Peer Review of *U.S. Fish and Wildlife Service Review of Lead Exposure and Effects to Scavenging Birds*

April 2014

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FORWARD

Atkins, North America, hereafter referred to as Atkins, and their subcontractor RESPEC, were retained by the U.S. Fish and Wildlife Service (USFWS) to facilitate an independent scientific review of the USFWS Environmental Contaminants Program's report on lead exposure to scavenging birds. Atkins believes the peer reviewers have successfully met the USFWS charge for their review and offer substantive suggestions for improving the quality and strengthening the scientific foundation of the draft report. Reviewer comments are focused on seven questions related to the report's objectives; conclusions; use of best available science; and strength of its scientific foundations, as well identification and characterization of uncertainties and their implications. In addition, reviewers adhered to instructions to avoid discussion of policy-related issues. Overall, the peer reviewers found the draft report to be a useful review on the topic of the effects of lead exposure to scavenging birds, with a strong scientific foundation, and they provide many opportunities to further strengthen the report's conclusions.

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1.0 INTRODUCTION

1.1 Background

The science regarding the exposure and subsequent effects of lead in the environment to birds, particularly species that scavenge on hunter-killed carcasses or offal piles, has received considerable attention from those examining the contribution of lead-containing ammunition. In response, the U.S. Fish and Wildlife Service (USFWS) has undertaken an evaluation of the scientific uncertainty surrounding these issues to help elucidate the effects of lead ammunition to scavenging birds that are trust resources of the USFWS. The draft report synthesizes the existing data regarding exposure and effects of lead on scavenging birds and assesses the extent to which ammunition is a contributing factor. Given the long-term conservation implications of this report and its influential information, it requires a formal, external, independent scientific peer review before distribution. If the report does not provide the best science-based information and analyses, any decisions or conservation actions based on this report may be less effective in the long-term conservation of migratory birds, bald and golden eagles, and endangered species such as the California condor.

1.2 Purpose and Scope of Peer Review

The purpose of this review is to provide a formal, independent, external scientific peer review of the information in the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* report. The report synthesizes the existing data regarding exposure and effects of lead on scavenging birds and assesses the extent to which ammunition is a contributing factor. Where available, the report relied on peer-reviewed literature to help answer questions of scientific uncertainty, and also incorporated selected cases of unpublished or grey literature that filled a significant data gap where peer-reviewed sources were not available. Peer reviewers were charged with reviewing this approach and assessing the sufficiency of the report's conclusions regarding outstanding questions of scientific uncertainty and the contribution of ammunition to lead exposure in the species under consideration.

The USFWS asked the reviewers to address the scientific merit of the report's technical review, which provides the basis for its conclusions regarding the effects of lead exposure in scavenging birds from ammunition sources. The reviewers were instructed to ensure that any scientific uncertainties are clearly identified and characterized, and the potential implications of the uncertainties for the technical conclusions drawn are clear. Peer reviewers were advised that they are not to provide advice on policy-related issues and should focus their review on identifying and characterizing scientific uncertainties.

Specifically, the USFWS requested that reviewers consider and respond to the questions listed below, at a minimum, in their reviews.

1. Are the objectives of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* clearly stated and logical? Is the content of the report within the parameters of these objectives? If not, please identify the specific objectives that are unclear or illogical, or where content has strayed from the stated objectives.

2. Do the authors of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* draw the correct conclusions for each section of the report (see Discussion Points boxes), and are they supported by the material presented within that section? If not, please identify those that are not and the specifics of each situation.

3. Do the authors of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* draw reasonable and scientifically sound conclusions from the scientific information presented in the report? Are there instances in the report where a different but equally reasonable and sound scientific conclusion might be drawn that differs from the conclusion drawn by the Service and is supported by data in the literature? If any instances are found where that is the case, please provide the specifics of that situation.

4. Does the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* base its interpretations, analyses and conclusions upon the best available science? If any instances are found where the best available science was not used, please provide the specifics of each situation.

5. Are there any seminal peer-reviewed scientific papers that the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* omits from consideration that would enhance the scientific quality of the document, or contribute to alternate conclusions that are scientifically sound? Please identify any such papers.

6. Are there other potential sources of lead contamination that are not addressed in the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* that should be considered as exposure routes for scavenging birds, and specifically in the assessment of condor recovery?

7. Is the scientific foundation of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* reasonable and how can it be strengthened? Please identify any options to strengthen the scientific foundations.

2.0 PEER REVIEW PROCESS

Atkins, North America, hereafter referred to as Atkins, and their subcontractor RESPEC, were retained by the USFWS to facilitate the peer review process. The terms of the contract include:

- organize, structure, lead and manage the scientific review;
- summarize the individual peer reviews and prepare a summary report for the USFWS;
- facilitate specific follow-up questions/answers between the USFWS and the reviewers, without attribution; and
- prepare and submit a final report and official record to the USFWS.

Atkins project manager Pam Latham, Ph.D., retained the services of RESPEC staff Rebecca Burns and Tom St. Clair, Ph.D., to assist in facilitation of this review (i.e., Atkins Team).

2.1 Selection of Reviewers

As part of its proposal, Atkins was required to submit a generic summary of the expertise and qualifications of three reviewers, along with two additional optional reviewers. Names and other identifying information were not included as this is an anonymous review. Atkins was instructed to determine the necessary experience and qualifications of the reviewers based on the content of the report to be reviewed. Suggested areas of expertise included toxicology, pathology and ecology. Ideally reviewers would have previously conducted similar reviews or regularly provided reviews of research and conservation articles for the scientific literature.

