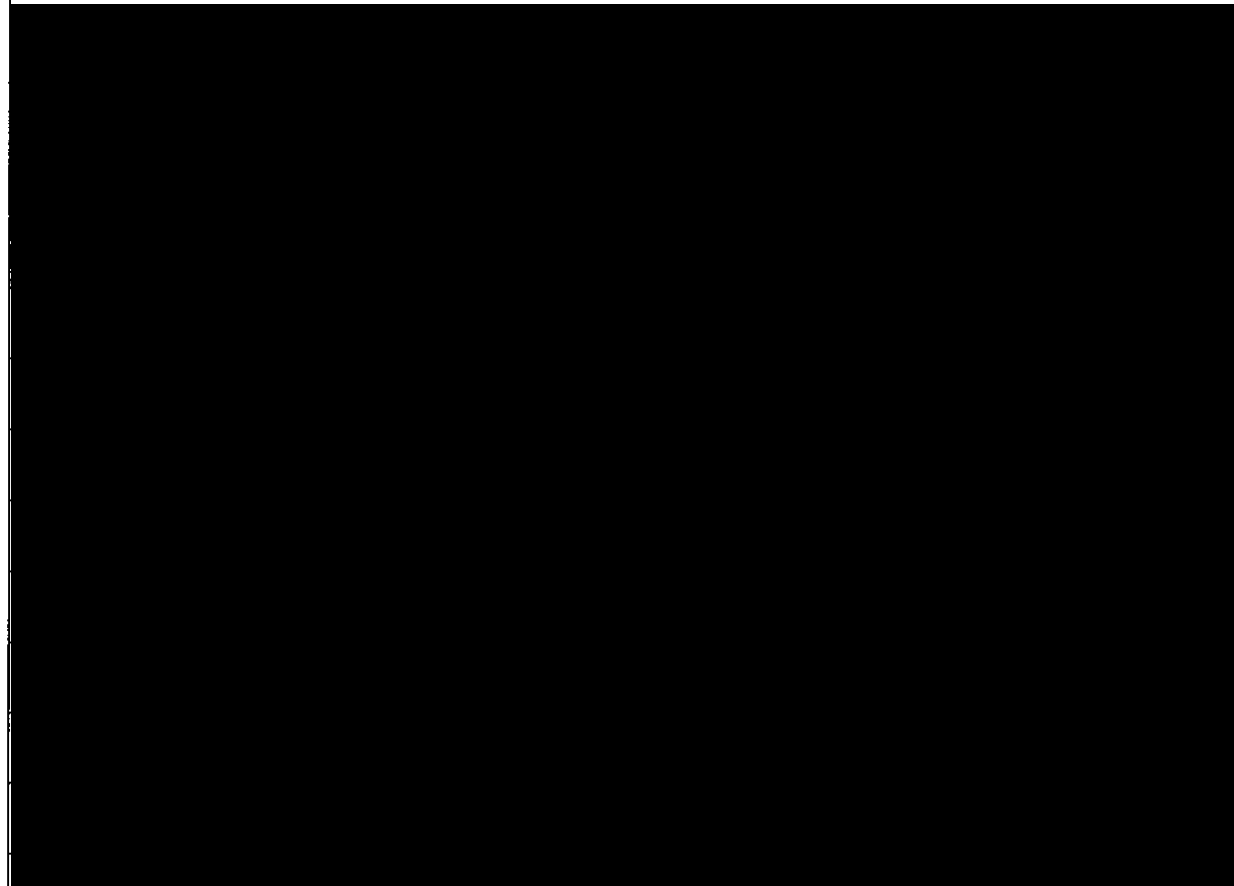




9.4 UNDERGRADUATE STUDENT SUPERVISION:



Summer Research Assistant



10. THESES SUPERVISED OR * CO-SUPERVISED:

- Carlson, R.I. 2011. Development and application of an antibody-based protein microarray to assess stress in grizzly bears (*Ursus arctos*). Ph.D. thesis, University of Saskatchewan.
- Phibbs, J. 2011. Evaluating dietary selenium uptake and speciation downstream of a uranium processing mill using caged small-bodied fish. M.Sc. thesis, University of Saskatchewan.
- Driedger, K. 2009. Effects of metal mine and municipal wastewater on growth and energy stores in juvenile fishes. M.Sc. thesis, University of Saskatchewan.
- Muscatello, J.R. 2009. Selenium accumulation and effects in aquatic organisms downstream of uranium mining and milling operations in northern Saskatchewan. Ph.D. thesis, University of Saskatchewan.
- Kelly, J.M. 2008. Ecotoxicological assessment of juvenile northern pike inhabiting lakes downstream of a uranium mill. M.Sc. thesis, University of Saskatchewan.
- Lin, L.L. 2007. Effects of binary mixtures of xenoestrogens on gonadal development and reproduction in zebrafish. M.Sc. thesis, University of Saskatchewan.

- Bennett, P.M. 2006. Effects of uranium mining and milling effluents on juvenile fish bioenergetics, growth and overwinter survival. M.Sc. thesis, University of Saskatchewan.
- * Rickwood, C.J. 2006. Improved environmental relevance of a standard fish bioassay. Ph.D. thesis, University of Saskatchewan.
- Brasfield, S.M. 2002. Development of a reptile model for assessment of endocrine-mediated toxicity. M.S. thesis, Oklahoma State University, Stillwater, OK.
- * Wilson, J.A. 2002. Ecotoxicological risks associated with land treatment of petrochemical wastes in Oklahoma. Ph.D. thesis, Oklahoma State University, Stillwater, OK.
- Hill, R.L., Jr. 2002. Effects of ethynyl estradiol and 4-nonylphenol on reproduction and development in zebrafish (*Danio rerio*). M.S. thesis, Oklahoma State University, Stillwater, OK.
- Johnson, J.L. 2001. A field study of metal effects on the reproductive physiology of teleosts. M.S. thesis, Oklahoma State University, Stillwater, OK.
- Savabieasfahani, M. 2001. The relevance of toxicant-induced apoptosis to reproductive impairment in fish and mammals. Ph.D. thesis, Oklahoma State University, Stillwater, OK.
- Carlson, R.I. 2001. Toxicological effects of landfarmed oil refinery wastes on cotton rat (*Sigmodon hispidus*) liver. M.S. thesis, Oklahoma State University, Stillwater, OK.

11. BOOKS, CHAPTERS IN BOOKS, EXPOSITORY AND REVIEW ARTICLES:

- Janz, D.M. 2011. Selenium. Pp. 327-374 In: C.W. Wood, A.P. Farrell and C.J. Brauner (Eds.) *Fish Physiology Vol 31A, Homeostasis and Toxicology of Essential Metals*. Elsevier, San Diego, CA.
- Janz, D.M., D.K. DeForest, M.L. Brooks, P.M. Chapman, G. Gilron, D. Hoff, W.D. Hopkins, D.O. McIntyre, C.A. Mebane, V.P. Palace, J.P. Skorupa and M. Wayland. 2010. Selenium toxicity to aquatic organisms. Pp. 141-231 In: P.M. Chapman, W.J. Adams, M.L. Brooks, C.G. Delos, S.N. Luoma, W.A. Maher, H.M. Ohlendorf, T.S. Presser and D.P. Shaw (Eds.) *Ecological Assessment of Selenium in the Aquatic Environment*. CRC Press, Boca Raton, FL.
- Wood, A.W., D.M. Janz and G. Van Der Kraak. 2005. Cell death: Investigation and application in fish toxicology. Pp. 303-330 In: T.P. Mommsen and T.W. Moon (Eds.) *Biochemistry and Molecular Biology of Fishes Vol. 6 – Environmental Toxicology*. Elsevier Press, New York, NY.
- Smits, J.E.G. and D.M. Janz. 2005. Immunotoxicology in terrestrial wildlife. Pp. 129-145 In: B. Blakley, P. Brousseau, M. Fournier, J.E.G. Smits and H. Tryphonas (Eds.).

Models and Approaches in Immunotoxicology. Taylor and Francis, London, UK.

Janz, D.M. 2005. Dinitrophenols. Pp. 59-60 In: P. Wexler (Ed.) *Encyclopedia of Toxicology, 2nd Edition*. Elsevier Press, Oxford, UK.

Janz, D.M. 2005. Dithiocarbamates. Pp. 86-89 In: P. Wexler (Ed.) *Encyclopedia of Toxicology, 2nd Edition*. Elsevier Press, Oxford, UK.

Janz, D.M. 2005. Chlorobenzilate. Pp. 559-561 In: P. Wexler (Ed.) *Encyclopedia of Toxicology, 2nd Edition*. Elsevier Press, Oxford, UK.

Janz, D.M. 2005. Hexachlorobutadiene. Pp. 511-513 In: P. Wexler (Ed.) *Encyclopedia of Toxicology, 2nd Edition*. Elsevier Press, Oxford, UK.

Janz, D.M. and L.P. Weber. 2000. Microscopic functional anatomy: Endocrine system. Pp. 415-439 In: G.K. Ostrander (Ed.). *The Handbook of Experimental Animals – Laboratory Fish*. Academic Press, London, UK.

Janz, D.M. 2000. Gross functional anatomy: Endocrine system. Pp. 189-217 In: G.K. Ostrander (Ed.). *The Handbook of Experimental Animals – Laboratory Fish*. Academic Press, London, UK.

Van Der Kraak, G., T. Zacharewski, D.M. Janz, B.M. Sanders and J.W. Gooch. 1998. Comparative endocrinology and mechanisms of endocrine modulation in fish and wildlife. Pp. 97-109 In: R. Kendall, R. Dickerson, J.P. Giesy and W. Suk (Eds.). *Principles and Processes in Evaluating Endocrine Disruption in Wildlife*. SETAC Press, Pensacola, FL.

Van Der Kraak, G., J.P. Chang and D.M. Janz. 1997. Reproduction. Pp. 465-488 In: D.H. Evans (Ed.). *The Physiology of Fishes, 2nd Edition*. CRC Press, Boca Raton, FL.

12. PAPERS IN REFEREED JOURNALS:

Phibbs, J., E. Franz, D. Hauck, M. Gallego, J.J. Tse, I.J. Pickering, K. Liber and D.M. Janz. 2011. Evaluating the trophic transfer of selenium in aquatic ecosystems using caged fish, X-ray absorption spectroscopy and stable isotope analysis. *Ecotoxicology and Environmental Safety*, in press (doi:10.1016/j.ecoenv.2011.06.015).

Macbeth, B.J., H.D. Frimer and D.M. Janz. 2011. Use of portable ultrasonography to determine ovary size and fecundity non-lethally in northern pike (*Esox lucius*) and white sucker (*Catostomus commersoni*). *Water Quality Research Journal of Canada*, in press (doi:10.2166/wqrjc.2011.011).

Wiseman, S., J.K. Thomas, E. Higley, O. Hursky, M. Pietrock, J.C. Raine, J.P. Giesy, D.M. Janz and M. Hecker. 2011. Chronic exposure to dietary selenomethionine increases gonadal steroidogenesis in female rainbow trout. *Aquatic Toxicology* 105, 218-226.

- Ashley, N.T., P.S. Barboza, B.J. Macbeth, D.M. Janz, M.R.L. Cattet, R.K. Booth and S.K. Wasser. 2011. Glucocorticosteroid concentrations in feces and hair of captive caribou and reindeer following adrenocorticotrophic hormone challenge. *General and Comparative Endocrinology* 172, 382-391.
- Driedger, K.L.F., L.P. Weber, I.K. Birtwell and D.M. Janz. 2011. Growth, condition and energy stores of Arctic grayling fry inhabiting natural and artificial constructed Arctic tundra streams. *Limnologia* 41, 63-69.
- Thomas, J.K. and D.M. Janz. 2011. Dietary selenomethionine exposure in adult zebrafish alters swimming performance, energetics and the physiological stress response. *Aquatic Toxicology* 102, 79-86.
- Phibbs, J., C.I.E. Wiramanaden, D. Hauck, I.J. Pickering, K. Liber and D.M. Janz. 2011. Selenium uptake and speciation in wild and caged fish downstream of a metal mining and milling discharge. *Ecotoxicology and Environmental Safety* 74, 1139-1150.
- Macbeth, B.J., M.R.L. Cattet, G.B. Stenhouse, M.L. Gibeau and D.M. Janz. 2010. Hair cortisol concentration as a noninvasive measure of long-term stress in free-ranging grizzly bears (*Ursus arctos*): Considerations and implications for other wildlife. *Canadian Journal of Zoology* 88, 935-949.
- Driedger, K., L.P. Weber, C.J. Rickwood, M.G. Dube and D.M. Janz. 2010. Growth and energy stores on juvenile fathead minnows exposed to metal mine waste water in simulated winter and summer conditions. *Ecotoxicology and Environmental Safety* 73, 727-734. **(Highlighted article)**
- Kelly, J.M. and D.M. Janz. 2009. Assessment of oxidative stress and histopathology in juvenile northern pike (*Esox lucius*) inhabiting lakes downstream of a uranium mill. *Aquatic Toxicology* 92, 240-249.
- Muscatello, J.R. and D.M. Janz. 2009. Assessment of larval deformities and selenium accumulation in northern pike (*Esox lucius*) and white sucker (*Catostomus commersoni*) exposed to metal mining effluent. *Environmental Toxicology and Chemistry* 28, 609-618.
- Muscatello, J.R. and D.M. Janz. 2009. Selenium accumulation in aquatic biota downstream of a uranium mining and milling operation. *Science of the Total Environment* 407, 1318-1325.
- Driedger, K., L.P. Weber, C.J. Rickwood, M.G. Dubé and D.M. Janz. 2009. Overwinter alterations in energy stores and growth in juvenile fishes inhabiting areas receiving metal mining and municipal wastewater effluents. *Environmental Toxicology and Chemistry* 28, 296-304.
- Muscatello, J.M., A.M. Belknap and D.M. Janz. 2008. Accumulation of selenium in aquatic systems downstream of a uranium mining operation in northern Saskatchewan, Canada. *Environmental Pollution* 156, 387-393.

- Weber, L.P., M.G. Dubé, C.J. Rickwood, K.L. Driedger, C. Portt, C.I. Brereton and D.M. Janz. 2008. Effects of multiple effluents on resident fish from Junction Creek, Sudbury, Ontario. *Ecotoxicology and Environmental Safety* 70, 433-445.
- Kelly, J.M. and D.M. Janz. 2008. Altered energetics and parasitism in juvenile northern pike (*Esox lucius*) inhabiting metal-mining contaminated lakes. *Ecotoxicology and Environmental Safety* 70, 357-369. **(Highlighted Article)**
- Rickwood, C.J., M.G. Dubé, L.P. Weber, K.L. Driedger and D.M. Janz. 2008. Assessing effects of a mining and municipal sewage effluent mixture on fathead minnow (*Pimephales promelas*) reproduction using a novel, field-based trophic-transfer artificial stream. *Aquatic Toxicology* 86, 272-286.
- Brasfield, S.M., L.G. Talent and D.M. Janz. 2008. Reproductive and thyroid hormone profiles in captive Western fence lizards (*Sceloporus occidentalis*) following a period of brumation. *Zoo Biology* 27, 36-48.
- Ogunkoya, A.E., A.G. Van Kessel, D.M. Janz and M.D. Drew. 2007. Dietary influence of replacing fish meal and oil with canola protein concentrate and vegetable oils on growth performance, fatty acid composition and organochlorine residues in rainbow trout (*Oncorhynchus mykiss*). *Aquaculture* 267, 260-268. **(Invited Article)**
- Bennett, P.M. and D.M. Janz. 2007. Bioenergetics and growth of young-of-the-year northern pike (*Esox lucius*) and burbot (*Lota lota*) exposed to metal mining effluent. *Ecotoxicology and Environmental Safety* 68, 1-12. **(Highlighted Article)**
- Bennett, P.M. and D.M. Janz. 2007. Seasonal changes in morphometric and biochemical endpoints in northern pike (*Esox lucius*), burbot (*Lota lota*) and slimy sculpin (*Cottus cognatus*). *Freshwater Biology* 52, 2056-2072.
- Bennett, P.M., L.P. Weber and D.M. Janz. 2007. Comparison of chloroform-methanol extracted and solvent-free triglyceride determinations in four fish species. *Journal of Aquatic Animal Health* 19, 179-185.
- Rickwood, C.J., M.G. Dubé, L.P. Weber, K.L. Driedger and D.M. Janz. 2006. Assessing effects of metal mining effluent on fathead minnow (*Pimephales promelas*) reproduction in a trophic transfer exposure system. *Environmental Science and Technology* 40, 6489-6497.
- Muscatello, J.R., P.M. Bennett, K.T. Himbeault, A.M. Belknap and D.M. Janz. 2006. Larval deformities associated with selenium accumulation in northern pike (*Esox lucius*) exposed to metal mining effluent. *Environmental Science and Technology* 40, 6506-6512.
- Lin, L.L. and D.M. Janz. 2006. Effects of binary mixtures of xenoestrogens on gonadal development and reproduction in zebrafish. *Aquatic Toxicology* 80, 382-395.