The Atkins Team applied a set of established criteria, including level of education (Ph.D. and/or D.V.M.), years of relevant experience (10+), number of peer-reviewed journal publications, including those related to lead and scavenging birds/wildlife. The Atkins Team screened each candidate for potential conflicts of interest and made sure the final composition was balanced in terms of expertise, affiliation and regional representation. The USFWS awarded the contract for five reviewers, so the Atkins Team confirmed five individuals who met the criteria listed above and were willing and available to participate in the review. The reviewers and their areas of expertise are listed below in alphabetical order; their resumes/CVs are included in Appendix A:

- Dr. John E. Elliott, wildlife eco-toxicology
- Dr. W. Grainger Hunt, avian biology
- Dr. Philip N. Smith, environmental toxicology
- Dr. Noel F.R. Snyder, avian biology
- Dr. Scott D. Wright, wildlife pathology

2.2 Document Review and Report Development

Upon award of the contract, the Atkins Team (Pam Latham, Ph.D., Tom St. Clair, Ph.D., and Rebecca Burns) participated on a call with USFWS technical representative Nancy Golden to discuss the scope of the review and address any questions. Dr. Golden provided additional materials to distribute with the review, including copies of the references that were examined in greater depth by the report authors as well as links to all of the unpublished/grey literature not already contained within the draft report. Dr. Golden also reviewed the draft scope of services for the peer review prior to its distribution.

On January 31, 2014 the Atkins Team held a conference call with the reviewers to describe the scope of services, including the charge to the reviewers and peer review schedule, and answer any questions. Following the call the draft report and additional materials were distributed to all of the reviewers and the independent desk reviews commenced.

Reviewers submitted their individual review comments to the Atkins Team by February 28, 2014. All attribution was removed and replaced with a number based on the order in which their reviews were received (i.e., Reviewer 1, Reviewer 2, etc.). The compiled individual reviews are included in this document as Appendix B. In the Results section, the Atkins Team summarized the responses to the seven questions posed to the reviewers. Additionally, two reviewers (Reviewer 2 and Reviewer 5) submitted versions of the PDF of the draft report that include their

comments and edits. Reviewer 2 referred substantially to comments in the edited version, so it is included as Appendix C to this report. Reviewer 5 submitted the edited version as a supplement to the individual review comments and the Atkins Team provided it to Dr. Golden separate from this peer review summary report. The draft peer review report was submitted to the USFWS for review and comment on March 7, 2014.

On March 28, 2014, Dr. Golden responded that the report authors were pleased with the peer review report and did not have any clarifying comments or requests of the reviewers, so the peer review report could be finalized.

3.0 RESULTS

The five reviewers provided detailed comments and insights on a wide array of topics relative to their charge and the questions they were posed. In general, the reviewers praised several aspects of the report, including its detailed descriptions; clear establishment of cause and effect between spent ammunition in carcasses and offal and lead exposure in scavenging birds; comprehensiveness and completeness; critical assessment of the available data; and organization (though one reviewer commented that the report tends to wander and did not find it to be well organized). Overall the reviewers found the report to be a useful review with a strong scientific foundation.

As part of their charge, reviewers were instructed to assess the sufficiency of the report's conclusions regarding outstanding questions of scientific uncertainty. All reviewers except one commented that the authors do not specifically indicate the uncertainties or evaluate their potential implications. Reviewers recommended that the scientific uncertainties the report is intended to address, as well as remaining data gaps, be clearly stated and described in order to meet the report's objectives and improve the overall flow of the document. Specific recommendations included listing uncertainties at the beginning of each relevant section then addressing them with available data and information, and/or devoting a section at the end of the report that lists and explains gaps in knowledge.

All reviewers but one also had concerns about the report's objectives statement and adherence to those objectives. Question 1 below specifically deals with objectives, but, in summary, reviewers recommended that the objectives be specified, clarified and inclusive of the major topics covered by the report (i.e., include alternatives to lead ammunition as part of objectives). Alternatively, the report could be revised to exclude conclusions not specifically linked to the objectives. As noted below, one reviewer revised the objectives statement to be more comprehensive and specific.

Below are brief summaries of the individual reviewers' responses to the seven questions posed by the USFWS. This section is not intended to be a comprehensive summary, but rather attempts to capture some of the primary comments in each reviewer's response to the individual questions, as well as any themes that emerged or comments that were raised by more than one reviewer independently. For the reviewers' full comments see Appendix B and Reviewer 2's edited version in Appendix C.

Question 1: Are the objectives of the USFWS Review of Lead Exposure and Effects to Scavenging Birds clearly stated and logical? Is the content of the report within the parameters of these objectives? If not, please identify the specific objectives that are unclear or illogical, or where content has strayed from the stated objectives.

All reviewers except Reviewer 3 raised issues with both the statement of the objectives and the adherence of the report's content to those objectives. Specifically, Reviewer 2 noted that the objectives are "broad and vague," whereas Reviewer 5 found them to be "somewhat indirect." Reviewer 2 suggested significant revision of the objective statement to help guide and focus the report, which lacks clear direction and is repetitive in numerous instances. Several reviewers questioned whether the report meets its stated objectives and/or includes content that goes beyond the objectives. Reviewers 1, 2, 4 and 5 observed that, though an objective of the report is to "address questions regarding scientific uncertainty," the document does not clearly identify those uncertainties or discuss their implications for the report's conclusions. This is one of the central themes that emerged from the reviewers' comments, as noted above.

Additionally, several reviewers noted content that appears to stray from the report's objectives, to varying degrees. Reviewer 1 found that the two case reports do not provide much additional support to the stated objectives and suggested the authors rethink their value. Reviewer 2 commented that the report includes an inordinate amount of information on the ban on lead shot in waterfowl hunting and is repetitive in its discussion of Great Lakes bald eagles, but in neither case does the report clearly state the lessons learned or take home message. Both reviewers 2 and 4 found information on lead alternatives (e.g., sections on "Bioavailability of ammunition to birds" and "Toxicity of alternative metals") to be outside the scope of the stated objectives and Reviewer 4 recommended it be explicitly stated in the objectives or removed from the report. Reviewer 5 offered a more complete rewording of the objectives statement which specifically includes alternatives to lead ammunition as one of the focus areas.

Question 2: Do the authors of the USFWS Review of Lead Exposure and Effects to Scavenging Birds draw the correct conclusions for each section of the report (see Discussion Points boxes), and are they supported by the material presented within that section? If not, please identify those that are not and the specifics of each situation.

With the exception of Reviewer 3, the reviewers found that, for the most part, the conclusions drawn for each section of the report were correct; however, there are specific examples of overstatements, misleading statements and instances where conclusions are not supported by the data presented in the section. For example, reviewers 1, 4 and 5 all identified the first bullet in the Discussion Points box on page 19 ("Lead toxicity to birds is extremely well understood…") as being an overstatement and offered recommendations for rephrasing. Both reviewers 1 and 4 pointed out several uncertainties that are either insufficiently discussed or omitted from the conclusions and should be included.

Additionally, reviewers 1, 2 and 4 noted numerous instances where the authors draw conclusions that are not sufficiently supported by the data presented in the section. For example, reviewers 1 and 2 both noted that the last bullet in the Discussion Points box on page 30 (regarding group feeding behavior enhancing vulnerability to lead exposure) would benefit from additional

discussion and clarification as the conclusion is not clearly established by the section. Additionally, reviewers 1 and 2 commented on the last bullet in the box on page 43 (regarding clinical signs of diagnostic lead poisoning); both noted that this point is not well supported by the section and needs further discussion.

Question 3: Do the authors of the USFWS Review of Lead Exposure and Effects to Scavenging Birds draw reasonable and scientifically sound conclusions from the scientific information presented in the report? Are there instances in the report where a different but equally reasonable and sound scientific conclusion might be drawn that differs from the conclusion drawn by the Service and is supported by data in the literature? If any instances are found where that is the case, please provide the specifics of that situation.

Reviewers 1 and 3 found no issue with the authors' interpretations of the literature or the report's conclusions.

Reviewer 2 identified several instances in which conclusions are not reasonable, scientifically sound or supported by the information presented; these specific instances are identified in the reviewer's marked-up version (see Appendix C). For example, Reviewer 2 made several comments regarding lead absorption into tissues, noting that throughout the report the authors oversimplified this process. The reviewer also questioned whether data are available to support the conclusion that complete absorption following ingestion is possible (page 43).

Reviewer 4 examined each individual conclusion, as stated on pages 86 – 87, and noted the relative strength of each. The reviewer raised concern with two conclusions: evidence for scavenging birds' relative sensitivity to lead in controlled dosing studies and the conclusions drawn on measures to reduce lead exposure. Regarding the first point (sensitivity to lead), Reviewer 4 does not believe good comparative data from controlled dosing studies are available to conclude that scavenging birds are relatively more sensitive to lead. On the second point (measures to reduce exposure) Reviewer 4 reiterated that this topic is not included in the report's objectives and it is comprehensively discussed, so it should either be addressed more thoroughly or removed from the report.

Reviewer 5 honed in on the authors' implied support of the argument that, without complete removal of lead ammunition from California condor habitat, the frequency of exposure in the population could remain high. The reviewer recommended that the authors reexamine the results of Green et al. (2009) regarding the effect of multiple exposures on lethality and include some of these points and/or modify existing statements.

Question 4: Does the USFWS Review of Lead Exposure and Effects to Scavenging Birds base its interpretations, analyses and conclusions upon the best available science? If any instances are found where the best available science was not used, please provide the specifics of each situation.

Reviewers 1 and 5 asserted that the authors based their interpretations, analyses and conclusions on the best available science, with Reviewer 5 noting that deeper and clearer explanations are needed.

Reviewer 2 also agreed, but pointed out two exceptions, one of which is significant. The reviewer stated that the report does not describe the mechanistic foundation(s) associated with the neurological and neuromuscular effects of lead exposure or the heightened sensitivity of developing organisms. Reviewer 2 also includes the citations of a few additional peer-reviewed published papers for consideration.

Reviewer 3 commented that the California condor case study does not accurately portray the history of how the lead issue came to the forefront. The reviewer provides a more complete summary along with several references, in addition to the Grantham (2007) paper already cited.

Reviewer 4 lists a number of studies in North America and Europe that are relevant to the subject report, noting a few particularly pertinent papers and bodies of work (i.e., Peterson et al. 2001; Elliott et al. 2008; and studies of lead in the white-tailed sea eagle in Germany).

Question 5: Are there any <u>seminal</u> peer-reviewed scientific papers that the USFWS Review of Lead Exposure and Effects to Scavenging Birds omits from consideration that would enhance the scientific quality of the document, or contribute to alternate conclusions that are scientifically sound? Please identify any such papers.

Reviewers 1, 2, 3 and 4 all recommended additional peer-reviewed scientific papers to enhance the quality of the report; however, only Reviewer 4 identified one additional paper considered to be "seminal," along with two other, more recent papers that may prove seminal. These citations are included in the individual comments found in Appendix B.

Question 6: Are there other potential sources of lead contamination that are not addressed in the USFWS Review of Lead Exposure and Effects to Scavenging Birds that should be considered as exposure routes for scavenging birds, and specifically in the assessment of condor recovery?

The reviewers' responses to this question varied significantly. Reviewer 1 found the report to be comprehensive in terms of its coverage of potential sources of lead contamination. Reviewer 2 stated that the authors tend to be "overly conservative" when acknowledging sources of lead exposure to scavenging birds other than ammunition. The reviewer commented that, although there is the potential for other unimagined sources, a more definitive statement is warranted, particularly with respect to condors. Reviewer 3 raised the possibility of lead in batteries as a potential source and suggested a brief discussion be added. Reviewer 4 mentioned mining and smelting operations as a potential source, citing a study of osprey in the Coeur d'Alene area of Idaho. The reviewer also noted studies on mining as a source of lead in passerine birds, which demonstrates movement of lead from mining and smelting into wildlife food chains. Finally, Reviewer 5 stated that varmint shooting is a common practice in the southwest U.S. and animals left in the field could be a source of a "great deal of consumable lead" to scavenging birds. The reviewer recommended that the specific bullets used for varmint shooting and the implications of this practice for scavenging birds be discussed more thoroughly in the report.

Question 7: Is the scientific foundation of the USFWS Review of Lead Exposure and Effects to Scavenging Birds reasonable and how can it be strengthened? Please identify any options to strengthen the scientific foundations.