- Wilson, J.A., R.L. Lochmiller and D.M. Janz. 2005. Population dynamics of cotton rats (*Sigmodon hispidus*) inhabiting abandoned petroleum landfarms in Oklahoma, USA. *Ecotoxicology* 15, 19-30.
- Wilson, J.A., R.L. Lochmiller and D.M. Janz. 2004. Dynamics of rodent assemblages inhabiting abandoned petroleum landfarms in Oklahoma. *Ecological Applications* 14, 1016-1027.
- Stokes, E.A., W. Lonergan, L.P. Weber, D.M. Janz, G.C. Balch, C.D. Metcalfe and M.S. Grober. 2004. Decreased apoptosis in the forebrain of adult male medaka (*Oryzias latipes*) after aqueous exposure to ethinylestradiol. *Comparative Biochemistry and Physiology (Part C)* 138, 163-167.
- Brasfield, S.M., K. Bradham, J.B. Wells, L.G. Talent, R.P. Lanno and D.M. Janz. 2004. Evaluation of fence lizard (*Sceloporus spp.*) eggs as a terrestrial vertebrate model for assessing bioavailability and *in ovo* effects of soil contaminants. *Chemosphere* 54, 1643-1651.
- Weber, L.P., G.C. Balch, C.D. Metcalfe and D.M. Janz. 2004. Increased kidney, liver, and testicular cell death after chronic exposure to 17 α -ethinylestradiol in medaka (*Oryzias latipes*). *Environmental Toxicology and Chemistry* 23, 792-797.
- Yoo, J.L. and D.M. Janz. 2003. Tissue specific HSP70 levels and reproductive physiological responses in fishes inhabiting a metal contaminated creek. *Archives of Environmental Contamination and Toxicology* 45, 110-120.
- Weber, L.P., P.S. Higgins, R.I. Carlson and D.M. Janz. 2003. Development and validation of methods for measuring multiple biochemical indices of condition in juvenile fishes. *Journal of Fish Biology* 63, 637-658.
- Hill, R.L. Jr. and D.M. Janz. 2003. Developmental estrogenic exposure in zebrafish (*Danio rerio*): I. Effects on sex ratio and breeding success. *Aquatic Toxicology* 63, 417-429.
- Weber, L.P., R.L. Hill, Jr. and D.M. Janz. 2003. Developmental estrogenic exposure in zebrafish (*Danio rerio*): II. Histological evaluation of gametogenesis and organ toxicity. *Aquatic Toxicology* 63, 431-446.
- Porter, C.M. and D.M. Janz. 2003. Treated municipal sewage discharge affects multiple levels of biological organization in fishes. *Ecotoxicology and Environmental Safety* 54, 199-206.
- Schroder, J.L., N.T. Basta, M. Payton, J.A. Wilson, R.I. Carlson, D.M. Janz and R.L. Lochmiller. 2003. Ecotoxicological risks associated with land treatment of petrochemical wastes: I. Residual soil contamination and bioaccumulation by cotton rats (*Sigmodon hispidus*). *Journal of Toxicology and Environmental Health* 66, 305-325.

- Carlson, R.I., J.A. Wilson, J.L. Schroder, N.T. Basta, R.L. Lochmiller, and D.M. Janz. 2003. Ecotoxicological risks associated with land treatment of petrochemical wastes: II. Effects on hepatic Phase I and Phase II detoxification enzymes in cotton rats. *Journal of Toxicology and Environmental Health* 66, 327-343.
- Wilson, J.A., R.I. Carlson, J.L. Schroder, N.T. Basta, D.M. Janz and R.L. Lochmiller. 2003. Ecotoxicological risks associated with land treatment of petrochemical wastes: III. Immune function and hematology of cotton rats. *Journal of Toxicology and Environmental Health* 66, 345-363.
- Brasfield, S.M., L.P. Weber, L.G. Talent and D.M. Janz. 2002. Dose response and time course relationships for vitellogenin induction in male Western fence lizards (*Sceloporus occidentalis*) exposed to ethinylestradiol. *Environmental Toxicology and Chemistry* 21, 1410-1416. (SETAC Europe Best Publication Award on Environmental Research)
- Talent, L.G., J.N. Dumont, J.A. Bantle, D.M. Janz and S. Talent. 2002. Evaluation of western fence lizards, *Sceloporus occidentalis*, and eastern fence lizards, *S. undulatus*, as laboratory reptile models for toxicological investigations. *Environmental Toxicology and Chemistry* 21, 899-905.
- Weber, L.P., S.L. Diamond, S.M. Bandiera and D.M. Janz. 2002. Expression of HSP70 and CYP1A protein in ovary and liver of juvenile rainbow trout exposed to β -naphthoflavone. *Comparative Biochemistry and Physiology (Part C)* 131, 387-394.
- Weber, L.P., Y. Kiparissis, G.S. Hwang, A.J. Niimi, D.M. Janz and C.D. Metcalfe. 2002. Increased cellular apoptosis after chronic aqueous exposure to nonylphenol and quercetin in adult medaka (*Oryzias latipes*). *Comparative Biochemistry and Physiology (Part C)* 131, 51-59.
- Weber, L.P. and D.M. Janz. 2001. Effect of β -naphthoflavone and dimethylbenz[a]anthracene on apoptosis and HSP70 expression in juvenile channel catfish (*Ictalurus punctatus*) ovary. *Aquatic Toxicology* 54, 39-50.
- Janz, D.M., M.E. McMaster, L.P. Weber, K.R. Munkittrick and G. Van Der Kraak. 2001. Recovery of ovary size, follicle cell apoptosis and HSP70 expression in fish exposed to bleached pulp mill effluent. *Canadian Journal of Fisheries and Aquatic Sciences* 58, 620-625.
- Porter, C.M., D.R. Butler and D.M. Janz. 2000. Central Oklahoma bioassessment study: Evaluation of stream health using fish and macroinvertebrate communities as biological indicators. *Proceedings of the Oklahoma Academy of Science* 80, 61-70.

- Savabieasfahani, M., R.L. Lochmiller and D.M. Janz. 1999. Elevated ovarian and thymic cell apoptosis in wild cotton rats inhabiting petrochemical-contaminated terrestrial ecosystems. *Journal of Toxicology and Environmental Health* 57, 101-107.
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- Janz, D.M. and G. Van Der Kraak. 1997. Suppression of apoptosis by gonadotropin, 17 β -estradiol and epidermal growth factor in rainbow trout preovulatory ovarian follicles. *General and Comparative Endocrinology* 105, 186-193.
- Sanderson, J.T., D.M. Janz, G.D. Bellward and J.P. Giesy. 1997. Effects of embryonic and adult exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin on hepatic microsomal testosterone hydroxylase activities in great blue herons (*Ardea herodias*). *Environmental Toxicology and Chemistry* 16, 1304-1310.
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- Sanderson, J.T., D.M. Janz, G.D. Bellward and J.P. Geisy. 1996. Effects of embryonic and adult exposure to 2,3,7,8-TCDD on hepatic microsomal testosterone hydroxylase activities in great blue herons (*Ardea herodias*). *Organohalogen Compounds* 29, 178-183.
- Janz, D.M. and G.D. Bellward. 1996. *In ovo* 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure in three avian species. 1. Effects on thyroid hormones and growth during the perinatal period. *Toxicology and Applied Pharmacology* 139, 281-291.
- Janz, D.M. and G.D. Bellward. 1996. *In ovo* 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure in three avian species. 2. Effects on estrogen receptor and plasma sex steroid hormones during the perinatal period. *Toxicology and Applied Pharmacology* 139, 292-300.
- Bellward, G.D., D.M. Janz and J.T. Sanderson. 1995. Effects of *in ovo* TCDD exposure on wild avian species: Summary of a case study. *Organohalogen Compounds* 25, 99-103.

Janz, D.M., T.L. Metcalfe, C.D. Metcalfe and G.D. Haffner. 1992. Relative concentrations of cytochrome P450-active organochlorine compounds in liver and muscle of rainbow trout from Lake Ontario. *Journal of Great Lakes Research* 18, 759-765.

Janz, D.M. and C.D. Metcalfe. 1991. Nonadditive interactions of mixtures of 2,3,7,8-TCDD and 3,3',4,4'-tetrachlorobiphenyl on aryl hydrocarbon hydroxylase induction in rainbow trout (*Oncorhynchus mykiss*). *Chemosphere* 23, 467-472.

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Morgan, J.D., G.A. Vigers, A.P. Farrell, D.M. Janz and J.F. Manville. 1991. Acute avoidance reactions and behavioral responses of juvenile rainbow trout (*Oncorhynchus mykiss*) to Garlon 4, Garlon 3A and Vision herbicides. *Environmental Toxicology and Chemistry* 10, 73-80.

Janz, D.M., A.P. Farrell, J.D. Morgan and G.A. Vigers. 1991. Acute physiological stress responses of juvenile coho salmon (*Oncorhynchus kisutch*) to sublethal concentrations of Garlon 4, Garlon 3A and Vision herbicides. *Environmental Toxicology and Chemistry* 10, 81-90.

13. PAPERS IN NON-REFEREED JOURNALS: None

14. INVITED PAPERS IN PUBLISHED CONFERENCE PROCEEDINGS AND ABSTRACTS: None

15. CONTRIBUTED PAPERS IN PUBLISHED CONFERENCES PROCEEDINGS AND ABSTRACTS: None

16. TECHNICAL REPORTS RELEVANT TO ACADEMIC FIELD:

Liber, K., D.M. Janz and L.E. Burrige (Editors). 2009. Proceedings of the 35th Annual Aquatic Toxicity Workshop: October 5 to 8, 2008, Saskatoon, SK. *Canadian Technical Report of Fisheries and Aquatic Sciences* 2841: xiv + 174 p.

Janz, D.M. 2008. A critical evaluation of winter stress syndrome. Pages 162-178 in Selenium Tissue Thresholds: Tissue Selection Criteria, Threshold Development Endpoints, and Potential to Predict Population or Community Effects in the Field. Washington, D.C.: Report by GEI Consultants, Golder Associates, Parametrix, and University of Saskatchewan Toxicology Centre to the North America Metals Council – Selenium Working Group.

Janz, D.M. and J.R. Muscatello. 2008. Standard operating procedure for evaluating selenium-induced deformities in early life stages of freshwater fish. Pages 140-160 in Selenium Tissue Thresholds: Tissue Selection Criteria, Threshold Development Endpoints, and Potential to Predict Population or Community Effects in the Field.

Washington, D.C.: Report by GEI Consultants, Golder Associates, Parametrix, and University of Saskatchewan Toxicology Centre to the North America Metals Council – Selenium Working Group.

- Dubé, M.G., L.P. Weber, C.J. Rickwood, K.L. Driedger and D.M. Janz. 2006. Integrated environmental effects monitoring assessment for fish exposed to metal mine effluent, Inco Limited, Sudbury, Ontario. Technical Report submitted to Environment Canada by Inco Ltd. 177 pp.
- Driedger, K.L., L.P. Weber, I.K. Birtwell and D.M. Janz. 2004. Comparison of morphometric and biochemical measures of growth and condition in arctic grayling fry inhabiting natural and artificial arctic tundra streams. Technical Report submitted to BHP Billiton Diamonds Inc. 32 pp.
- Birtwell, I.K., J.S. Korstrom, P.M.F. Walton, C.J. Whitfield and D.M. Janz. 2003. An examination of the growth, behaviour and biochemical responses of juvenile coho salmon (*Oncorhynchus kisutch*) at the Capilano River Hatchery, North Vancouver, BC, in relation to changes in water quality and food between November 2001 and May 2002. *Canadian Technical Report of Fisheries and Aquatic Sciences* 2499:127p.
- Janz, D.M. and G. Van Der Kraak. 1996. Endocrine-modulating chemicals in the environment: Is there a scientific basis for concern? Manuscript written for Dow Chemical. (Revised and updated in January 2000)
- Morgan, J.D., D.M. Janz, G.A. Vigers and A.P. Farrell. 1989. Determination of threshold concentrations of forest-use herbicides causing rapid sublethal toxic effects towards salmonid fish. British Columbia Ministry of Forests and Lands. FRDA Project #2.46. pp. 1-90.

17. BOOK REVIEWS: None

17.1 SCIENTIFIC ARTICLE REVIEWS:

Editorial Board Member, *Aquatic Toxicology* (1998 – present)

Ad Hoc Manuscript Reviewer for Journals:

Acta Hydrochimica et Hydrobiologica
African Journal of Agricultural Research
Agriculture, Ecosystems and Environment
Aquaculture
Aquatic Toxicology
Archives of Environmental Contamination and Toxicology
Australasian Journal of Ecotoxicology
Brazilian Journal of Morphological Sciences
Bulletin of Environmental Contamination and Toxicology
Canadian Journal of Fisheries and Aquatic Sciences
Chemico-Biological Interactions

Chemosphere
Clean – Soil Air Water
Comparative Biochemistry and Physiology
Continental Shelf Research
Ecotoxicology and Environmental Safety
Environment International
Environmental Pollution
Environmental Science and Pollution Research
Environmental Science and Technology
Environmental Technology
Environmental Toxicology
Environmental Toxicology and Chemistry
Environmental Toxicology and Pharmacology
Fish Physiology and Biochemistry
General and Comparative Endocrinology
Human and Ecological Risk Assessment
Integrated Environmental Assessment and Management
International Journal of Environmental Analytical Chemistry
Journal of Environmental Monitoring
International Journal of Environmental Research and Public Health
Journal of Experimental Biology
Journal of Fish Biology
Journal of Toxicology and Environmental Health
Journal of Wildlife Diseases
Life Sciences
Polar Biology
The Open Toxicology Journal
Theriogenology
Toxicological Sciences
Toxicological and Environmental Chemistry
Water, Air and Soil Pollution
Water Quality Research Journal of Canada
Wildlife Biology

Manuscript Reviewer, United States Environmental Protection Agency

17.2 GRANT PROPOSAL REVIEWS:

Canada Foundation for Innovation, Leaders Opportunity Fund

Natural Sciences and Engineering Research Council of Canada, Discovery Grant Program

Natural Sciences and Engineering Research Council of Canada, Strategic Grant Program

Natural Sciences and Engineering Research Council of Canada, Collaborative Research and Development Grants Program

Natural Sciences and Engineering Research Council of Canada, E.W.R. Steacie Fellowships Program

National Science Foundation (USA)

Grant Panel Member, Advanced Research Program of the Texas Higher Education Coordinating Board (USA)

17.3 MISCELLANEOUS PEER REVIEW ACTIVITIES:

External reviewer for tenure application package, McMaster University, Hamilton, ON.

External reviewer for internship report, Université de Lyon, Lyon, France.

External reviewer for Canada Research Chair (Tier II) renewal application.

External reviewer for promotion to full professor application package, Simon Fraser University, Burnaby, BC.

External reviewer for tenure application package, University of Missouri, Rolla, MO.

External reviewer for tenure application package, University of Victoria, BC

Book chapter review, *Techniques in Aquatic Toxicology, Volume II*, Ostrander, G.K. (ed.), CRC Press, Boca Raton, FL.

Animal Care and Use protocol review, Fisheries and Oceans Canada, Freshwater Institute, Winnipeg, MB.

18. INVITED LECTURES OUTSIDE U OF S AND INVITED CONFERENCE PRESENTATIONS:

Janz, D.M.. 2010. Selenium and fish: The good, the bad, and the ugly. Invited departmental seminar, Department of Biology, University of Ottawa, Ottawa, ON.

Janz, D.M.. 2010. Ecotoxicology of selenium downstream of metal mines in Canada: When a good nutrient goes bad. Invited departmental seminar, Environmental Resource Studies, Trent University, Peterborough, ON.

Janz, D.M. 2010. Molecules to mountains: Relating long-term stress in Alberta grizzly bears to landscape change. Invited departmental seminar, Department of Biological Sciences, Simon Fraser University, Burnaby, BC.

Janz, D.M. 2010. When a good nutrient goes bad: Ecotoxicology of selenium in fish. Distinguished Lecture Series, Department of Biology and Chemistry, City University of Hong Kong.

Janz, D.M. 2009. Determination of long-term stress markers in skin and hair collected from free-ranging mammals. Invited seminar, United States Office of Naval Research, Arlington, VA.

Janz, D.M., J.M. Muscatello and J.K. Thomas. 2009. Principles and procedures for embryo-larval deformity analyses in fish exposed to selenium. Society of Environmental Toxicology and Chemistry, New Orleans, LA.