On the whole, the reviewers found the scientific foundation of the report to be reasonable and offered options to strengthen it. Reviewer 1 found it difficult to comment on this question given that the report summarizes other studies and does not present original data or analyses.
Reviewer 2 found the foundation of the report to be "solid, sound and reasonable," with the notable exception of the discussion of neurotoxic effects (see Question 4). The reviewer also noted numerous opportunities to improve the report through organization, clarification, further explanation and inclusion of additional information (see appendices B and C).

The primary recommendation from Reviewer 4 was to broaden the spatial scope of the literature considered in the report to capture the continent-wide nature of the problem. The reviewer offered specific ideas for broadening the scope, including a tabular and/or graphic summary of studies throughout the U.S. or all of North America.

Both reviewers 3 and 5 listed a number of distinct areas that could be added, revised or otherwise improved to strengthen the report (see Appendix B). Both reviewers brought up issues surrounding scavenging of large vs. small mammalian bones, as well as ingestion and regurgitation of fur and implications for lead exposure, indicating this is an area the authors may wish to reexamine. Reviewers 3 and 5 also clarified that DDT/DDE is a reproductive impairment, not a mortality factor. Finally, both reviewers 1 and 5 indicated that lead-related deaths are likely underestimated, an uncertainty not sufficiently acknowledged in the report.

4.0 REFERENCES

The following references were cited in Section 3.0 above. The citations for numerous other references recommended by the reviewers are included in their individual comments in Appendix B.

Elliott, J.E., A. Birmingham, L.K. Wilson, M. McAdie, and P. Mineau. 2008. Fonofos poisons raptors and waterfowl several months after labeled application. *Environmental Toxicology & Chemistry*. 27:452-460.

Grantham, J. 2007. Reintroduction of California condors into their historic range: The recovery program in southern California. In Mee A, Hall LS, eds, California Condors in the 21st Century. Nuttall Ornithological Club, Cambridge, MA, and American Ornithologists' Union, Washington, DC, pp 123-139.

Green, R., W.G. Hunt, C.N. Parish and I. Newton. 2009. Effectiveness of action to reduce exposure to free-ranging California condors in Arizona and Utah to lead from spent ammunition. In RT Watson, M Fuller, M Pokras, G. Hunt, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA, pp 240-253. Available from: http://www.peregrinefund.org/subsites/conferencelead/PDF/0218%20Green.pdf.

Peterson, C.A., S.L. Lee and J.E. Elliott. 2001. Scavenging of waterfowl carcasses by avian predators in the Fraser River delta of British Columbia. *Journal of Field Ornithology* 72: 150-159.

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5.0 APPENDICES

Appendix A: Individual Reviewer Comments
Appendix B: Reviewer 2 Edited Version of Draft Report
Appendix C: Reviewer CVs/Resumes

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APPENDIX A: REVIEWER CVS/RESUMES

(Note: references highlighted in yellow were identified in the reviewer's COI disclosure agreements)

JOHN E. ELLIOTT

CURRICULUM VITAE

January 30, 2014

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PRESENT POSITION: Research Scientist, Ecotoxicology
Science & Technology Branch
Environment Canada
Pacific and Yukon Region

Concurrent Appointments:

- Adjunct Professor, Faculty of Land and Food Systems, University of British Columbia, 1996-present.
- Adjunct, Professor, Biological Sciences, Simon Fraser University, 2003-present.

EDUCATION:

BSc (hons)	1979	Carleton University, Canada; First Class Honours, Integrated Science (Biology major)
MSc	1989	University of Ottawa, Canada; Biology (Toxicology)
PhD	1995	University of British Columbia, Canada; Animal Science/ Pharmaceutical Science

RESEARCH INTERESTS

- Wildlife ecotoxicology:
  - Effects of contaminants (persistent organic pollutants, heavy metals) on top predators (raptorial birds, waterbirds, seabirds, aquatic mustelids);
  - Effects of rodenticides and anticholinesterase compounds on birds;
  - Agricultural pollutant effects on amphibians.
- Patterns, trends and source determination of pollutants.
- Early developmental toxicology of persistent contaminants.
- Ecology of raptors.

PROFESSIONAL EXPERIENCE:

Research Scientist, Ecotoxicology	2006 - present	Science & Technology Branch, Environment Canada, Pacific Wildlife Research Centre, Delta, BC, Canada
Research Scientist, Ecotoxicology	1996 - 2005	Canadian Wildlife Service, Pacific Wildlife Research Centre, Delta, BC, Canada
Acting Head, Ecosystem Research	1999 - 2002	Canadian Wildlife Service, Pacific Wildlife Research Centre, Delta, BC, Canada
Research Biologist, Toxicology	1989-1995	Canadian Wildlife Service, Pacific Wildlife Research Centre, Delta, BC, Canada
Environmental Contaminants Evaluator	1986-1989	Canadian Wildlife Service, National Wildlife Research Centre, Ottawa, ON, Canada
Monitoring Program Biologist	1981-1986	Canadian Wildlife Service, National Wildlife Research Centre, Ottawa, ON, Canada

Data Analysis Officer	1979-1981	Water Quality Branch, Environment Canada, Ottawa, ON, Canada
Environmental Technician	1977	ESCO Environmental Services, Calgary, AB, Canada

TEACHING:

Postgraduate:

- External examiner for PhD defence of Marie Noel, University of Victoria, "Bioaccumulative contaminants in marine mammals: uptake and effects", March 2013.

- Taught graduate level course (BISC 655), *Wildlife Toxicology*, Simon Fraser University (winter 2013).

- Thesis co-supervisor for MET student, Maria Yu, on an investigation of early developmental effects of mercury in birds. Simon Fraser University, Burnaby, BC. (2013-).

- Thesis co-supervsor for MSc student, Andrew, Huang, on population genetics of the barn owl in western North American and genetic variation in the barn owl VKOR gene. University of British Columbia, Vancouver, BC (2012-).

- Thesis co-supervisor for MET student, Alexandra Reers, on investigation of organic flame retardant effects on endocrine endpoints. Simon Fraser University, Burnaby, BC. (2012-).

- Supervisor for post-doctoral fellow, Margaret Eng, on toxicology of replacement flame retardants in avian systems. Simon Fraser University (2012-2014).

- Departmental Seminar, Wildlife Ecotoxicology: Old and new contaminant threats to terrestrial wildlife. Natural Resources Research Institute, University of Minnesota, Duluth, MN, Oct 2011.

- Thesis co-supervisor for MET student, Lillian Wong, on an investigation of in vitro screening of the DDT metabolite DDE on endocrine endpoints. Simon Fraser University, Burnaby, BC. (2011-).

- Thesis co-supervisor for MSc student, Jason Brogan, on an investigation of sources and effects of persistent contaminants in Cooper's Hawks nesting in an urban environment, Simon Fraser University (2011-).

- Taught graduate level course (BISC 655), *Wildlife Toxicology*, Simon Fraser University (winter 2011).

- External Examiner, PhD defence, Juan Jose Alava Saltos, "POPs in wildlife from Ecuador and the Galapagos". Simon Fraser University (Jan 2011).

- Taught post-graduate course in *Wildlife Ecotoxicology*, University of Coimbra, Coimbra, Portugal (June 2010).

- Faculty Opponent, PhD defence, Asa Berglund, “Responses to reduced industrial metal emissions: an ecotoxicological study on pied flycatcher (*Picedula hypoleuca*), University of Umea, Umea, Sweden (2010).

- Taught post-graduate course in *Wildlife Ecotoxicology*, University of Coimbra, Coimbra, Portugal (July 2009).

- Thesis co-supervisor for PhD student, Heidi Scherr, on an investigation of the effects of brominated flame retardants on early developmental process in birds. Simon Fraser University (2009-).

- Thesis co-supervisor for MSc student, Caitlin Nelson, on use of DNA genotyping to assess the exposure of river otters to persistent contaminants. University of British Columbia (2009-2012).

- Thesis co-supervisor for MSc student, Mikaela Davis, on the spatial variation in diet and feeding ecology of the glaucous-winged gull. Simon Fraser University (2009-2013).

- Thesis co-supervisor for MSc student, Victoria Khamzina, on effects of egg injected PBDE-99 on growth and development of the zebra finch. Simon Fraser University (2009-2011).

- Thesis committee member for PhD student, Louise Blight, on the ecology of the glaucous-winged gull. University of British Columbia (2008-2012).

- Taught graduate level course (BISC 655), *Wildlife Toxicology*, Simon Fraser University (2009).

- Thesis co-supervisor for M.Sc. student, Kirstin Webster, on effects of anti-coagulant rodenticides on clotting processes in birds. Simon Fraser University (2008-2010).

- Thesis co-supervisor for PhD student, Margaret Eng, on early developmental effects of PBDEs in avian systems. Simon Fraser University (2007-2013).

- Thesis committee member for M.Sc. student, Sophi Hindmarsh, on ecology of barn owls in the Fraser River Delta, Simon Fraser University (2007-2009).

- Thesis committee member for PhD. student, Sarah Lord, on effects of chlorinated hydrocarbon in alpine ecosystems on avian wildlife. University of Alberta (2006-).

- Thesis co-supervisor for M.Sc. student, Dan Guertin, on non-invasive techniques to investigate population ecology and contaminant exposure of river otter. Simon Fraser University (2005-2009).

- Thesis committee member for M.Sc. student, Iain Jones, on interactions between bald eagles and great blue herons on south coast of B.C. Simon Fraser University (2005-2009).

- Thesis committee member for M.Sc. student, Alexandra de Jong-Westman, on the effects of orchard pesticides on reproduction of native amphibians. University of British Columbia (2006-2008)

- External Examiner, PhD Defence, Elizabeth Mos. “Physiological and Molecular Biomarkers of Environmental Contaminant-Associated Immunotoxicity in Harbour Seals (*Phoca vitulina*)”. University of Victoria (2006).

- Thesis co-supervisor for M.Sc. student, Courtney Albert, on effects of monosodium methane arsonate (MSMA) on a model songbird, Simon Fraser University (2004-2006).

- Supervisor, post doctoral fellow, Christy Morrissey, on effects of monosodium methane arsonate (MSMA) on cavity nesting birds, University of British Columbia (2003-2006).

- Thesis co-supervisor for M.Sc. student, Lilly Cesh, on contaminant dynamics and effects in bald eagles, Simon Fraser University (2002-2005).

- Member of supervisory committee for PhD. student, Paula Enrique, on the distribution, abundance, and genetic variation of the Bearded Screech Owl in the highlands of Chiapas, Mexico, University of British Columbia (2001-2007).

- Taught graduate level course (ANSC 530), *Selected Topics in Wildlife Toxicology*, University of British Columbia (2000-).

- Thesis co-supervisor for M.Sc. student, Patti Dods, on effects on nonyl-phenol and other chlorinated hydrocarbon contaminants on reproductive success of tree swallows nesting at a sewage treatment plant, Simon Fraser University (2000-2002).

- Thesis co-supervisor for M.Sc. student, J. Christensen, on the effects of environmental contaminants on metamorphosis in *Rana catesbeiana* and sperm motility in *Xenopus laevis*, University of British Columbia (2000-2002).

- External examiner for M.Sc. thesis defence of W. Simms on the effects of chlorinated hydrocarbon contaminants on harbour seals, University of Victoria (Sept 1999).

- Thesis co-supervisor for PhD. student, S. Weech, on mercury in fish and fish-eating birds, with special reference to the Pinchi Lake region of British Columbia, Canada, University of British Columbia (1999-2003).

- Thesis co-supervisor for M.Sc. student, H. Gill, on the effects of orchard pesticides on songbirds, Simon Fraser University (2000-2003).

- Thesis co-supervisor for PhD. student, C. Morrissey, on the ecology and ecotoxicology of American dippers in the Chilliwack River watershed, Simon Fraser University (1998-2003).

- Thesis co-supervisor for M.Sc student, E. Birmingham, on the effects of endocrine disrupting compounds on avian species in the Georgia Basin, Simon Fraser University (1998-2001).