- Kelly, J.M. and D.M. Janz. 2008. Ecotoxicological assessment of juvenile northern pike inhabiting lakes downstream of a metal mine. Invited platform presentation for Dr. Richard Playle Award for Best Master's Thesis in Aquatic Toxicology, Aquatic Toxicity Workshop, Saskatoon, SK.
- Janz, D.M. 2007. Integrated responses of fish to environmental estrogens. Invited seminar, Comparative Physiology Invited Speaker Series, Department of Zoology, University of British Columbia, Vancouver, BC.
- Janz, D.M. 2007. Effects of xenoestrogens on gametogenesis and reproduction in fishes. Invited seminar, Department of Biological Sciences, University of Alberta, Edmonton, AB.
- Janz, D.M. 2006. Aquatic ecotoxicology of metal mining in northern Saskatchewan. Invited seminar, Department of Biological Sciences, University of Lethbridge, Lethbridge, AB.
- Janz, D.M., J.R. Muscatello, P.M. Bennett and J.M. Kelly. 2006. Applicability of proposed fish tissue thresholds for selenium to coldwater aquatic ecosystems. Invited seminar, Workshop on the Fate and Effects of Selenium in Aquatic Systems, Bromont, QC.
- Janz, D.M., J.R. Muscatello, P.M. Bennett and J.M. Kelly. 2006. Ecotoxicological effects of selenium bioaccumulation in northern pike (*Esox lucius*). Invited platform presentation, International Congress on the Biology of Fish, St. John's, NL
- Janz, D.M. 2004. Molecular to ecological effects of environmental estrogens in fishes. Invited Departmental seminar, Department of Biology, University of Regina
- Janz, D.M., L.G. Talent, S.M. Brasfield, M.S. Redick and L.P. Weber. 2002. Development of a reptile model for terrestrial wildlife ecotoxicological studies. Invited platform presentation, Society of Environmental Toxicology and Chemistry, Salt Lake City, UT.
- Janz, D.M. 2001. Sex, sewage and fish: The role of apoptosis in reproductive impairment. Invited Departmental seminar, Department of Biological Sciences, Simon Fraser University.
- Janz, D.M. 2001. Sex, sewage and fish: The role of apoptosis in reproductive impairment. Invited Departmental seminar, Faculty of Pharmaceutical Sciences, University of British Columbia.
- Janz, D.M. and L.G. Talent. 2000. Evaluation of fence lizards (*Sceloporus spp.*) as reptile models for assessment of endocrine-mediated toxicities. Invited Plenary Speaker, South Central SETAC Regional Meeting, Denton, TX.
- Janz, D.M. 2000. Hormonal and environmental regulation of ovarian cell apoptosis in fish. Invited Departmental seminar, Biology Department, Southwest Missouri State University, Springfield, MO.

Janz, D.M. 2000. The relevance of toxicant-induced apoptosis to reproductive impairment in fish. Invited Departmental seminar, Department of Biological Sciences, Wichita State University, Wichita, KS.

Janz, D.M. 1999. Hormonal and environmental effects on ovarian follicular cell apoptosis in fish. Invited seminar, College of Osteopathic Medicine, Oklahoma State University, Tulsa, OK.

19. PRESENTATIONS AT CONFERENCES (Non-Invited):

19.1 PLATFORM (ORAL) PRESENTATIONS:

Hauck, D., J. Phibbs, M.M. Goertzen, L.P. Weber and D.M. Janz. 2010. Altered bioenergetics and stress responses in spottail shiner (*Notropis hudsonius*) from a lake receiving metal mining discharges. Society of Environmental Toxicology and Chemistry, Portland, OR. (3rd place, Best Student Platform Award)

Phibbs, J.R., E.D. Franz, D.W. Hauck, C.I.E. Wiramanaden, M. Gallego, I.J. Pickering, K. Liber and D.M. Janz. 2010. Evaluating dietary selenium uptake and speciation in caged lake chub (*Couesius plumbeus*) downstream of a uranium processing mill. Society of Environmental Toxicology and Chemistry, Portland, OR.

Thomas, J.K. and Janz, D.M. 2010. Dietary selenomethionine exposure alters swim performance and energetics of adult zebrafish. International Congress on the Biology of Fish, Barcelona, Spain.

Thomas, J.K. and D.M. Janz. 2010. Behavioural and physiological consequences of dietary selenomethionine exposure to adult zebrafish (*Danio rerio*). Prairie Northern SETAC Regional Meeting, Saskatoon, SK.

Goertzen, M.M., D.W. Hauck, J.R. Phibbs, L.P. Weber and D.M. Janz. 2010. Investigation of swim performance and bioenergetics in spottail shiner (*Notropis hudsonius*) downstream of a uranium mill. Prairie Northern SETAC Regional Meeting, Saskatoon, SK. (2nd place, Best Student Platform Award)

Thomas, J.K. and D.M. Janz. 2010. Behavioural and physiological consequences of dietary selenomethionine exposure to adult zebrafish (*Danio rerio*). Canadian Society of Zoologists, Vancouver, BC.

Goertzen, M.M., D.W. Hauck, J. Phibbs, L.P. Weber and D.M. Janz. 2010. Investigation of swim performance and bioenergetics in spottail shiner (*Notropis hudsonius*) downstream of a uranium mill. Canadian Society of Zoologists, Vancouver, BC.

Phibbs, J.R., E.D. Franz, C.I.E. Wiramanaden, M. Gallego, D.W. Hauck, I.J. Pickering, K. Liber and D.M. Janz. 2009. Evaluating dietary selenium uptake and speciation in lake chub (*Couesius plumbeus*) using feeding cages downstream of a uranium processing mill. Canadian Society of Zoologists, Vancouver, BC.

- Wiramanaden, CIE, A. Anton, J.E. Bird, M. Driessnack, E. Franz, J. Phibbs, R. Pollock, M.G. Dubé, K. Liber, I.J. Pickering and D.M. Janz. 2008. An holistic approach to understanding selenium distribution, concentration, bioavailability and transfer through trophic levels in a northern aquatic environment. Aquatic Toxicity Workshop, Saskatoon, SK.
- Janz, D.M., P.M. Bennett, K. Driedger, J.M. Kelly, L.P. Weber, and J.M. Muscatello. 2008. Testing the winter stress syndrome hypothesis in juvenile fish exposed to metal mining discharges. 8th International Congress on the Biology of Fishes, Portland, OR.
- Carlson, R.I., M.R.L. Cattet, G.B. Stenhouse and D.M. Janz. 2007. Monitoring long-term stress in grizzly bears, a new use of protein microarray technology. Society of Environmental Toxicology and Chemistry, Milwaukee, WI.
- Macbeth, B.J., H. Frimer and D.M. Janz. 2006. Development of non-lethal techniques to determine reproductive indices in wild fish. Society of Environmental Toxicology and Chemistry, Montreal, QC.
- Muscatello, J.R. and D.M. Janz. 2006. Selenium trophic transfer and accumulation downstream of a metal mining and milling area in northern Saskatchewan, Canada. Society of Environmental Toxicology and Chemistry, Montreal, QC.
- Kelly, J.M. and D.M. Janz. 2006. Bioenergetics, biochemistry and histology of juvenile northern pike (*Esox lucius*) downstream of a uranium mill. Society of Environmental Toxicology and Chemistry, Montreal, QC.
- Rickwood, C.J., M.G. Dubé, L.P. Weber, K.L. Driedger and D.M. Janz. 2006. Assessing effects of metal mining effluent on fathead minnow reproduction in a field-based trophic transfer artificial stream system. Aquatic Toxicity Workshop, Jasper, AB.
- Muscatello, J.R., A. Belknap and D.M. Janz. 2006. Selenium accumulation in aquatic organisms downstream of a metal mining and milling area in northern Saskatchewan, Canada. Aquatic Toxicity Workshop, Jasper, AB.
- Kelly, J.M. and D.M. Janz. 2006. Integrated responses of juvenile northern pike (*Esox lucius*) collected downstream of a metal mining effluent. Aquatic Toxicity Workshop, Jasper, AB.
- Muscatello, J.R. and D.M. Janz. 2005. Selenium toxicosis in northern pike collected along a gradient of uranium milling effluent. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Lin, L.L. and D.M. Janz. 2005. Additive and non-additive interactions of binary xenoestrogen mixtures in zebrafish (*Danio rerio*). Society of Environmental Toxicology and Chemistry, Baltimore, MD.

- Rickwood C.J., M.G. Dubé, L.P. Weber, K.L. Driedger and D.M. Janz. 2005. Assessing effects of metal mining effluent on fathead minnow (*Pimephales promelas*) reproduction in a trophic-transfer system. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Redick-Harris, M.S., L.G. Talent and D.M. Janz. 2005. Effects of in ovo exposure to sodium perchlorate on growth, breeding success and hormone levels of eastern fence lizards. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Ogunkoya, A.E., A.G. Van Kessel, D.M. Janz and M.D. Drew. 2005. Effect of replacing fish meal and oil with plant proteins and oils on growth performance, intestinal morphology and organochlorine residues in rainbow trout. AquaNet V, Victoria, BC.
- Dubé, M.G., L.P. Weber, C.J. Rickwood, K.L. Driedger, C.I. Brereton, D.M. Janz and D.L. MacLachy. 2005. Investigation of cause for metal mining environmental effects monitoring (EEM): complexities and considerations. Aquatic Toxicity Workshop, Waterloo, ON.
- Bennett, P.M. and D.M. Janz. 2005. Potential impacts of uranium milling effluent on juvenile fish bioenergetics, growth and overwinter survival. Aquatic Toxicity Workshop, Waterloo, ON.
- Muscatello J.R. and D.M. Janz. 2005. Selenium toxicosis in northern pike exposed to uranium milling effluent. Aquatic Toxicity Workshop, Waterloo, ON.
- Driedger, K.L., L.P. Weber, C.J. Rickwood, C.I. Brereton, M.G. Dube and D.M. Janz. 2005. Effect of complex metal mine effluent on the overwinter survival potential of juvenile fish. Aquatic Toxicity Workshop, Waterloo, ON.
- Cleary, J.S., M.J. Bradford, D.M. Janz and R.M. Peterman. 2005. Examining the use of lipid analysis as an indicator of fish habitat condition in large rivers of central British Columbia. American Fisheries Society, Anchorage, AK.
- Weber, L.P., M.G. Dubé, C.J. Rickwood, K.L. Driedger and D.M. Janz. 2005. Development of investigation of cause approaches for the metal mining industry: Past, present and future. Junction Creek Environmental Effects Monitoring Workshop, Sudbury, ON.
- Redick, M.S., D.M. Janz, T.A. Anderson and L.G. Talent. 2004. Effects of sodium perchlorate on hormone levels and metabolic rates of the western fence lizard. Society of Environmental Toxicology and Chemistry, Austin, TX.
- Redick, M.S., D.M. Janz, T.A. Anderson and L.G. Talent. 2003. Effects of sodium perchlorate on hormone levels and metabolic rates of the western fence lizard. SETAC Regional Meeting, St. Louis, MO.

- Janz, D.M., L.G. Talent, S.M. Brasfield, M.S. Redick and L.P. Weber. 2002. Development of a reptile model for terrestrial wildlife ecotoxicological studies. Society of Environmental Toxicology and Chemistry, Salt Lake City, UT.
- Talent, L.G., D.M. Janz and S.G. Talent. 2002. Evaluation of two species of fence lizards as potential reptile models for ecotoxicological studies. Society of Environmental Toxicology and Chemistry, Salt Lake City, UT.
- Talent, L.G., D.M. Janz and S.G. Talent. 2002. Evaluation of fence lizard eggs as a reptile egg screening assay for endocrine disrupting chemicals. Society of Environmental Toxicology and Chemistry, Salt Lake City, UT.
- Janz, D.M., J.N. Dumont, L.P. Weber, K. Selcer and S.R. Hutchins. 2002. Endocrine modulating effects of confined animal feed operation discharges. Aquatic Toxicity Workshop, Whistler, BC.
- Janz, D.M., L.P. Weber and R.L. Hill, Jr. 2002. Reproductive and histopathological effects of developmental estrogenic exposure in zebrafish. Aquatic Toxicity Workshop, Whistler, BC.
- Brasfield, S.M., L.P. Weber, L.G. Talent and D.M. Janz. 2002. Time course and dose-response relationships for vitellogenin induction in male Western fence lizards (*Sceloporus occidentalis*) exposed to ethinylestradiol. Society of Environmental Toxicology and Chemistry, Vienna, Austria.
(SETAC Europe Best Publication Award on Environmental Research)
- Carlson, R.I., J.A. Wilson, D.P. Rafferty, R.L. Lochmiller, N.T. Basta, J.L. Schroder and D.M. Janz. 2001. Cotton rat detoxification enzyme induction as an indicator of exposure to petroleum waste on land farmed sites. American Society of Mammalogists, Missoula, MT.
- Brasfield, S.M., L.P. Weber, L.G. Talent and D.M. Janz. 2001. Is plasma alkaline-labile phosphate a suitable alternative to measuring vitellogenin? SETAC Regional Meeting, Stillwater, OK.
- Carlson, R.I., J.A. Wilson, D.P. Rafferty, R.L. Lochmiller, N.T. Basta, J.L. Schroder and D.M. Janz. 2001. Cotton rat detoxification enzyme induction as an indicator of exposure to petroleum waste on landfarmed sites. SETAC Regional Meeting, Stillwater, OK.
- Brasfield, S.M., L.P. Weber, L.G. Talent and D.M. Janz. 2001. Development of a reptile model for assessment of endocrine-mediated toxicity. Invited Departmental seminar, Dept of Biology, University of New Brunswick, St. John, NB.
- Talent, L.G., D.M. Janz, J.A. Bantle and J.N. Dumont. 2000. Evaluation of fence lizards, *Sceloporus spp.*, as reptile models for assessment of endocrine-mediated toxicity. Society of Environmental Toxicology and Chemistry, Nashville, TN.

- McMaster, M.E., M. Hewitt, J. Parrott, J. Sherry, M. Koli, G. Van Der Kraak, K. Oakes, D.M. Janz, C.B. Portt and K.R. Munkittrick. 2000. Detailed endocrine assessments in wild fish downstream of pulp and paper mills: Application to the Northern Rivers Ecosystem Initiative endocrine program in Northern Alberta, Canada. In Proceedings of the 4th International Conference on Environmental Impacts of the Pulp and Paper Industry. Eds. M. Rouppa, J. Paasivirta, K.J. Lehtinen and S. Ruonala. Finnish Environmental Institute 417:232-237.
- Johnson, J.L. and D.M. Janz. 2000. Tissue-specific expression of HSP70 in two fish species inhabiting a metal contaminated creek. SETAC Regional Meeting, Leavenworth, KS. **(Best Student Platform Award)**
- Weber, L.P., S.L. Diamond and D.M. Janz. 1999. Effect of β -naphthoflavone and dimethylbenz[a]anthracene on ovarian cell apoptosis in juvenile channel catfish (*Ictalurus punctatus*). Society of Environmental Toxicology and Chemistry, Philadelphia, PA.
- McMaster, M.E., L.M. Hewitt, G. Van Der Kraak, K. Oakes, D.M. Janz, C. Portt, and K.R. Munkittrick. 1999. Detailed endocrine assessments in wild fish downstream of pulp and paper mills: application to the Northern Rivers Ecosystem Initiative Endocrine Program. Aquatic Toxicity Workshop, Edmonton, AB.
- Van Der Kraak, G., E.J. Squires, K. Wells, K. Oakes, A. Lister, L. Tremblay, D.M. Janz, T. Zacharewski, M.E. McMaster, M. Servos, M. Hewitt and K.R. Munkittrick. 1999. Developing methods to assess reproductive endocrine function in fish. Canadian Network of Toxicology Centres Annual Research Symposium, Hull, QC.
- Janz, D.M., M.E. McMaster, K.R. Munkittrick and G. Van Der Kraak. 1998. Elevated ovarian follicular cell apoptosis and stress protein expression in fish exposed to pulp mill effluents. Society of Environmental Toxicology and Chemistry, Charlotte, NC.
- Van Der Kraak, G., L. Tremblay, K. Wells, D.M. Janz, T. Zacharewski, E.J. Squires, M.E. McMaster, M. Servos, M. Hewitt and K.R. Munkittrick. 1998. Validation of reproductive endocrine biomarkers across different levels of biological complexity. Canadian Network of Toxicology Centres Annual Research Symposium, Toronto, ON.
- Janz, D.M., M.E. McMaster, K.R. Munkittrick and G. Van Der Kraak. 1997. Apoptosis and induction of stress proteins in ovarian follicles from white sucker exposed to pulp mill effluents. International Conference on Environmental Fate and Effects of Bleached Pulp Mill Effluents, Rotorua, NZ.
- Van Der Kraak, G., D.M. Janz, M.E. McMaster and K.R. Munkittrick. 1997. Pulp mill effluent induces cell suicide in fish ovarian follicles. Aquatic Toxicity Workshop, Niagara, ON.