- Thesis co-supervisor for M.Sc. student, R. Loveridge, on the effects of endocrine disrupting compounds on amphibians in the lower Fraser Valley, Simon Fraser University (1998-2002).

- Member of supervisory committee for PhD student, L. Smith, on effects of endocrine disrupting chemicals on behaviour of songbirds, University of British Columbia (1997-2003).

- Thesis co-supervisor for an M.Sc. student, C. Gill, on the influence of contaminants and food supply on reproduction of Bald Eagles on south-east Vancouver Island, Simon Fraser University (1995-1998).

- Thesis co-supervisor for an M.Sc. student, K. Pettit, on effects of agricultural pollutants on amphibians in the lower Fraser Valley, University of British Columbia (1995-1998).

- Thesis co-supervisor for M.Sc. student, G. Howald, on risk to wildlife from use of brodifacoum to control rats on a seabird colony, University of British Columbia (1994-1997).

- Occasional guest lecturer for graduate courses in environmental toxicology, Simon Fraser University (1995-present).

<u>Undergraduate teaching experience:</u>

Occasional guest lecturer at Simon Fraser University, Department of Biological Sciences for undergraduate courses in environmental toxicology (1991-present).

AWARDS

2013 **Queen Elizabeth II Diamond Jubilee Medal** In recognition for my contributions to Canada, specifically to the conservation of wildlife from the science and regulation of environmental toxicants.

2012 **Presidential Citation for Exemplary Service,** SETAC North America 33rd Annual Meeting. Long Beach CA, Nov 11-15 2012.

2011 **Salish Sea Science Prize**, University of California, Davis CA, Seadoc Society. "*For research on the exposure and effects of persistent contaminants on avian and marine top predators in that marine system and*

its adjacent terrestrial and aquatic drainage, and working with regulatory and non-regulatory processes to reduce the impacts of such contaminants."

2011 **National Citation of Excellence**, Environment Canada, Teamwork, Partnering and Collaboraton. "*For outstanding contribution to the collaborative teamwork of the Chemical Management Plan Science Team."*

2010 **Government Service Award**, Society of Environmental Toxicology and Chemistry (SETAC). "*For demonstrating exemplary dedication and service toward promoting the application of environmental toxicology and chemistry to risk assessments with respect to key issues of government policy*."

2008 **National Citation of Excellence**, Environment Canada, Teamwork, Partnering and Collaboration "*For your outstanding work in identifying the threat to migratory birds by the toxic insecticide MSMA (monosodium methanearsonate) used to fight the Mountain Pine Beetle infestation, and for tackling many other complex science issues in past years*".

2002 **National Citation of Excellence**, Environment Canada for: *Outstanding Performance in the Field of Environmental Toxicology*

1994 **Award of Excellence**, Environment Canada for: *Pesticide monitoring, research and extension work.*

1983 National Bureau of Standards (USA) **travel grant**.

1979 Carleton University, Undergraduate, **First Class Honours**.

1979 **Steinberg Scholarship** for academic excellence.

1972 **Ontario Scholar**.

1972 Carleton University **Entrance Scholarship**.

1972 Canadian Forces Europe Scholarship, **Top All-round Scholar**.

RESEARCH GRANTS AND CONTRACTS

- Development of an adverse outcome pathway (AOP) approach to examine the toxicity of novel flame retardants to birds. Chemical Management Plan. $ 23,500 per annum (2011 -).

- Investigating sources, transport, accumulation and effects of persistent contaminants in urban environments using a top predator as indicator. National Sciences and Engineering Research Council of Canada (NSERC). $ 125,000 (2011-2015).

- Investigating the effects of mercury on the early development and later behaviour of birds. Canadian Atmospheric Regulatory Agenda (CARA). $ 125,000 (2012-2017).

- Application of genomics technologies to understanding population ecology of threatened and endangered species. Environment Canada Species at Risk Program (SARA). $ 105,000 (2013-2016).

- Ecology of SARA listed owls in support of risk assessments of anti-coagulant rodenticides. Pesticide Science Fund III, $ 129,000, (2009-2012).

- Validation of fecal DNA genotyping for assessment of contaminated sites using radio telemetry. STAGE (Strategic Application of Genomics Technology). $ 91,000 (2009-2011).

- Development and validation of an integrated avian laboratory and field model system for characterizing the effects of priority chemicals on early developmental processes in avian species. Canadian Federal Chemical Management Plan Research. $ 162,000 (2008-2011).

- Terrestrial Effects Research. Jointly with C. Bishop and P. Mineau, $ 310,500. Pesticides Science Fund II, Environment Canada (2006-2009).

- Impacts on Amphibians. Jointly with C.A. Bishop, $ 96,000. Pesticides Science Fund, Environment Canada (2006-2009).

- Effects of contaminants, particularly PCBs, on river otters in and around Victoria Harbour, B.C., jointly with A. Harestad, $ 94,000, Habitat Conservation Trust Fund, British Columbia (2006-2008).

- Impact of the use of MSMA (monosodium methanearsonate) for bark beetle control on cavity nesting birds in British Columbia forests. $ 126,000 from the Pesticides Science Fund, Environment Canada (2003 -2006).

- Assessment of pesticide exposure and effects on amphibians and snakes using agricultural habitat, South Okanagan, British Columbia. $ 111,000 from the Pesticide Science Fund, Environment Canada (2003 – 2006).

- Raptor and waterfowl exposure to pesticides in agricultural ecosystems of southwestern British Columbia. With L.K. Wilson, $ 120,000 from the Pesticides Science Fund, Environment Canada (2003 – 2004).

- Effects of mercury on fish-eating wildlife in the region of the Pinchi Fault, British Columbia. $ 60,000 from Cominco Inc. (2000-2003).

- Factors influencing domestic and international sources of chlorinated hydrocarbons to fish and osprey in B.C. $ 259,000 from the Toxic Substance Research Initiative (1999 - 2002).

- Effects of orchard pesticides on terrestrial and aquatic life. $ 27,000 from the Toxic Substances Research Initiative (1999 - 2002).