Janz, D.M., M.E. McMaster, K.R. Munkittrick and G. Van Der Kraak. 1997. Elevated apoptosis and stress protein levels in ovarian follicles from white sucker exposed to pulp mill effluents. Great Plains Limnology Conference, Stillwater, OK.

Janz, D.M. and C.D. Metcalfe. 1990. Relative induction of aryl hydrocarbon hydroxylase by 2,3,7,8-TCDD and two coplanar PCBs in rainbow trout (*Oncorhynchus mykiss*). Society of Environmental Toxicology and Chemistry, Arlington, VA.

Janz, D.M. and C.D. Metcalfe. 1990. Relative induction of aryl hydrocarbon hydroxylase by 2,3,7,8-TCDD and two coplanar PCBs in rainbow trout (*Oncorhynchus mykiss*). Aquatic Toxicity Workshop, Vancouver, BC.

19.2 POSTER PRESENTATIONS:

Thomas, J.K. and D.M. Janz. 2011. Comparative toxicities of maternally deposited and microinjected selenomethionine to larval and juvenile zebrafish (*Danio rerio*). Prairie Northern SETAC Regional Meeting, Winnipeg, MB.

Thomas, J.K. and D.M. Janz. 2011. Comparative toxicities of maternally deposited and microinjected selenomethionine to larval and juvenile zebrafish (*Danio rerio*). Prairie University Biology Symposium, Saskatoon, SK.

Thomas, J.K. and D.M. Janz. 2011. Comparative toxicities of maternally deposited and microinjected selenomethionine to larval and juvenile zebrafish (*Danio rerio*). University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.

Goertzen, M.M., M. Driessnack, J. Phibbs, M.G. Dube, L.P. Weber and D.M. Janz. 2010. Effects of uranium mill effluent exposure on swimming performance and intermediary metabolism in small-bodied fish. Society of Environmental Toxicology and Chemistry, Portland, OR.

Thomas, J.K. and D.M. Janz. 2011. Comparative toxicities of maternally deposited and microinjected selenomethionine to larval and juvenile zebrafish (*Danio rerio*). Society of Environmental Toxicology and Chemistry, Portland, OR.

Weber, L.P., Thomas, J.K., Marit, J.M., Janz, D.M. 2010. Oxygen consumption and swimming in sub-adult versus adult zebrafish. FitFish Workshop on the Swimming Physiology of Fish, Barcelona, Spain.

Macbeth, B.J., M.R.L. Cattet, G.B. Stenhouse, M.L. Gibeau and D.M. Janz. 2010. Validation and application of hair cortisol concentration as a potential non-invasive biomarker of long-term stress in free ranging grizzly bears. Canadian Society of Zoologists, Vancouver, BC.

- Thomas, J.K. and D.M. Janz. Consequences of dietary and *in ovo* selenium exposure in zebrafish. Society of Environmental Toxicology and Chemistry, New Orleans, LA.
- Goertzen, M.M., M. Driessnack, J. Phibbs, M.G. Dube, L.P. Weber and D.M. Janz. 2009. Swim performance and bioenergetic effects of metal milling effluent exposure in two native fish species. Society of Environmental Toxicology and Chemistry, New Orleans, LA.
- Phibbs, J.R., E.D. Franz, C.I.E. Wiramanaden, D.W. Hauck, K. Liber, I.J. Pickering and D.M. Janz. 2009. Use of feeding cages to evaluate dietary selenium uptake and speciation in lake chub (*Couesius plumbeus*). Society of Environmental Toxicology and Chemistry, New Orleans, LA.
- Franz, E.D., C.I.E. Wiramanaden, I.J. Pickering, D.M. Janz and K. Liber. 2009. Selenium bioaccumulation and speciation in *Chironomus dilutus* exposed to organic and inorganic forms of selenium. Society of Environmental Toxicology and Chemistry, New Orleans, LA.
- Franz, E.D., C.I.E. Wiramanaden, I.J. Pickering, D.M. Janz and K. Liber. 2009. Bioaccumulation of selenium by *Chironomus dilutus*: comparing sediment versus surface water exposure routes using an *in situ* method. Society of Environmental Toxicology and Chemistry, New Orleans, LA.
- Thomas, J.K., K. Smith and D.M. Janz. 2009. Effects of dietary, aqueous and *in ovo* selenium exposure in zebrafish. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK. (**Best Poster Award, Environmental Sciences**)
- Macbeth, B.J., M.R.L. Cattet, G.B. Stenhouse and D.M. Janz. 2009. Hair cortisol concentration: A potential non-invasive biomarker of long-term stress in free ranging grizzly bears. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK. (**Best Poster Award, Animal Sciences**)
- Thomas, J.K., K. Smith and D.M. Janz. 2008. Physiological consequences of selenomethionine-induced larval deformities in zebrafish. Society of Environmental Toxicology and Chemistry, Tampa, FL.
- Tompsett, A.R., D. Vardy, M. Hecker, M. Adzic, M. Allan, J.H. Smith, X. Zhang, K. Liber, D.M. Janz and J.P. Giesy. 2008. White sturgeon hatch and survival after exposure to Columbia River surface water at two sites in British Columbia, Canada. Society of Environmental Toxicology and Chemistry, Tampa, FL.
- Vardy, D., A.R. Tompsett, M. Hecker, J. Duquette, D.M. Janz, K. Liber, M. Adzic and J.P. Giesy. 2008. Effects of selected metals on early white sturgeon (*Acipenser transmontanus*) life stages. Society of Environmental Toxicology and Chemistry, Tampa, FL.

- Muscatello, J.R., C. Levesque and D.M. Janz. 2008. Movement patterns of northern pike (*Esox lucius*) within gradients of uranium mining effluent discharges. Aquatic Toxicity Workshop, Saskatoon, SK.
- Franz, ED, C.I.E. Wiramanaden, I.J. Pickering, D.M. Janz and K. Liber. 2008. Uptake and depuration of environmentally relevant selenium species in *Chironomus tentans*. Aquatic Toxicity Workshop, Saskatoon, SK.
- Phibbs, J, E. Franz, C. Wiramanaden, D. Hauck, K. Liber and DM Janz. 2008. Evaluating selenium uptake and toxicity in small bodied fish downstream of a uranium milling operation. Aquatic Toxicity Workshop, Saskatoon, SK.
- Thomas, J.K., K. Smith and D.M. Janz. 2008. Effects of dietary, aqueous and *in ovo* selenium exposure in zebrafish. Aquatic Toxicity Workshop, Saskatoon, SK.
- Vardy, D., A.R. Tompsett, M. Hecker, J. Duquette, D.M. Janz, K. Liber, M. Adzic and J.P. Giesy. 2008. Sensitivity of early white sturgeon (*Acipenser transmontanus*) life stages to copper, cadmium and zinc. Aquatic Toxicity Workshop, Saskatoon, SK.
- Macbeth, B.J., M.R.L. Cattet, G.B. Stenhouse and D.M. Janz. 2008. Hair cortisol concentration: A potential non-invasive biomarker of long-term stress in free ranging grizzly bears. Wildlife Disease Association, Edmonton, AB.
- Muscatello, J.M. and D.M. Janz. 2008. Selenium bioaccumulation and effects in native fish species. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.
- Muscatello, J.M., A. Belknap and D.M. Janz. 2007. Selenium accumulation and effects in aquatic organisms downstream of a uranium mining and milling area in northern Saskatchewan, Canada. Society of Environmental Toxicology and Chemistry, Milwaukee, WI.
- Muscatello, J.M. and D.M. Janz. 2007. Deformity evaluation in northern pike and white sucker exposed to uranium mining and milling effluent. Society of Environmental Toxicology and Chemistry, Milwaukee, WI.
- Muscatello, J.R. and D.M. Janz. 2007. Selenium toxicosis in northern pike exposed to metal mining and milling effluent. Aquatic Toxicity Workshop, Halifax, NS.
- Muscatello, J.M., A. Belknap and D.M. Janz. 2007. Selenium accumulation in aquatic organisms downstream of a metal mining and milling area in northern Saskatchewan, Canada. Aquatic Toxicity Workshop, Halifax, NS.
- Kelly, J.M. and D.M. Janz. 2007. Integrated responses of juvenile northern pike (*Esox lucius*) downstream of a metal mining effluent. Aquatic Toxicity Workshop, Halifax, NS.

- Carlson, R.I., M.R.L. Cattet, G.B. Stenhouse and D.M. Janz. 2007. Development of a protein microarray to detect long-term stress in grizzly bears. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.
- Muscatello, J.R. and D.M. Janz. 2007. Selenium toxicosis in northern pike exposed to metal mining and milling effluent. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.
- Lux, S.E., Rickwood C.J., Dubé, M.G., Janz, D.M. and L.P. Weber. 2006. Larval deformities and energetics in fathead minnow exposed to metal mining and sewage effluent. Society of Environmental Toxicology and Chemistry, Montreal, QC.
- Heather, B.R. and D.M. Janz. 2006. Effects of benzo[a]pyrene on gonadal cell death in zebrafish. Society of Environmental Toxicology and Chemistry, Montreal, QC.
- Muscatello, J.R. and D.M. Janz. 2006. Selenium toxicosis in northern pike exposed to metal mining and milling effluent. Society of Environmental Toxicology and Chemistry, Montreal, QC.
- Carlson, R.I., M.R.L. Cattet, G.B. Stenhouse and D.M. Janz. 2006. Development of a protein microarray to detect long-term stress in grizzly bears. The Wildlife Society Annual Meeting, Anchorage, AK.
- Muscatello, J.R. and D.M. Janz. 2006. Selenium toxicosis in northern pike exposed to metal mining and milling effluent. Aquatic Toxicity Workshop, Jasper, AB.
- Frimer, H. and D.M. Janz. 2006. Development of non-lethal, ultrasonographic techniques to estimate reproductive indices in white sucker (*Catostomus commersoni*). Merck-Merial Veterinary Scholar National Symposium, Baton Rouge, LA.
- Muscatello, J.R. and D.M. Janz. 2006. Selenium toxicosis in northern pike collected along a gradient of uranium milling effluent. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK. **(2nd Place, Poster Competition)**
- Bennett, P.M., L.P. Weber and D.M. Janz. 2006. Assessing potential impacts of two uranium milling effluents on juvenile northern pike bioenergetics, growth and overwinter survival. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.
- Heather, B.R., S. Lux, L.P. Weber and D.M. Janz. 2006. Stress responses of juvenile zebrafish exposed to β -naphthoflavone. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.
- Kelly, J.M. and D.M. Janz. 2006. Assessment of various indicators of health in juvenile northern pike (*Esox lucius*) downstream of a uranium mill. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.

- Carlson, R.I., M.R.L. Cattet, G.B. Stenhouse and D.M. Janz. 2006. Monitoring long-term stress in grizzly bears using protein microarray technology. World Microarray Congress, Vancouver, BC.
- Ogunkoya, A.E., A.G. Van Kessel, D.M. Janz and M.D. Drew. 2006. Replacement of fish meal and oil by canola protein concentrate and vegetable oils in diets fed to rainbow trout (*Oncorhynchus mykiss*). XII International Symposium on Fish Nutrition and Feeding, Biarritz, France.
- Bennett, P.M., L.P. Weber and D.M. Janz. 2005. Assessing potential impacts of two uranium milling effluents on juvenile northern pike bioenergetics, growth and overwinter survival. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Heather, B.R., S. Lux, L.P. Weber and D.M. Janz. 2005. Stress responses of juvenile zebrafish exposed to β -naphthoflavone. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Driedger, K.L., L.P. Weber, C.J. Rickwood, C.I. Brereton, M.G. Dube and D.M. Janz. 2005. Potential effect of complex metal mine effluent on the overwinter survival potential of juvenile fish. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Kelly, J.M. and D.M. Janz. 2005. Assessment of various indicators of health in juvenile northern pike (*Esox lucius*) downstream of a uranium mill. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Weber, L.P., M.G. Dubé, C.J. Rickwood, K.L. Driedger, C. Portt, C.I. Brereton and D.M. Janz. 2005. Effects of multiple mining and municipal effluents on fish from Junction Creek, Sudbury, Ontario. 1. Basic Environmental Effects Monitoring (EEM) end-points. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Weber, L.P., M.G. Dubé, C.J. Rickwood, K.L. Driedger, C. Portt, C.I. Brereton and D.M. Janz. 2005. Effects of multiple mining and municipal effluents on fish from Junction Creek, Sudbury, Ontario. 2. Sub-organismal responses. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Redick-Harris, M.S., L.G. Talent and D.M. Janz. 2005. Effects of in ovo exposure to sodium perchlorate on histology and hormone levels of hatchling and mature male western fence lizards. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Lin, L.L. and D.M. Janz. 2005. Additive and non-additive interactions of binary xenoestrogen mixtures in zebrafish (*Danio rerio*). Aquatic Toxicity Workshop, Waterloo, ON. (**Runner-up, Best Student Poster Award**)
- Macbeth, B.J. and D.M. Janz. 2005. Development of a non-destructive technique to

assess ovary size and fecundity in wild fish. Aquatic Toxicity Workshop, Waterloo, ON.

Weber, L.P., M.G. Dubé, C.J. Rickwood, K.L. Driedger, C. Portt, C.I. Brereton and D.M. Janz. 2005. Effects of multiple mining and municipal effluents on fish from Junction Creek, Sudbury, Ontario. 1. Basic Environmental Effects Monitoring (EEM) end-points. Aquatic Toxicity Workshop, Waterloo, ON.

Weber, L.P., M.G. Dubé, C.J. Rickwood, K.L. Driedger, C. Portt, C.I. Brereton and D.M. Janz. 2005. Effects of multiple mining and municipal effluents on fish from Junction Creek, Sudbury, Ontario. 2. Sub-organismal responses. Aquatic Toxicity Workshop, Waterloo, ON.

Macbeth, B.J. and D.M. Janz. 2005. Development of a non-destructive technique to assess ovary size and fecundity in wild fish. Merck-Merial Veterinary Scholar National Symposium, Baton Rouge, LA.

Bennett, P.M. and D.M. Janz. 2005. Assessing potential impacts of uranium milling effluent on juvenile fish bioenergetics, growth and over winter survival. Prairie University Biological Symposium, Saskatoon, SK.

Driedger, K.L., B.R. Heather, L.P. Weber and D.M. Janz. 2005. Effects of aryl hydrocarbon receptor activation on growth and condition factors, protein concentration and HSP70 expression in juvenile zebrafish. Prairie University Biological Symposium, Saskatoon, SK.

Lin, L.L. and D.M. Janz. 2005. Effects of binary mixtures of xenoestrogens in zebrafish (*Danio rerio*). Prairie University Biological Symposium, Saskatoon, SK.

Bennett, P.M. and D.M. Janz. 2005. Assessing potential impacts of uranium milling effluent on juvenile fish bioenergetics, growth and overwinter survival. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.

Driedger, K.L., B.R. Heather, L.P. Weber and D.M. Janz. 2005. Effects of aryl hydrocarbon receptor activation on growth and condition factors, protein concentration and HSP70 expression in juvenile zebrafish. University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.

Lin, L.L. and D.M. Janz. 2005. Effects of binary mixtures of xenoestrogens in zebrafish (*Danio rerio*). University of Saskatchewan Life and Health Sciences Research Conference, Saskatoon, SK.

Talent, L.G., N.L. Cooper, M.S. Redick and D.M. Janz. 2004. Window of sensitivity when the phenotypic sex of male fence lizards can be altered. Society of Environmental Toxicology and Chemistry, Portland, OR.