- Impact of endocrine disrupting substances on amphibian health in agricultural ecosystems. $ 75,000 from the Toxic Substances Research Initiative (1999 - 2002).

- Effects of endocrine disrupting compounds on wildlife of the Georgia Basin. $ 240,000 from the Georgia Basin Ecosystem Initiative (1998 - 2002).

- Persistence of granular insecticides in Fraser Delta soils and implications for wildlife. Jointly with Dr. Suny Szeto, Agriculture Canada, Pacific Research Station, $ 30,000 from the Pesticide Management and Review Agency, Health Canada, and $ 20,000 from Agriculture Canada, Pacific Region (1994 -1997).

- Secondary poisoning of birds of prey in south-western British Columbia, $ 20,000, British Columbia Ministry of Environment, Lands and Parks, Integrated Program Branch (1994 – 1996).

- Risk to wildlife from use of brodifacoum to control rats on the Langara Island seabird colony. $ 110,000, The Nestucca Fund (1994 – 1995).

- Persistent pollutants in arctic seaducks wintering on the British Columbia Coast, Jointly With Dr. Birgit Braune, National Wildlife Research Centre, $ 16,000, Green Plan Arctic Initiative (1993 - 94).

- Pesticide effects on California Quail and American robins using orchards in the Okanagan Valley of British Columbia. Jointly with Dr. Kim Cheng, Animal Science, University of British Columbia, $ 30,000, Wildlife Toxicology Fund (1993 - 1994.

- Effects of granular insecticides on wildlife in the lower Fraser Valley of British Columbia. $ 40,000, Pest Fund, Environment Canada (1991-1992).

- Risk to fisheries and wildlife of pesticide use on golf courses in British Columbia. Jointly with Dr. Lee Nickl, Department of Fisheries and Oceans, New Westminister, $ 30,000, Pest Fund, Environment Canada (1990-91).

PROFESSIONAL ACTIVITIES

Review of manuscripts

Archives of Environmental Contamination & Toxicology (33)
Auk (3)
Colonial Waterbirds (3)
Comparative Biochemistry and Physiology (3)
Condor (2)
Ecological Applications (2)
Ecotoxicology (5)
Environment International (2)
Environmental Health Perspectives (5)
Environmental Pollution (18)

Environmental Monitoring and Assessment (5)
Environmental Science & Technology (5)
Environmental Toxicology & Chemistry (24)
Ibis (1)
International Journal of Great Lakes Research 92)
Journal of Applied Ecology (3)
Journal of Environmental Monitoring (3)
Journal of Great Lakes Research
Journal of Raptor Research (5)
Journal of Toxicology and Environmental Health (2)
Journal of Wildlife Management (5)
Marine Ecology Progress Series (2)
Marine Environmental Research (3)
Marine Pollution Bulletin (2)
Northwestern Naturalist (2)
Pesticide Biochemistry and Physiology (1)
Plos One (3)
Polish Journal of Environmental Science (1)
Wilson Bulletin (2)
Book chapters (11)

Editorships and editorial Boards

2013-2014	Guest Editor, *Proceedings of the National Academy of Science*
2012-ongoing	Associate Editor, *Ecotoxicology*
2006-2009	Member of the editorial board of *Environmental Toxicology and Chemistry.*
2003	Hoffman, D.J., B.A. Rattner, G.A. Burton, Jr., J. Cairns, Jr. Handbook of Ecotoxicology, 2nd Edition
1994 - 2000	Member of the editorial board of *Environmental Toxicology and Chemistry.*

Review of research proposals

National Science and Engineering Research Council, Discovery Grants; member, Evolution & Ecology Evaluation Group, 2011-12.

The Canadian Wildlife Service Research Support Fund (5)
The Endangered Species Fund (Canada) (12)
The Hudson River Foundation (1)
North Pacific Science Fund (5)
UC Davis Wildlife Health Centre Marine Ecosystem Health Program (2)
The Wildlife Toxicology Fund (Canada) (5)

Professional Societies:

Elected member, North American Board of Directors, Society of Environmental Toxicologists and Chemists (2010-2013).

Member, Society of Environmental Toxicologists and Chemists (1987 - present).

Member, Raptor Research Foundation (1986-87, 1995-present).

Member, The Wildlife Society (1999-present)

Media Communications:

Interviewed on many occasions by regional, national and international media outlets: television (15), radio (16) and print media (30) on issues relating to environmental toxicology.

Examples: Boston Globe, CBC Nature of Things, CBC National News, CTV National News, Discovery Channel, Global Television National News, NKK Japan Television, Toronto Globe & Mail, New York Times, New Zealand Herald, Science (News Section), Vancouver Sun, Vancouver Province.

Public Communications:

An average of 6 presentations per year to: professional associations, natural history clubs, community groups and schools.

Consulting and Advice *(select examples)*

1981 - present	**National Wildlife Toxicology Committee (Canada)** - Member, Secretary, 1981-1989.
1982	**German Environment Ministry** - Site visits and advice on ecosystem monitoring and specimen banking using wildlife as indicator species.
1983	**National Bureau of Standards** and **EPA** (US) - Site visits and advice on ecosystem monitoring and specimen banking using wildlife as indicator species.
1984	**Inter-departmental Task Force on Cadmium** (Canada) - member, advice to other departments on Cadmium exposure and effects in wildlife.
1985	**Manitoba Department of Natural Resources** - advice on establishment of a wildlife contaminants monitoring program.
1985	**International Council for Exploration of the Sea (ICES)** - advice and report on use of marine birds for monitoring pollution of the oceans.