- Bennett, P.M. and D.M. Janz. 2004. Assessing potential impacts of uranium milling effluent on juvenile fish bioenergetics, growth and overwinter survival. Society of Environmental Toxicology and Chemistry, Portland, OR.
- Muscattello J. and D.M. Janz. 2004. Assessment of selenium toxicosis in northern pike and white sucker larvae exposed to uranium milling effluents. Society of Environmental Toxicology and Chemistry, Portland, OR.
- Driedger, K.L., B.R. Heather, L.P. Weber and D.M. Janz. 2004. Effects of aryl hydrocarbon receptor activation on growth and condition factors, protein concentration and HSP70 expression in juvenile zebrafish. Society of Environmental Toxicology and Chemistry, Portland, OR.
- Lin, L.L. and D.M. Janz. 2004. Effects of binary mixtures of xenoestrogens in zebrafish (*Danio rerio*). Society of Environmental Toxicology and Chemistry, Portland, OR.
- Redick, M.S., D.M. Janz and L.G. Talent. 2004. Effects of long term in ovo exposure to sodium perchlorate on incubation and growth of sceloporine lizards. Society of Environmental Toxicology and Chemistry, Portland, OR.
- Cleary, J.S., M.J. Bradford, D.M. Janz and R.M. Peterman. 2004. Lipid analysis in juvenile salmon: Examining the use of lipid analysis as an indicator of fish habitat condition in large rivers of central British Columbia. Canadian Conference for Fisheries Research, St. John's, NL.
- Liber, K., S. Stoughton, D.M. Janz and T. Moulding. 2004. How important is onset of toxicant exposure in early life-stage uranium toxicity tests with fish? Society of Environmental Toxicology and Chemistry, Prague, CZ.
- Driedger, K., L.P. Weber and D.M. Janz. 2004. Effects of aryl hydrocarbon receptor activation on growth and HSP70 expression in juvenile zebrafish. University of Saskatchewan Annual Life Sciences Conference, Saskatoon, SK,
- Liber, K., S. Stoughton, D.M. Janz and T. Moulding. 2003. Toxicity of uranium to early life stage lake trout (*Salvelinus namaycush*). Society of Environmental Toxicology and Chemistry, Hamburg, DL.
- Weber, L.P., G.C. Balch, C.D. Metcalfe and D.M. Janz. 2002. Increased kidney and testicular cell death after developmental exposure to 17 α -ethinylestradiol in medaka (*Oryzias latipes*). Society of Environmental Toxicology and Chemistry, Salt Lake City, UT.
- Redick, M.S., D.M. Janz and L.G. Talent. 2002. Effects of perchlorate on perinatal thyroid hormone levels and hatchability of the western fence lizard. Society of Environmental Toxicology and Chemistry, Salt Lake City, UT.
- Porter, C.M. and D.M. Janz. 2001. Treated municipal sewage discharge affects

multiple levels of biological organization in fishes. Society of Environmental Toxicology and Chemistry, Baltimore, MD.

- Brasfield, S.M., K. Bradham, J.B. Wells, L.G. Talent, R.P. Lanno and D.M. Janz. 2001. Evaluation of fence lizard eggs as a terrestrial vertebrate model for assessing bioavailability and *in ovo* effects of soil contaminants. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Talent, L.G., J.N. Dumont, J.A. Bantle, D.M. Janz and S.G. Talent. 2001. Evaluation of fence lizard eggs as a reptile model for testing endocrine disrupting chemicals. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Weber, L.P., N. Abdel-Hamide, R.L. Hill and D.M. Janz. 2001. Developmental estrogenic exposure alters gametogenesis and produces organ toxicity in zebrafish. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Weber, L.P., J.N. Dumont, K.W. Selcer, S.R. Hutchins and D.M. Janz. 2001. Endocrine modulating effects of lagoon water from confined animal feed operations on amphibians. Society of Environmental Toxicology and Chemistry, Baltimore, MD.
- Porter, C.M. and D.M. Janz. 2001. Treated municipal sewage discharge affects multiple levels of biological organization in fishes. SETAC Regional Meeting, Stillwater, OK. **(Best Student Poster Award)**
- Carlson, R.I., J.A. Wilson, D.P. Rafferty, R.L. Lochmiller and D.M. Janz. 2000. Liver detoxification enzyme induction in cotton rats residing on petroleum waste sites. Society of Environmental Toxicology and Chemistry, Nashville, TN.
- Brasfield, S.M., L.P. Weber, L.G. Talent and D.M. Janz. 2000. Time course and dose-response relationships for vitellogenin induction in male Western fence lizards (*Sceloporus occidentalis*) exposed to ethynylestradiol. Society of Environmental Toxicology and Chemistry, Nashville, TN.
- Johnson, J.L. and D.M. Janz. 2000. Tissue-specific expression of HSP70 in two fish species inhabiting a metal contaminated creek. Society of Environmental Toxicology and Chemistry, Nashville, TN.
- Hill, R.L. Jr. and D.M. Janz. 2000. Effects of ethynylestradiol and nonylphenol on sexual development and reproduction in zebrafish (*Danio rerio*). Society of Environmental Toxicology and Chemistry, Nashville, TN.
- Savabieasfahani, M. and D.M. Janz. 2000. Effects of apoptogenic and survival factors on ovarian cell apoptosis in cultured fathead minnow (*Pimephales promelas*) follicles. Society of Environmental Toxicology and Chemistry, Nashville, TN.

- Weber, L.P., Y. Kiparissis, G.S. Hwang, A.J. Niimi, D.M. Janz and C.D. Metcalfe. 2000. Increased cellular apoptosis after chronic aqueous exposure to nonylphenol and quercetin in adult medaka (*Oryzias latipes*). Society of Environmental Toxicology and Chemistry, Nashville, TN.
- Carlson, R.I., J.A. Wilson, D.P. Rafferty, R.L. Lochmiller and D.M. Janz. 2000. Induction of liver detoxification enzymes in cotton rats residing on petroleum waste sites. The Wildlife Society, Nashville, TN.
- Brasfield, S.M., L.P. Weber, L.G. Talent and D.M. Janz. 2000. Time course and dose-response relationships for vitellogenin induction in male Western fence lizards (*Sceloporus occidentalis*) exposed to ethinylestradiol. SETAC Regional Meeting, Leavenworth, KS.
- Carlson, R.I., J.A. Wilson, D.P. Rafferty, R.L. Lochmiller and D.M. Janz. 2000. Liver detoxification enzyme induction in cotton rats residing on petroleum waste sites. SETAC Regional Meeting, Leavenworth, KS.
- Savabieasfahani, M. and D.M. Janz. 2000. Effects of apoptogenic and survival factors on ovarian cell apoptosis in cultured fathead minnow (*Pimephales promelas*) follicles. SETAC Regional Meeting, Leavenworth, KS. (**Best Student Poster Award**)
- Savabieasfahani, M., R.L. Lochmiller and D.M. Janz. 1999. Elevated ovarian and thymic cell apoptosis in wild cotton rats inhabiting petrochemical-contaminated terrestrial ecosystems. Society of Environmental Toxicology and Chemistry, Philadelphia, PA.
- Savabieasfahani, M., R.L. Lochmiller and D.M. Janz. 1999. Elevated ovarian and thymic cell apoptosis in wild cotton rats inhabiting petrochemical-contaminated terrestrial ecosystems. SETAC Regional Meeting, Carbondale, IL. (**Best Student Poster Award**)
- Diamond, S.L. and D.M. Janz. 1999. Induction of hepatic EROD activity and HSP70 expression in channel catfish exposed to β -naphthoflavone or 7,12-dimethylbenz[α]anthracene. Oklahoma Partners for Biological Sciences Symposium, Stillwater, OK.
- Janz, D.M., M.E. McMaster, K.R. Munkittrick and G. Van Der Kraak. 1998. Elevated ovarian follicular cell apoptosis and stress protein expression in fish exposed to pulp mill effluents. Watkins Life Science Conference, Wichita, KS. (**Young Investigator Award**)
- Van Der Kraak, G., T. MacDougall, D.M. Janz and R.K. Srivastava. 1997. Epidermal growth factor: A multifactorial regulator of ovarian function in teleosts. IUBS Symposium on Advances in the Molecular Endocrinology of Fish, Toronto,

ON.

- Janz, D.M., and G. Van Der Kraak. 1997. Hormonal regulation of apoptosis in teleost ovarian follicles. IUBS Symposium on Advances in the Molecular Endocrinology of Fish, Toronto, ON.
- Janz, D.M. and G. Van Der Kraak. 1996. Developmental and hormonal regulation of apoptosis in teleost ovarian follicles. Canadian Network of Toxicology Centres Symposium, Toronto, ON.
- Janz, D.M., M.E. McMaster, K.R. Munkittrick and G. Van Der Kraak. 1997. Elevated ovarian follicular apoptosis in white sucker exposed to bleached kraft pulp mill effluent. Society of Toxicology, Cincinnati, OH.
- Janz, D.M., M.E. McMaster, K.R. Munkittrick and G. Van Der Kraak. 1996. Hormonal and environmental effects on ovarian follicular apoptosis in fish. Society of Environmental Toxicology and Chemistry, Washington, DC.
- Janz, D.M. and G. Van Der Kraak. 1996. Developmental and hormonal regulation of apoptosis in teleost ovarian follicles. International Symposium on Cell Death in Reproductive Physiology, Chicago, IL.
- Janz, D.M. and G.D. Bellward. 1995. Hepatic estrogen receptor and plasma 17 β -estradiol concentrations as biological markers of 2,3,7,8-TCDD exposure in avian hatchlings. Society of Environmental Toxicology and Chemistry, Vancouver, BC.
- Janz, D.M. and G.D. Bellward. 1994. Effects of early *in ovo* 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) exposure on hepatic ethoxyresorufin O-deethylase (EROD) induction and growth-related parameters in domestic and wild avian hatchlings. Society of Toxicology of Canada Symposium, Montreal, QC.
- Janz, D.M. and G.D. Bellward. 1994. Effects of early *in ovo* 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) exposure on hepatic ethoxyresorufin O-deethylase (EROD) induction and growth-related parameters in domestic and wild avian hatchlings. International Conference on Environmental Fate and Effects of Bleached Pulp Mill Effluents, Vancouver, BC.
- Janz, D.M. and G.D. Bellward. 1994. Effects of early *in ovo* 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) exposure on hepatic ethoxyresorufin O-deethylase (EROD) induction and growth-related parameters in domestic and wild avian hatchlings. Society of Environmental Toxicology and Chemistry, Denver, CO.
- Janz, D.M. and G.D. Bellward. 1994. Effects of *in ovo* 2,3,7,8-TCDD exposure on hepatic ethoxyresorufin O-deethylase (EROD) induction and growth-related parameters in avian embryos and hatchlings. International Symposium on

Microsomes and Drug Oxidations, Toronto, ON.

Metcalf, C.D., D.M. Janz, G.E. Harris, and Y. Kiparissis. 1993. Toxic equivalency factors for coplanar PCBs in fish. Society of Environmental Toxicology and Chemistry, Houston, TX.

Janz, D.M. and C.D. Metcalfe. 1991. Nonadditive interactions of mixtures of 2,3,7,8 TCDD and 3,3',4,4'-tetrachlorobiphenyl on aryl hydrocarbon hydroxylase induction in rainbow trout (*Oncorhynchus mykiss*). Society of Environmental Toxicology and Chemistry, Seattle, WA.

20. PATENTS GRANTED OR PENDING:

United States Patent Application No. 61/088,390: Antibody-Based Protein Microarray to Detect Long Term Stress in Animal Tissues. Patent pending, submitted August 2009.

21. RESEARCH GRANT INFORMATION:

Period	Role	Title (Co-PIs)	Funding Source	Annual (Janz award)	Total (Janz award)
2011-2014	Co-PI	Variability of Hormonal Stress Markers and Stress Responses in a Large Cross-Sectional Sample of Elephant Seals (D.E. Crocker (PI), D.S. Houser, N. Kellar, J. Cockrem)	United States Department of Defence, Office of Naval Research	\$118,610 (\$17,250)	\$355,829 (\$61,706)
2011-2014	Co-PI	Pathophysiology of Stress in Wild and Managed-Care Bottlenose Dolphins (P.A. Fair (PI), G.D. Bossart, J. Reif, T. Romano, A. Dove, M. Peden-Adams)	United States Department of Defence, Office of Naval Research	\$116,042 (\$6,900)	\$348,126 (\$13,800)
2011-2015	Co-PI	Exotic Chemical Contaminants in the South Saskatchewan River (M. Waiser, A. Cessna, P. Jones, M. Hecker, G. Puta, J. Kells, S. Siciliano, J.P. Giesy)	CERC	\$147,500 (\$10,000)	\$590,000 (\$40,000)
2010-2015	PI	Cellular Mechanisms and Ecophysiological Consequences of Selenium Toxicity in Fish	NSERC, Discovery Grant Program	\$42,000	\$210,000
2008-2011	PI	Non-Invasive Stress Monitoring in Wild Mammals Using Hair Cortisol Concentration (Co-PI: M.R.L. Cattet)	Morris Animal Foundation	41,046	\$82,091
2008-2011	PI	Investigation of Temporal and Spatial Distribution, Fate and	NSERC, Collaborative	\$125,630 (\$31,408)	\$376,890 (\$94,223)

		Biological Effects of Selenium in a Boreal Aquatic Ecosystem (Co-PIs: K. Liber, M. Dube, I. Pickering)	Research and Development Grant Program		
2008-2011	PI	Investigation of Temporal and Spatial Distribution, Fate and Biological Effects of Selenium in a Boreal Aquatic Ecosystem (Co-PIs: K. Liber, M. Dube, I. Pickering)	Cameco Corp.	\$143,549 (\$35,887)	\$430,648 (\$107,662)
2006-2011	Co-PI	Understanding and Tracking Landscape Change and Animal Health for Effective Management and Conservation of Species at Risk (S. Franklin (PI), G. Stenhouse, M. Cattet, M. Vijayan)	NSERC, Collaborative Research and Development Grant Program	\$141,208 (\$15,038)	\$706,041 (\$75,190)
2005-2011	Co-PI	Understanding and Tracking Landscape Change and Animal Health for Effective Management and Conservation of Species at Risk (S. Franklin (PI), G. Stenhouse, M. Cattet, M. Vijayan)	Foothills Model Forest	\$108,117 (\$71,544)	\$540,584 (\$357,719)
2004-2010	PI	Endogenous and Exogenous Factors Influencing Gonadal Cell Death and Development in Fish	NSERC, Discovery Grant Program	\$36,533	\$182,665
2007-2008	Co-PI	Toxicological Assessment of White Sturgeon (<i>Acipenser transmontana</i>) Early Life Stages Exposed to Liquid Effluents of the Teckcominco Metals Ltd. Trail Facility (J. Giesy (PI), M. Hecker, K Liber)	Teckcominco Ltd.	\$337,590 (\$15,000)	\$337,590 (\$15,000)
2007-2008	Co-PI	Hair Cortisol Concentration: A Potential Biomarker of Long-Term Stress in Grizzly Bears (Co-PI: M. Cattet)	World Wildlife Fund, Endangered Species Recovery Program	\$5,500	\$5,500
2006	Co-PI	Aquatic Toxicology Research Facility. Major infrastructure grant (K. Liber (PI), J.P. Geisy, M.G. Dubé, P. Jones, P. Krone, K. Solomon, D. Muir, C. Janssen)	New Initiatives Fund, Canada Foundation for Innovation	N/A	\$7,015,000 total; CFI contribution \$2,806,000
2006	Co-PI	Aquatic Toxicology Research Facility. Major infrastructure	Western Economic Diversification	N/A	\$465,000

		grant (K. Liber (PI), J.P. Geisy, M.G. Dubé, P. Jones, P. Krone, K. Solomon, D. Muir, C. Janssen)			
2006	Co-PI	Aquatic Toxicology Research Facility. Major infrastructure grant (K. Liber (PI), J.P. Geisy, M.G. Dubé, P. Jones, P. Krone, K. Solomon, D. Muir, C. Janssen)	Areva Resources Canada, Inc. and Cameco Corporation	N/A	\$250,000
2006-2007	Co-PI	New Tools to Map, Understand, and Track Landscape Change and Animal Health for Effective Management and Conservation of Species at Risk (G. Stenhouse (PI), S. Franklin, M. Cattet, M. Vijayan)	Alberta Innovation and Science	\$973,000 (\$123,500)	\$1,946,000 (\$247,000)
2005-2006	PI	Development of a Protein Microarray to Determine Chronic Physiological Stress in Grizzly Bears	Foothills Model Forest	\$25,000	\$25,000
2005-2006	PI	Development of a Proteomics Technique to Investigate Chronic Physiological Stress in Grizzly Bears and Polar Bears	WCVM Research Trust Fund	\$4,800	\$4,800
2005-2006	PI	Development of a Stress-Activated Protein Microarray to Investigate Chronic Physiological Stress in Grizzly Bears and Polar Bears	WCVM Wildlife Health Fund	\$4,800	\$4,800
2004-2006	PI	Establishment of Contaminant-specific, Site-independent Methods for Assessing Potential Adverse Effects of Uranium Mining Operations on Fish Populations	NSERC, Collaborative Research and Development Grant Program	\$55,171	\$165,514
2003-2006	PI	Establishment of Contaminant-specific, Site-independent Methods for Assessing Potential Adverse Effects of Uranium Mining Operations on Fish Populations	Uranium Producers of Saskatchewan (Cameco Corporation and Areva Resources Canada Inc.)	\$50,667	\$152,000
2004-2007	Co-PI	Development of Investigation of Cause Approaches for the Metal Mining Industry (M.G. Dubé)	NSERC, Collaborative Research and Development Grant Program	\$41,746	\$125,238

2004-2007	Co-PI	Development of Investigation of Cause Approaches for the Metal Mining Industry (M.G. Dubé)	INCO Ltd.	\$49,736	\$149,209
2004-2007	Co-PI	Development of Investigation of Cause Approaches for the Metal Mining Industry (M.G. Dubé)	Environment Canada	\$11,433	\$34,300
2004-2006	Co-PI	Canola Protein Concentrate: Optimization of Feed Formulation and Effects on Intestinal Physiology and Carcass Quality of Salmonid Fish (M.D. Drew, A.G. Van Kessel)	AquaNet	\$46,000	\$92,000
2003	PI	Development of Nondestructive, Field-Deployable, Integrative Approaches to Evaluate Effects of Environmental Stressors on Fishes	Canada Foundation for Innovation, New Opportunities Fund	\$50,000	\$50,000
2003	PI	Development of Nondestructive, Field-Deployable, Integrative Approaches to Evaluate Effects of Environmental Stressors on Fishes	Saskatchewan Science and Innovation Fund	\$50,000	\$50,000
2003	PI	Start-up Funds	University of Saskatchewan	\$35,000	\$35,000
2003	PI	Establishment of Zebrafish (<i>Danio rerio</i>) as a Comparative Biomedical Whole Animal Model for Toxicological Research	WCVM Research Trust Fund	\$3,850	\$3,850
2002-2005	Co-PI	Development of a Reptile Egg Screening Assay for Endocrine Disrupting Chemicals (L.G. Talent)	American Chemistry Council, Long Range Research Initiative	\$173,667	\$521,000
2003-2004	PI	Bioenergetics and Growth of Juvenile Arctic Grayling Inhabiting an Artificial Tundra Stream	BHP Billiton Diamonds Inc.	\$7,200	\$7,200
2003	Co-PI	Uranium Toxicity Testing using Early Life Stage Northern Pike (K. Liber)	Saskatchewan Environment	\$30,000	\$30,000
2002-2003	Co-PI	Uranium Toxicity Testing using Early Life Stage Lake Trout (K. Liber)	Saskatchewan Environment	\$38,000	\$38,000
2001-2002	Co-PI	Effects of Suspended Sediment on Growth and Performance of Juvenile Salmonids (I.K.	Fisheries and Oceans Canada/Greater	\$123,500	\$247,000

		Birtwell)	Vancouver Regional District		
2000- 2002	Co- PI	A Study of Potential Endocrine Disrupting Compounds Present in Ground and Surface Water from Confined Animal Feed Operations (J.N. Dumont)	United States Environmental Protection Agency	\$25,000	\$50,000
2000- 2002	PI	Evaluation of Biochemical Measures of Condition as Methods for Monitoring Fish Response to Altered Instream Flow Regimes	British Columbia Hydro and Power Authority, Strategic Environmental Initiatives Program	\$62,500	\$125,000
1998- 2001	PI	Ecotoxicity Risks Associated with the Land Treatment of Petrochemical Wastes	United States Environmental Protection Agency, Exploratory Research Program	\$135,667	\$407,000
1999- 2001	Co- PI	Evaluation of Eastern and Western Fence Lizards as Reptile Models for Assessment of Endocrine-Mediated Toxicity (L.G. Talent)	American Chemistry Council, Long Range Research Initiative	\$84,000	\$252,000
2000- 2001	PI	Development of an <i>In Vivo</i> Model in Zebrafish for Evaluating Long Term Reproductive Effects of Estrogenic and Androgenic Contaminants	Center for Water Research, Oklahoma State University	\$25,000	\$25,000
1999- 2000	PI	Field Evaluation of Reproductive Endocrine Function in Fish Exposed to Toxicants in Oklahoma Watersheds	Center for Water Research, Oklahoma State University	\$25,000	\$25,000
1998- 1999	PI	Regulation and Perturbation of Reproductive Endocrine Function in Ovarian Follicles from Catfish	Center for Water Research, Oklahoma State University	\$24,000	\$24,000
1998- 1999	Co- PI	Development of Dough Baits for Trout and Carp (D. Bellmer, R.P. Lanno, C.I. Abramson)	Oklahoma Center for Advancement of Science and Technology	\$38,000	\$38,000
1998- 1999	Co- PI	Development of Methods to Assess Reproductive Endocrine Function in Fish (G. Van Der Kraak, L. Tremblay, M. McMaster, M. Hewitt)	Canadian Network of Toxicology Centres	\$85,000	\$85,000
1998-	PI	Development of an <i>In Vitro</i>	Center for Sensors	\$10,000	\$10,000

1999		Biological Sensor for Assessing Ovarian Toxicants in Fish	and Sensor Technologies, Oklahoma State University		
1997	PI	Start-up Funds	Oklahoma State University	\$70,000	\$70,000

22. ARTISTIC EXHIBITIONS OR PERFORMANCES: None

23. PROFESSIONAL PRACTICE:

23.1 CLINICAL PRACTICE: N/A

23.2 OTHER FORMS OF PROFESSIONAL ACTIVITY:

Member, Advisory Committee on University-Industry Grants (ACUIG), Natural Sciences and Engineering Research Council of Canada(2010 – 2013)
 President, Prairie Northern Regional Chapter of the Society of Environmental Toxicology and Chemistry (2009 – present)
 Editorial Board Member, *Aquatic Toxicology* (1998 – present)
 Member, Nominations Committee, Society of Environmental Toxicology and Chemistry (2010-present)
 Member, Regional Chapters Committee, Society of Environmental Toxicology and Chemistry (2009-present)
 Member, North American Metals Council, Selenium Working Group (2008 – present)
 Member, Canadian Industry Selenium Working Group (2006 – present)
 Workgroup Chair, Pellston Workshop on the Ecological Assessment of Selenium in the Aquatic Environment, Society of Environmental Toxicology and Chemistry, Pensacola, FL, USA, February 21-27, 2009
 Chair, Scientific Program, Aquatic Toxicity Workshop, Saskatoon, SK, October 5-8, 2008
 Session Chair, Society of Environmental Toxicology and Chemistry Annual Meeting, Montreal, QC (2006)
 Session Chair, Aquatic Toxicity Workshop, Jasper, AB (2006)
 Member, Tissue Threshold Sub-Committee, North American Industry Selenium Working Group (2006 - 2008)
 Invited Session Chair, Aquatic Toxicity Workshop, Waterloo, ON (2005)
 Session Chair, Society of Environmental Toxicology and Chemistry Annual Meeting, Austin, TX (2003)
 Past President, Ozark-Prairie Chapter of the Society of Environmental Toxicology and Chemistry (2000-2002)
 Session Chair, Society of Environmental Toxicology and Chemistry Annual Meeting Salt Lake City, UT (2002)

24. CONSULTING WORK UNDERTAKEN: None

25. DEPARTMENTAL AND COLLEGE COMMITTEES:

Aquatic Toxicology Research Facility (ATRF) Management Committee (2008 – 2010)

WCVN Local Safety Committee (Co-Chair) (2004 – present)

Wildlife Health Fund Committee (2004 – 2005)

Library Committee (2002 – 2005)

Seminar Committee, Department of Zoology, Oklahoma State University (1998 – 2000)

Planning Committee, Department of Zoology, Oklahoma State University (Chair) (2000-2002)

26. UNIVERSITY COMMITTEES:

University Council, WCVN Representative (2008 – 2011)

Research, Scholarly and Artistic Work (RSAW) Committee of University Council (Chair, 2009-2011) (2008 – 2011)

University Council, Coordinating Committee (2009 – 2011)

University Council, Assessment Advisory Committee (2009 – 2010)

University Council, Indirect Costs Committee (2009 – 2010)

Centres Subcommittee (2010-2012)

Joint Board-Council Committee on Chairs and Professorships (2010-2011)

Canada Research Chairs Advisory Committee (2010-2012)

Distinguished and New Researcher Selection Committee (2010-2011)

Centres Forum (2010-2012)

Canada Research Chairs Forum (2010-2011)

Toxicology Undergraduate Program Advisory Committee (2004 – present)

**27. PROFESSIONAL AND ASSOCIATION OFFICES AND COMMITTEE ACTIVITY
UNIVERSITY: None**

28. PUBLIC AND COMMUNITY CONTRIBUTIONS:

28.1 UNIVERSITY RELATED:

- Janz, D.M. 2009. Development of stress biomarkers in skin and hair. Invited seminar, Workshop on Remote Sensing and Environmental Management, University of Saskatchewan, June 5, 2009.
- Interviewed for article in *Green Living Online* titled "A Tough Pill to Swallow", June 4, 2009. <http://green.sympatico.msn.ca/green-living-online-article.aspx?cp-documentid=1078909>
- Judge for Life and Health Sciences Research Conference, University of Saskatchewan, March 16, 2008
- Interviewed and quoted in article for CanWest News Service (appeared in Saskatoon Star Phoenix, Vancouver Sun and Ottawa Citizen) titled "Flushing Away Our Future", August 4, 2007.
- Judge for Life and Health Sciences Research Conference, University of Saskatchewan, March 16, 2007
- Janz, D.M. Aquatic ecotoxicology of uranium mining in northern Saskatchewan. Invited seminar, WCVM Chapter of the Wildlife Disease Association, February 13, 2007
- Invited speaker, Canadian Water Resources Association, Students and Young Professionals Chapter, October 12, 2006.
- Judge for Life and Health Sciences Research Conference, University of Saskatchewan, March 17, 2006
- Janz, D.M. Molecular to ecological effects of environmental estrogens in fish. Reproductive Science and Medicine seminar, March 17, 2005.
- Janz, D.M. 2002. Hormonal and environmental control of gonadal cell apoptosis in fishes. Invited seminar, Department of Biology, University of Saskatchewan.
- Interviewed by Shaw Cable News, August 2002 (antimicrobial drug tetracycline detected in South Saskatchewan River).
- Judge for Graduate Student Research Day, Western College of Veterinary Medicine, 2002.

28.2 NOT UNIVERSITY RELATED: None

Vince Palace



Employment:

April 1999-Present **Government of Canada, Department of Fisheries and Oceans
Habitat Impacts Research Section**

Position: Research Scientist
Director – Centre for Environmental Research on Pesticides

Responsibilities: Develop, design, and implement research projects to create and advance knowledge of factors affecting productivity and health of fish and fish habitat. Emphasis is on the biochemistry, histology and physiology measures of fish health and impacts on those by specific environmental contaminants, including pesticides.
Identify research opportunities, write research proposals, seek and foster collaborative research, and communicate research results in the primary scientific literature and at national and international conferences and meetings.
Supervise a research team and maintain large databases.
Train undergraduate and graduate students.
Critically review and evaluate manuscripts for scientific journals.
Represent the region and Department at national and international meetings.

September 1999-Present **University of Manitoba, Departments of Zoology
And Environmental Studies**

Position: Adjunct Professor and Lecturer

Responsibilities: Coordinate, deliver and grade the 4th year *Environmental Toxicology* course as well as Contribute to the *Aquatic Biology Research Group*
Graduate Student teaching
Supervise graduate students
Design, supervise and grade graduate level *Topics in Zoology* courses

- February 1999-
March 1999 **Government of Canada, Department of Fisheries and Oceans
Habitat Management Division**
- Position:* Impact Assessment Biologist, Alberta Area
- Responsibilities:* Reviewed projects potentially impacting freshwater fisheries resources under the framework of the *Fisheries Act* and the *Canadian Environmental Assessment Act*
Approved, provided advice for mitigating effects, or withheld authorization under the *Fisheries Act*
Conducted site inspections
Maintained public record of all relevant documents
- 1995-1997 **BEAR Environmental Science**
- Position:* Consultant and Principal
- Responsibilities:* Acted as an independent consultant to review environmental impact statements for their thoroughness of biological effects monitoring considerations.
- 1990-1994 **Government of Canada, Department of Fisheries and Oceans
Contaminants and Toxicology Research Section**
- Position:* Project Leader, Great Lakes Lake Trout Study
- Projects:* Research examined the mechanistic links between exposure to toxic chemicals, oxidative stress and reproductive failure in fish from the Great Lakes. High performance liquid chromatography (HPLC) techniques to quantify vitamins were developed.
- 1991-1995 **University of Manitoba, Department of Zoology**
- Position:* Teaching Assistant, Environmental Toxicology
- Responsibilities:* Prepared lecture materials and evaluated students' oral and written course work.

1987-1991 **University of Manitoba, Department of Zoology**

Position: Teaching Assistant, Human Anatomy and Physiology

Responsibilities: Lectured and demonstrated cellular, tissue level and whole body examinations of human anatomy and physiology; prepared and graded student examinations.

Education:

July 1996-
January 1999 **Manitoba Health Research Council Postdoctoral Fellow**
Cardiovascular Research Institute
St. Boniface Hospital, Winnipeg, Manitoba

Project: The role of non-enzymatic antioxidants in the pathogenesis and progression of heart failure.

1992-1996 **Doctor of Philosophy (Ph.D.)**
Department of Zoology
University of Manitoba

Thesis Title: Oxidative Stress in Lake Trout (*Salvelinus namaycush*)
Exposed to Organochlorines that Induce the Phase I
Biotransformation Enzyme System

1988-1991 **Masters of Science (MSc.)**
Department of Zoology
University of Manitoba

Thesis Title: Superoxide Dismutase, Catalase and Glutathione Peroxidase
Enzyme Activities as Indicators of Cadmium Exposure in
Freshwater Fish from Small Canadian Pre-Cambrian Shield Lake

Primary Current Research:

Title: **Effects based evaluation of current use pesticides on fish and fish habitat by the Center for Environmental Research on Pesticides**

Role: National Director

Annual Budget: \$1M/yr

Project focus:

- carry out effects based research related to the potential impacts of pesticides on fish and fish habitat in marine and freshwater ecosystems.
- consult with the Pest management Regulatory agency (PMRA) and other federal departments to identify high priority research needs related to the environmental effects of pesticides on fish and fish habitat.
- present results of effects based research to PMRA and other regulatory bodies in order to influence regulatory decisions in favour of increased protection for fish, fish habitat and marine ecosystems.
- provide advice to PMRA and other regulatory units on the potential for pesticides to impact fish and fish habitat.
- Administer a research fund to support a network of pesticide expertise of regional research to address national pesticide priorities.

Refereed Publications:

Marvin CM, Tomy GT, Armitage JM, Arnot JA, McCarty L, Covaci A, **Palace V**. 2011. Hexabromocyclododecane: current understanding of chemistry, environmental fate and toxicology and implications for global management. *Environ. Sci. Technol.* 45, 8613–8623

Tomy GT, **Palace VP**, Marvic C, Stapleton HM, Covaci A, Harrad S. 2011. Biotransformation of HBCD in Biological Systems Can Confound Temporal-Trend Studies. *Environ. Sci. Tech.* 45, 364–365.

Gemmill B, Pleskach K, Peters L, **Palace V**, Wautier K, Park B, Darling C, Rosenberg B, McCrindle R, Tomy GT. 2011. Toxicokinetics of tetrabromoethylcyclohexane (TBECH) in juvenile brown trout (*Salmo trutta*) and effects on plasma sex steroids. *Aquat. Toxicol.* 101:309-317.

Friedrich LA, Orr PL, Halden NM, Yang P, **Palace VP**. 2011. Exposure histories derived from selenium in otoliths of three cold-water fish species captured downstream from coal mining activity. *Aquat. Toxicol.* 105:492-496.

Marteinson SC, Kimmins S, Letcher RJ, **Palace VP**, Bird DM, Ritchie IJ, Fernie KJ. 2011. Diet

exposure to technical hexabromocyclododecane (HBCD) affects testes and circulating testosterone and thyroxine levels in American kestrels. (*Falco sparverius*). *Environ. Res.* 111:1116-1123.

Park BJ, **Palace V**, Wautier K, Gemmill B, Tomy G. 2011. Thyroid axis disruption in juvenile Brown trout (*Salmo trutta*) exposed to the flame retardant β -tetrabromocyclohexane (β -TBECH) via the diet. *Environ. Sci. Technol.* 45:7923-7927.

Boudreau HS, Rossiter JP, Williams LD, **Palace VP**, Ross GM. 2009. The Effects of Metal Ion Exposure with α -synuclein-like immunoreactivity in the Central Nervous System of Fish, *Catostomus commersoni*. *Aquat. Toxicol.* 92:258-263

Zhang S, Bursian SJ, Martin PA, Chan HM, Tomy G, **Palace VP**, Mayne GJ, Martin JW. 2009. Reproductive and developmental toxicity of a pentabrominated diphenyl ether mixture, DE-71, to ranch mink (*Mustela vison*) and hazard assessment for wild mink in the Great Lakes region. *Toxicol. Sci.* 110:107-116.

Johnson KE, Park BJ, **Palace VP**, Ollson CA, Reimer KJ. 2009. Biomarkers as indicators of marine ecosystem recovery at a polychlorinated biphenyl contaminate site at Saglek Bay, Labrador. (????)

Miller L, Rasmussen JB, **Palace VP**, Hontela A. 2009. The physiological stress response and oxidative stress biomarkers in rainbow trout and brook trout from selenium impacted streams in a coal mining region. *J. Applied Toxicol.* 29:681-688.

Godard D, Park BJ and **Palace VP**. 2009. Histopathological assessment of the sub-lethal effects of instantaneous pressure changes (IPCs) on the internal organs of fish. *Environ. Sci. Technol.* (Submitted Nov. 2010)

Godard D and **VP Palace**. 2008. A review of the evolution, structure, development and function of the teleost swimbladder and factors governing its susceptibility to pressure induced effects from underwater detonations. DFO Technical report (In preparation)

Miller, LL, Rasmussen JB, **Palace VP**, Hontela A. 2009. The physiological stress response in white suckers from agricultural drain waters containing pesticides and selenium. *Ecotox. Environ. Saf.* 72:1249-1256

Goodmanson, AT, **VP Palace** and D Gillis. 2008. Assessing site fidelity of golden shiners (*Notemigonus chrysoleucas*) in a riverine environment using mark-recapture analysis and Monte Carlo data simulation. DFO Technical Report (In preparation).

Palace, VP, BJ Park, K Pleskach, B Gemmill and GT Tomy. 2009. Biotransformation enzymes and thyroid axis disruption in juvenile Rainbow trout (*Oncorhynchus mykiss*) exposed to HBCD Diastereoisomers. *Environ. Sci. Tech.* 42:1967-1972.

Palace VP, RE Evans, KG Wautier, KH Mills, PJ Blanchfield, BJ Park, CL Baron and KA Kidd. 2008. Interspecies differences in biochemical, histopathological, and population responses in four wild fish species exposed to ethynyestradiol added to a whole lake. *Can. J. Fish. Aquat. Sci.* 66:1920-1935.

Palace VP, K Pleskach, T Halldorson, R Danell, K Wauiter, RE Evans, M Alae, C Marvin and GT Tomy. 2007. Biotransformation enzymes and thyroid axis disruption in juvenile rainbow trout (*Oncorhynchus mykiss*) exposed to hexabromocyclododecane diastereoisomers. *Environ. Sci. Technol.* 42:1967-1972.

Werner J, **V Palace**, R Shiu and A Yarmil. 2008. A real-time PCR method for the quantification of the two isoforms of metallothionein in lake trout (*Salvelinus namaycush*). *Arch. Environ. Contam. Toxicol.* 54:84-91.

Couillard CM, RW Macdonald, SC Courtenay and **VP Palace**. 2007. Interactions between toxic chemicals and other environmental factors affecting the risk of impacts on aquatic organisms: a review with a Canadian perspective – Interactions affecting exposure. *Environmental Reviews.* 16:1-17.

Palace VP, NM Halden, P yang, RE Evans, G sterling. 2007. Determining residence patterns of rainbow trout using laser ablation inductively coupled plasma mass spectrometry (LA-ICP-MS) analysis of selenium in otoliths. *Environ. Sci. Tech.* 41:3679-3683.

Martin, P.A., Mayne, G.J., Bursian, S.J., Tomy, G., **Palace, V.**, Pekarik, C., and Smits, J. 2007. Immunotoxicity of the commercial polybrominated diphenyl ether mixture DE-71 in ranched mink. *Environ Toxicol Chem.* 26: 988-997.

Kidd KA, PJ Blanchfield, KH Mills, **VP Palace**, RE Evans, J Lazorchak and B Flick. 2007. Collapse of a fish population following exposure to a synthetic estrogen. *PNAS.* 104:8897-8901.

Miller LL, F Wang, VP Palace and A Hontela. 2007. Effects of acute and subchronic exposures to waterborne selenium on the physiological stress response and oxidative stress indicators in juvenile rainbow trout. *Aquat. Toxicol.* 83:263-271.

Fudge TS, KG Wautier and **VP Palace**. 2008. Escapement success of rainbow trout (*Oncorhynchus mykiss*) fry from artificial redds with different fine sediment loadings. *NAJFM.* 28:758–765.

Tomy GT, **VP Palace**, K Pleskach, N Ismail, T Oswald, R Danell, K Wautier, RE Evans. 2007. Dietary exposure of juvenile rainbow trout (*Oncorhynchus mykiss*) to 1,2(bis(2,4,6-tribromophenoxy)ethane (BTBPE): bioaccumulation parameters and biochemical effects. *Environ. Sci. Technol.* 41:4913-4918.

Palace VP, S Kollar, RE Evans, LE Peters, CL Baron, KG Wautier and M Parsosn. 2007. An

initial assessment of arsenic mobilization in an area of historic gold mining in Nova Scotia, Canada and bioaccumulation of arsenic and mercury in resident American Eels (*Anguilla rostrata*). *Geochem. Explor. Environ. Anal.* (Invited paper, In Press).

Law K, **VP Palace**, T Halldorson, R Danell, K Wautier, RE Evans, M Alaei, C Marvin, G Tomy. 2006. Dietary accumulation of hexabromocyclododecane diastereoisomers in juvenile rainbow trout (*Oncorhynchus mykiss*) I: bioaccumulation parameters and evidence of bioisomerization. *Environ. Toxicol. Chem.* 25: 1757-1761.

Palace VP, Werner J. 2006. Vitamins A and E in the Maternal Diet Influence Egg Quality and Early Life Stage Development in Fish: A Review. *Proceedings of the 29th Larval Fish Conference, Barcelona Sp, July 11-14, 2005. Scientia Marina.* 70S2:41-57

Klaverkamp JF, **VP Palace**, CL Baron, RE Evans, KG Wautier. 2006. Cumulative effects of multiple contaminants on caged fish. *Can. Wat. Qual. Res. J.* 41:244-255.

Doebel C, Baron CL, Evans RE, **Palace VP**. 2005. The use of small bodied forage fish in caged exposures for metal mining environmental effects monitoring. *Proceedings of the International Conference on Heavy Metals in the Environment. Rio De Janeiro, Brazil. June 5-9, 2005.* (In press).

Gray M, Munkittrick K, Dahl J, Baron C, Wautier K, Kollar S, Werner J, **Palace V**. 2005. METALS and metallothionein in slimy sculpin (*Cottus cognatus*) captured at a Diamond mining Site in Northern Canada. *Proceedings of the International Conference on Heavy Metals in the Environment. Rio De Janeiro, Brazil. June 5-9, 2005.* (In press).

Werner J, Wautier K, Mills K, Chalanchuk S, Kidd K, **Palace VP**. 2005. Reproductive fitness of lake trout (*Salvelinus namaycush*) exposed to environmentally relevant concentrations of the potent estrogen ethynylestradiol (EE2) in a whole lake exposure experiment. *Proceedings of the 29th Larval Fish Conference, Barcelona Sp, July 11-14, 2005 Scientia Marina,* 70S2:59-66.

Hill MF, **Palace VP**, Kaur K, Kumar D, Khaper N, Singal PK. 2005. Reduction in oxidative stress and modulation of heart failure subsequent to myocardial infarction in rats. *Exper. Clin. Cardiol.* 10:146-153.

Palace VP, C Doebel, CL Baron, RE Evans, KG Wautier, JF Klaverkamp, J Werner, S Kollar. 2005. Caging small bodied fish as an alternative method for environmental effects monitoring (EEM). *Can. Wat. Qual. Res. J.* 40:328-333.

Palace VP, Wautier KG, Evans RE, Blanchfield P, Mills K, Chalanchuk S, Godard D, McMaster ME, Tetrault G, Peters LE, Vandenbyllaardt L and Kidd KA. 2006. Biochemical and Biochemical and Histopathological Effects of Ethynylestradiol in Pearl Dace (*Margariscus margarita*) Chronically Exposed to the Synthetic Estrogen in a Whole Lake Experiment. *Environ.*

Toxicol. Chem.. 25:260-271.

Holm J, **VP Palace**, P. Siwik, G Sterling, RE Evans, CL Baron, J Werner, K Wautier. 2005. Developmental effects of bioaccumulated selenium in eggs and larvae of two salmonid species. Environ. Toxicol. Chem. 24:2373-2381.

Bauder MB, **Palace VP**, Hodson PV. 2005. Is oxidative stress the mechanism of blue sac disease in retene-exposed trout larvae? Environ. Toxicol. Chem. 24:694-702.

Spallholz JE, Boylan LM, **Palace VP**, Chen J, Smith L, Rahman MM, Robertson JD. 2005. Arsenic and selenium in human hair: a comparison of five countries with and without arsenicosis. Biological Trace Element Research. 106(galley stage)

Martin PA, Mayne G, Bursian S, **Palace VP**, Kurunthachalam, K. 2005. Thyroid and vitamin A status in mink fed PCB-contaminated carp from Saginaw Bay, Michigan. Environ. Res. 101:53-67.

Dwillow R, **Palace V**, Kaur K, Singal PK. 2004. Regional changes in metallothioneins during global ischemia-reperfusion in rats. In Heart Disease- pathogenesis, Diagnosis and Treatment. A. Kimchi [Ed]. Medimond International Proceedings, Bologna, Italy, pp. 327-333.

Tomy GT, **Palace VP**, Halldorson T, Braekevelt E, Danell R, Wautier K, Evans B, Brinkworth L, Fisk AT. 2004. Bioaccumulation, biotransformation and biochemical effects of brominated diphenyl ethers in juvenile lake trout (*Salvelinus namaycush*). Environ. Sci. Technol. 38:1496-1504.

Tomy,GT, SA Tittlemier, **VP Palace**, E Braekevelt, WR Budakowski, B Lau, G Eales, JC Plohman. 2003. Biotransformation of Perfluorooctane Sulfonamide and Ethyl Perfluorooctane Sulfonamide by Rainbow Trout (*Onchorhynchus mykiss*) Liver Microsomes. *Environ. Sci. Technol.* 38:758-762.

Brown, SB, RE Evans, L Vandenbyllardt, KW Finnson, **VP Palace**, AS Kane, AY Yarechewski, DCG Muir. 2003. Altered Thyroid Status in Lake Trout (*Salvelinus namaycush*) Exposed to Co-planar 3,3',4,4',5-Pentachlorobiphenyl. Aquat. Toxicol. 67:75-85.

Doebel C, CL Baron, RE Evans, KG Wautier, J Werener and **VP Palace**. 2004. The potential for using caged pearl dace (*Semotilus margarita*) for metal mining Environmental Effects Monitoring. (EEM). 2003. Bull. Environ. Contam. Toxicol. 72:409-414.

Palace, VP. 2003. Vitamin E as a mediator and indicator of toxicant induced stress in fish. In Vitamin E in Health and Disease. K. Dabrowski [Ed]. (*In Press:Invited Paper*).

Palace, VP, CL Baron, RE Evans and K Wautier 2003. Mobility of Arsenic in a Canadian Freshwater System Receiving Gold Mine Effluents. J. Physique IV 107:1009-1012.

Palace, VP, CL Baron, RE Evans, K Wautier and JF Klaverkamp. 2003. Metals, Metalloids and Metallothionein in Tissues of Fish from A Canadian Freshwater System Receiving Gold Mining Effluents and in Fish from Nearby Reference Areas. *J. Physique IV* 107:1005-1008.

Palace, VP, JE Spallholz, J Holm, K Wautier, RE Evans and CL Baron. 2004. Metabolism of selenomethionine by rainbow trout (*Oncorhynchus mykiss*) embryos can generate oxidative stress. *Ecotoxicol. Environ. Saf.* 58:17-21

Palace, VP, RE Evans, K Wautier, CL Baron, S Kollar, J Werner, J Holm, P Siwik, G Sterling, CF Johnson. 2003. An assessment of the potential for selenium to impair reproduction in bull trout (*Salvelinus confluentus*) from an area of active coal mining. *Environ. Biol. Fishes.* (In Press).

Spallholz, JE, **VP Palace**, TW Reid. 2004. Methioninase and selenomethionine but not Se-methylselenocysteine generate methylselenol and superoxide in an in vitro chemiluminescence assay: experiments and review. *Biochem. Pharmacol.* 67:547-554.

Nicholas, D, S Ramamoorthy, **VP Palace**, S Spring, J Moore, RF Rosenzweig. 2002. Biogeochemical Transformations of Arsenic in Circumneutral Freshwater Sediments. *Biodegradation* 14(2):123-37.

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Abstracts Presented:

Carroll L, Thébeau N, Halden N, Hanson M, Palace V. 2011. A Retrospective Analysis of Manganese in Lake Trout (*Salvelinus namaycush*) Otoliths: links to reproductive failure. 32nd

Annual Meeting of the Society of Environmental Toxicology and Chemistry, Boston MA, Nov. 13-17.

Park B, **Palace VP**, Wautier K, Blanchfield P, Kidd K. 2011. Recovery of wild fish populations after a whole-lake estrogen addition. 32nd Annual Meeting of the Society of Environmental Toxicology and Chemistry, Boston MA, Nov

Beaudry M, Mandrak N, Campbell R, Glebe B, **Palace VP**. 2011. Assessment of the potential effects of the aquatic herbicide, Diquat, on the growth of juvenile Spotted Gar (*Lepisosteus oculatus*). 21st Annual Meeting of the European Society of Environmental Toxicology and Chemistry, Milan, Italy. May 15-19.

Fernie K, Letcher R, Alae M, Champoux L, Brimble S, **Palace VP**. 2011. Changes in retinol and thyroid function of wild peregrine falcon (*Falco peregrinus*) nestlings relative to brominated flame retardants. 21st Annual Meeting of the European Society of Environmental Toxicology and Chemistry, Milan, Italy. May 15-19.

Friedrich LA, Halden NM, **Palace VP**. 2011. Selenium uptake in otoliths from cold-water fish species captured downstream from coal mining. Goldschmidt Conference, Prague Czech republic August 14-19.

Darling C, **Palace VP**, Tomy G. 2011 Thyroid hormone extraction from plasma: exploring pH and solvent choice. 31st International Symposium on Halogenated Persistent Organic Pollutants POPs' Science in the Heart of Europe. Brussels, Belgium. August 21-25.

Fernie K, Champoux L, Brimble S, Alae M, **Palace VP**. 2011. Spatial patterns and correlations of retinol, thyroid hormones and flame retardants in nestling Peregrine Flacons across the Canadian Great Lakes-St. Lawrence basin. 31st International Symposium on Halogenated Persistent Organic Pollutants POPs' Science in the Heart of Europe. Brussels, Belgium. August 21-25.

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Doebel C, Baron CL, Evans RE, **Palace VP**. 2005. The use of small bodied forage fish in caged exposures for metal mining environmental effects monitoring. Proceedings of the International Conference on Heavy Metals in the Environment. Rio DeJaneiro, Brazil. June 5-9, 2005.

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Doebel, C, Baron, C, Wautier, K, Evans, R, **Palace, V**. 2004. The Use of Small Bodied Forage Fish in Caged Exposures for Metal Mining Environmental Effects Monitoring (EEM). 25th Annual Society of Environmental Toxicology and Chemistry Meeting, Portland, OR, Nov. 14-18.

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Kidd, K, **Palace, V**, Blanchfield, P, Mills, K, Wautier, K, Lazorchak, J, Lattier, D. 2004. Differences in the biochemical- through population-level responses of two minnow species exposed to a potent estrogen mimic. . 25th Annual Society of Environmental Toxicology and Chemistry Meeting, Portland, OR, Nov. 14-18.

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Martin, P, Bursian, S, Mayne, G, Tomy, G, **Palace, V**. 2004. Immunomodulation in Ranch Mink (*Mustela vison*) Induced by Dietary Exposure to a Commercial PBDE Mixture. 25th Annual Society of Environmental Toxicology and Chemistry Meeting, Portland, OR, Nov. 14-18.

Doebel C, CL Baron, KG Wautier, RE Evans and **VP Palace**. 2004. The Use of Small Bodied Forage Fish in Caged Exposures For Metal Mining Environmental Effects Monitoring (EEM). 31st Annual Aquatic Toxicity workshop, Charlottetown, PEI. Oct. 24-27.

Palace VP, Doebel C, CL Baron, KG Wautier, RE Evans. 2004. Caged, Small Bodied Forage Fish For Metal Mining Environmental Effects Monitoring (EEM). 31st Annual Aquatic Toxicity workshop, Charlottetown, PEI. Oct. 24-27.

Gray M, C Smith, B Park, **V Palace**, K Kidd. 2004. Depression of brain acetylcholinesterase (AChE) in fish collected downstream of agricultural activities. 31st Annual Aquatic Toxicity

workshop, Charlottetown, PEI. Oct. 24-27.

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VP Palace, RE Evans, KG Wautier, CL Baron, J Holm. 2004. Toxicity of Selenium to Freshwater Fishes. Atelier Workshop on Selenium: Selenium and hydroelectric reservoirs. March 22, Montreal, QC.

V.P. Palace, C. Doebel, C.L. Baron, R.E. Evans, K.G. Wautier, J. Werner, S. Kollar and J.F. Klaverkamp. 2004. The potential for using caged small bodied fish for metal mining environmental effects monitoring (EEM). Environmental Effects Monitoring Science Symposium, Feb. 16-18, Fredericton NB.

Martin PA, Bursian SJ, **Palace VP**, Mayne G. 2004. Thyroid and vitamin A status in mink fed PCB-contaminated carp from Saginaw Bay. 24th Annual Society of Toxicology and chemistry Meeting, November 9-13, Austin TX.

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V.P. Palace, C. Doebel, C.L. Baron, R.E. Evans, K.G. Wautier, J. Werner, S. Kollar and J.F. Klaverkamp. 2004. The Potential for Using Caged Small Bodied Fish For Metal Mining Environmental Effects Monitoring (EEM). EEM Science Symposium, Fredericton, New Brunswick, Feb. 16-18.

V.P. Palace, J.Werner, K. Wautier, C.L. Baron, R.E. Evans and D. Cobb. 2003. Lipid Soluble Vitamins A and E as Integrative Measures of Fish Health for EEM. Offshore Oil and Gas Environmental Effects Monitoring Workshop: Approaches and Technologies. Bedford Institute of Oceanography, Nova Scotia, Canada, May 26-30, 2003

Palace, VP, CL Baron, RE Evans, K Wautier 2003. Mobility of Arsenic in A Canadian Freshwater System Receiving Gold Mine Effluents. Proceedings of the XIIth conference on Heavy Metals in the Environment. Grenoble, France. May 26-30.

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Klaverkamp, JF, **VP Palace**, CL Baron, RE Evans, KG Wautier. 2002. Cumulative impacts on caged fish of gold mine and municipal wastewater effluents and of garbage-disposal run-off. 23rd Annual Society of Environmental Toxicology and Chemistry Meeting. Salt Lake City, UT. Nov. 16-20.

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Palace, VP, RJ Letcher, J Li, T Halldorson, E Braekevelt, R Danell, K Wautier, A Fisk, G Tomy. 2002. Dietary accumulation and biochemical effects of PBDEs in juvenile lake trout. 23rd Annual Society of Environmental Toxicology and Chemistry Meeting. Salt Lake City, UT. Nov. 16-20.

Letcher, RJ, J Li, T Halldorson, E Braekevelt, R Danell, **VP Palace**, K Wautier, A Fisk, G Tomy. 2002. Dietary PBDE Exposure and Phenolic Metabolites in Juvenile Lake Trout (*Salvelinus namaycush*). 23rd Annual Society of Environmental Toxicology and Chemistry Meeting. Salt Lake City, UT. Nov. 16-20.

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Palace, VP, J Holm, K Wautier, RE Evans, CL Baron, G Sterling, P Siwik, JE Spallholz. 2002. Selenium exposure and effects in wild rainbow trout and brook trout from Alberta's Northeast Slopes region. 23rd Annual Society of Environmental Toxicology and Chemistry Meeting. Salt Lake City, UT. Nov. 16-20. (Invited Paper)

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K Kidd, C Podemski, M Paterson, D Findlay, A Salki, **VP Palace**, B Evans, P Blanchfield, K Mills, B Park, J Werner, S Brown, M McMaster, G Van Der Kraak, J Lazorchak, G Toth, D Lattier, M Bagley, K Liber, D Graham, K Londry. 2001. A Whole-Lake Experiment to Examine the Effects of a Synthetic Estrogen on Aquatic Populations. Toxic Substances Research Initiative Regional Conference 9-10 November, Hamilton, ON.

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MB Sept. 30-Oct. 2.

Kidd, K, **VP Palace**, P Blanchfield, K Mills, B Evans, M McMaster, SB Brown, G van Der Kraak, D Lattier, J Lazorchak. 2001. A whole-lake experiment to examine the effects of a synthetic estrogen on fish populations. Aquatic Toxicity Workshop, 30 September - 3 Oct., Winnipeg, MB

Kidd, K, C Podemski, M Paterson, D Findlay, A Salki, **VP Palace**, B Evans, P Blanchfield, K Mills, B Park, J Werner, S Brown, M McMaster, G Van Der Kraak, J Lazorchak, G Toth, D Lattier, M Bagley, K Liber, D Graham, K Londry. 2001. A Whole-Lake Experiment to Examine the Effects of a Synthetic Estrogen on Aquatic Populations. Endocrine TIP Review Meeting, American Chemistry Council, 30 Oct., Chicago, IL.

Kidd, K, **VP Palace**, RE Evans, K Mills, P Blanchfield, L Vandenbyllardt, K Wautier, J Lazorchak, D Lattier, R Flick. 2002. Biochemical and physiological effects in wild fathead minnows from a lake experimentally treated with the synthetic estrogen, ethynylestradiol. Canadian Symposium on Water Quality, 4-5 February, Burlington, ON.

Kidd, K, C Podemski, M Paterson, D Findlay, A Salki, **VP Palace**, B Evans, P Blanchfield, K Mills, B Park, J Werner, SB Brown, M McMaster, G van Der Kraak, J Lazorchak, G Toth, D Lattier, M Bagley, K Liber, D Graham, K Londry. 2002. A Whole-Lake Experiment to Examine the Effects of a Synthetic Estrogen on Aquatic Populations. Canadian Society of Limnologists Meeting, 3-5 January, Vancouver, BC

Baron, CL, JF Klaverkamp, RE Evans, K Wautier, **VP Palace**. 2002. Applying environmental effects monitoring strategies and mitigation approaches to gold mines in Northwestern Ontario. 1st national DFO science Workshop, Sidney, B.C. Nov. 14-16.

Kidd, K, **VP Palace**, P Blanchfield, K Mills, RE Evans, M McMaster, S Brown, G van Der Kraak, D Lattier, J Lazorchak. 2001. A whole lake experiment to examine the effects of a synthetic estrogen on fish populations. 28th Aquatic Toxicity Workshop, Winnipeg, MB. Oct. 30-Sept. 3.

Danelisen, I, **VP Palace**, H Lou, PK Singal. 2001. Maintenance and utilization of retinol (Vitamin A) in adriamycin-induced cardiomyopathy. J of Mol and Cell Cardiol, 33: A25.

Kumar, D, **VP Palace**, I Danelisen, BI Jugdutt, PK Singal. 2001. Probuocol induced antioxidants confers protection against I-R injury. J of Mol and Cell Cardiol, 33: A63, 2001.

Majewski, A, P Blanchfield, K Wautier, **VP Palace**. 2001. Environmentally relevant doses of ethynylestradiol affect reproductive behaviour in male fathead minnows (*Pimephales promelas*). 28th Aquatic Toxicity Workshop, Winnipeg, MB. Oct. 30-Sept. 3.

Palace, VP, K Kidd, K Wautier, RE Evans, TA Dick, J Werner, CL Baron. 2001. Freshwater fish exposed to environmental estrogen contaminants have altered lipid soluble vitamin status.

28th Aquatic Toxicity Workshop, Winnipeg, MB. Oct. 30-Sept. 3.

Holm, J, RE Evans, K Wautier, CL Baron, P Siwik, G Sterling, **VP Palace**. 2001. Evaluations of the reproductive fitness of wild rainbow trout and brook trout exposed to elevated selenium concentrations in an area of active coal mining. 22nd Annual Society of Environmental Toxicology and Chemistry Conference, Baltimore, MD, Nov. 11-15.

Palace, VP, K Kidd, K Wautier, RE Evans, TA Dick, J Werner, C.L. Baron. 2001. Freshwater fish exposed to environmental estrogens have altered lipid soluble vitamin status. 22nd Annual Society of Environmental Toxicology and Chemistry Conference, Baltimore, MD, Nov. 11-15.

Holm, J, **VP Palace**, P Siwik, G Sterling, RE Evans, CL Baron. 2001. Potential effects of selenium on the reproductive fitness of rainbow trout downstream from a coal mine endpit lake. Proceedings of the 40th Annual Meeting of the Society of Toxicology, San Francisco, CA. March 25-29.

Palace, VP, RE Evans, K Kidd, TA Dick. 2001. Long term effects to somatic growth and gonadal maturation in yellow perch (*Perca flavescens*) exposed for short durations to waterborne ethynylestradiol. Proceedings of the 40th Annual Meeting of the Society of Toxicology, San Francisco, CA. March 25-29.

Palace, VP, JP Werner, RE Evans, K Wautier, CL Baron, K Kidd. 2000. Effects of waterborne ethynylestradiol exposure on metallothionein and lipid soluble vitamins in lake trout. 21st Annual Society of Environmental Toxicology and Chemistry Conference, Nashville, TN, Nov. 12-16.

Danelisen, I, T Li, **VP Palace**, PK Singal. 2000. Vitamin A metabolism in adriamycin-induced cardiomyopathy. XXII Annual Meeting of the American Society of the International Society for Heart Research. Louisville, KY. June 14-18. J. Mol. Cell. Cardiol. 32(5): A47.

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Palace, VP, M Hill, F Farahmand and PK Singal. 1998. Antioxidant vitamin pools and hemodynamic function following myocardial infarction in rats. XXth Annual Meeting of the International Society for Heart Research-American Section, Ann Arbor MI, August 9-12. J. Mol. Cell. Cardiol. 30(7):A239

Kumar D, L Kirshenbaum, T Li, **V Palace**, and PK Singal. 1998. Apoptotic and necrotic cell death in adriamycin cardiomyopathy. XXth Annual Meeting of the International Society for Heart Research-American Section, Ann Arbor MI, August 9-12. *J. Mol. Cell. Cardiol.* 30(7):A103.

Palace, VP, D Kumar, MF Hill, PK Singal. 1998. Regional distribution of non-enzymatic antioxidants and oxidative stress in isolated perfused rat hearts. XVI World Congress of the International Society for Heart Research, Rhodes, Greece, May 27-31.

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Kumar, D, L Kirshenbaum, T Li, **VP Palace**, PK Singal. 1998. Apoptotic and necrotic cell death in adriamycin cardiomyopathy. XVI World Congress of the International Society for Heart Research, Rhodes, Greece, May 27-31.

Pichardo, J, **VP Palace**, N Khaper, PK Singal. 1997. Myocardial oxidative stress changes during compensated right heart failure in rats. Fifth World Congress of the International Society for Adaptive Medicine. Framingham, MA. September 7-10.

Palace, VP, D Kumar, J Pichardo, N Khaper, PK Singal. 1997. Ischemia-reperfusion induced changes in non-enzymatic antioxidants and oxidative stress in different regions of the isolated rat heart. Second World Congress of the International Society of Molecular Nutrition and Therapy. Winnipeg, MB. August 2-4.

Palace, VP, D Kumar, MF Hill, PK Singal. 1997. Regional distributions of non-enzymatic antioxidants and oxidative stress in isolated perfused rat hearts. Second World Congress of the International Society of Molecular Nutrition and Therapy. Winnipeg, MB. August 2-4.

Palace VP, SB Brown, CL Baron, J Fitzsimons, JF Klaverkamp. 1996. Oxidative stress indices in adult lake trout (*Salvelinus namaycush*) from Lake Ontario and Lake Superior. American Fisheries Society Early Mortality Special Symposium. Dearborn, MI. August 26-29. Invited Paper.

Brown, SB, **VP Palace**, JF Klaverkamp, J Fitzsimons. 1996. Vitamin dynamics in developing lake trout (*Salvelinus namaycush*) from Lake Ontario. American Fisheries Society Early Mortality Special Symposium. Dearborn, MI. August 26-29. Invited Paper.

Palace VP, SB Brown, CL Baron, JF Klaverkamp. 1996. Insights into retinoid metabolism provided by recoveries of ³H-retinol from tissues of lake trout (*Salvelinus namaycush*) pre-exposed to PCB 126. International Association for Great Lakes Research. Erindale College,

Mississauga, ON. May 25-30. Invited Paper.

Brown, SB, **VP Palace**. 1996 Vitamin dynamics in fish exposed to contaminated environments. 39th Conference of the International Association for Great Lakes Research. Erindale College, Mississauga, ON. May 25-30. Invited Paper.

Palace, VP, JF Klaverkamp. 1996. Oxidative stress in freshwater fish exposed to organochlorines. University of Manitoba Zoology Seminar Series, Winnipeg, Manitoba, March 21.

Brown, SB, **VP Palace**, JF Klaverkamp, J Fitzsimmons. 1995. Vitamins and oxidative stress in lake trout (*Salvelinus namaycush*) displaying early mortality syndrome. 2nd Workshop on Reproductive Disturbances in Fish. Stockholm, Sweden, November 20-23.

Palace, VP, JF Klaverkamp, CL Baron, SB Brown. 1995. Retinoid metabolism in lake trout (*Salvelinus namaycush*) exposed to 3,3',4,4',5-pentachlorobiphenyl (PCB 126). 16th Annual Society of Environmental Toxicology and Chemistry Conference, Vancouver, B.C., Nov. 4-9.

Palace, VP, TA Dick, SB Brown, CL Baron, JF Klaverkamp. 1994. Oxidative Stress in Lake Sturgeon Orally Exposed to 2,3,7,8-tetrachlorodibenzofuran. 15th Annual Society of Environmental Toxicology and Chemistry Conference, Denver, Colorado, October 29-November 3.

Palace, VP, JF Klaverkamp, AS Kane. 1993. Phase I and Phase II metabolism enzyme activities and oxidative stress in lake trout from Lake Ontario. 14th Annual Society of Environmental Toxicology and Chemistry Conference, Houston, Texas, November 14-18.

Brown, SB, **VP Palace**, DA Metner, JF Klaverkamp, WL Lockhart. 1993. Effect of 3,3',4,4',5-pentachlorobiphenyl on retinoids, tocopherol and hepatic enzyme levels in lake trout (*Salvelinus namaycush*). 36th Conference of the International Association For Great Lakes Research, De Pere, Wisconsin, June 4-10.

Palace, VP, JF Klaverkamp, SB Brown, WL Lockhart, DA Metner. 1992. An assessment of lipid peroxidative indices in lake char (*Salvelinus namaycush*) exposed to mixed-function oxidase inducing 2,3',4,4',5-pentachlorobiphenyl. 13th Annual Society of Environmental Toxicology and Chemistry Conference, Cincinnati, Ohio, November 8-12.

Palace, VP, HS Majewski, JF Klaverkamp. 1990. Vitamin E and C deficiency and Cd toxicity in rainbow trout (*Onchorhynchus mykiss*). 19th Annual Fish Feed and Nutrition Workshop, Winnipeg, MB, October 15-19.

Brown, SB, DF Malley, CL Baron, PSS Chang, E DeBruyn, R Evans, D Findlay, S Harrison, M Holoka, RV Hunt, J Klaverkamp, D Majewski, R McNichol, **V Palace**, CRanson, E Scherer, E Schindler, L Wesson. 1990. Cadmium whole lake experiment at the Experimental Lakes Area,

northwestern Ontario and associated studies on cadmium. Proceedings of the 5th Annual LRTAP Workshop, St. John's Newfoundland, October 9-11.

Students Supervised Directly

A) Postdoctoral Fellows

[Redacted]

[Redacted]

[Redacted]

[Redacted]

[Redacted]

[Redacted]

[Redacted]

C) Undergraduate Students

[Redacted]

[Redacted]

[Redacted]






Awards and Distinctions:

2011 Sesion chair, SETAC Europe Milan Italy May 15-19 2011.

2007 Editorial Board member of Environmental Toxicology and Chemistry

2007 Session Chair for Aquatic Toxicity Workshop, Halifax NS.

2006 Session Chair for ANCAP / SETAC Africa International Conference on Pesticide Use in
Developing Countries, Tanzania

2001 Session Chair for Annual Society of Environmental Toxicology and Chemistry Meeting,
Baltimore, MD

2001 Session Chair for Aquatic Toxicology Workshop, Winnipeg, MB

1999 Arnold Naimark Young Investigator Award for excellence in postdoctoral research by a
Canadian in the field of Cardiovascular Research

1996-98 Manitoba Health Research Council (MHRC) postdoctoral fellowship.

1996 University of Manitoba postdoctoral fellowship.

1994 NSERC/DFO Science Subvention Grant (Awarded to Dr. J.F. Klaverkamp on the basis of
my written Ph.D. proposal)

1993 Freshwater Institute graduate studentship