1988	**New York State Department of Environmental Conservation** - advice on monitoring of contaminants in wildlife, particularly contaminant concentrations in game birds.
1988 - present	**State of the Environment (Canada) reporting:** Provision of data and expert reviewer.
1991	**Washington State Department of Conservation** - advice on effects of persistent pulp mill pollutants on wildlife.
1994	**US Fish & Wildlife Service and State of California** - Expert review of report on - Injury of seabirds from DDT and PCB residues in the southern California Bight ecosystem.
1993 - present	**Multi-stakeholder working group on pesticide impacts on wildlife in the Lower Fraser Valley ("Wireworm Taskforce");** Members: Agriculture Canada, BC Ministry of Agriculture, BC Ministry of Environment, Delta Farmer's Institute, Delta Farmland and Wildlife Trust, Health Canada, Pesticide Management and Review Agency.
1995 - 1998	**US Fish and Wildlife Service, Olympia WA** - advice on monitoring of persistent contaminants in estuarine and marine wildlife.
1995 - present	**Federal-Provincial Bird Kill Contingency Committee,** member.
1995 - present	**Regional Toxic Chemicals Advisory Committee, Pacific & Yukon,** member.
1995 - present	**Federal-Provincial Interagency Pesticide Committee**, member.
1995 - present	**Regional Priority Substance List (PSL) Review Committee,** member.
1996	**Chinese Ministry of Agriculture, Ornithological Field Station -** Invited to Qingdo, China to advise on ecotoxicological problems in Northeast China deferred because of other time commitments. Advice and comments provided by mail.
1996	**National Biological Service, Corvallis OR** - Invited to act as external reviewer for Senior Research Scientist promotion documents.
1996	**Puget Sound-Georgia Basin International Working Group** - Invited to review monitoring proposals for wildlife and for contaminants.
1996	**North Pacific Marine Science Organization (PICES**) - Invited wildlife scientist and Canadian representative on a sponsored international study of effects of pollution in Jiaozhou Bay, China (declined due to schedule conflicts).

1997	**National Committee on Environmental Effects of Endocrine Disrupting Chemicals (Canada)** - Invited member for Pacific and Yukon Region.
1997	**Transport Canada** - advice on potential impact on the bald eagle population of the Squamish region of B.C. posed by a proposed airport development.
1997	**Korean Oceanographic Research Institute** - Invited to Seoul, Korea to advise and participate in research on contaminant effects on wildlife. (declined due to schedule conflicts)
1997	**Bolivian National Environment Ministry, Departments of Ornithology and Protected Areas** - Invited to advise and co-operate on issues relating to pesticides and wildlife - deferred pending development of regional strategy regarding participation in Latin American Program issues.
1997 - 1998	**Council of Environment Ministers (Canada) -** review of Tissue Residue Guidelines documents for 1) polychlorinated dioxins and 2) polychlorinated biphenyls (PCBs).
1998	**Peregrine Falcon Recovery Team** - Invited to serve as British Columbia delegate.
1998	**South African Agricultural Research Council** - Advice and collaboration on a study of non-target wildlife poisoning.
1998	**Heinrich-Pette Institute, Hamburg, Germany** - Invited to collaborate in research into avian sources of Hepatitis B virus, interaction with contaminants and role in autoimmune disease.
1999	**British Columbia Ministry of Environment Lands and Parks** - Review of “Draft criteria for managing contaminated sediment in BC”.
1999	**US Fish and Wildlife Service, Alaska** - advice on poisoning of birds of prey.
1999	**US Fish and Wildlife Service** - Review and advise on “Biological Opinion - Impact of Pulp and Paper Mill Effluents on Bald Eagles in Maine”.
2000	**Hawk Watch International** - Review of document “Research Priorities for Raptor Conservation”.
2000	**Industrial Economics, Cambridge MA and US Fish & Wildlife Service, Sacramento CA** - Expert review and advisor at expert witness deposition for report - Assessment of the Alleged Injury to Wildlife from DDT and its metabolites in the Southern California Bight ecosystem.

2000	**US Fish and Wildlife Service, Maine** - Review of biological opinion pertaining to bald eagles and issues of discharge permits to Maine's kraft pulp and paper mills.
2000	**Organization for Economic Cooperation and Development (OECD)** - Invited to contribute an article on 'Genotoxicity, cytotoxicity and ecotoxicity of pesticides' to an Encyclopedia of the Environment project.
2001	**Victoria Harbour Environmental Action Plan** - presentation and advice on effects of chlorinated hydrocarbons, particularly PCBs on river otter and other wildlife in the Harbour.
2001	**Fisheries & Oceans Canada** - Review of document on Southern resident killer whales at risk: toxic chemicals in the British Columbia marine environment.
2001	**Hudson River Foundation, Expert Panel on Effects of Contaminants on Avifauna of the Hudson River** - Invited member (declined due to schedule conflicts).
2001	**Stratus Consulting, Boulder CO and US Department of Interior** - Expert reviewer and panel member for report - Preliminary Injury Assessment for Avian Resources, Hudson River NRDA.
2001 (ongoing)	**U.S. Fish & Wildlife Service, Sacramento, CA** - Member of expert panel on the recovery of Bald Eagles on the Channel Islands of Southern California.
2002	**US Fish & Wildlife Service** - Review of document on contaminant monitoring of Ospreys in Oregon and the Pacific Northwest.
2002	**US Fish & Wildlife Service** - Review of document on exposure and effects of chemical contaminants on tree swallows nesting along the Housatonic River.
2002 -	**Teck-Cominco Technical Advisory Committee** - member, expert on contaminant effects of Trail smelter on wildlife.
2002	**Fisheries & Oceans Canada** - Review of document of global versus local contaminants signals in harbour seals.
2003	**US Fish and Wildlife Service, Maine** - Review of report on PCBs and toxic equivalents in bald eagle samples.
2003	**US Department of Interior, Albany, NY; New York State Attorney General, New York, NY; Stratus Consulting, Boulder CO –** expert reviewer and panel member for report on contaminants in Hudson River birds.

2004 **Review of Canadian Environmental Protection Act (CEPA)** Priority Substances List assessment of polybrominated diphenyl ethers (PBDEs).

2004 **US Department of Interior, Albany, NY; New York State Attorney General, New York, NY, and Stratus Consulting, Boulder, CO** – Preparation of proposal to investigate: Potential injury of PCBs to Belted Kingfishers and Spotted Sandpipers breeding along the Hudson River.

2004 **US Fish and Wildlife Service, Olympia WA** - review of report on contaminants in bald eagles on the Columbia River.

2004 - 2006 **US Department of Interior, Albany, NY; New York State Attorney General, New York, NY, and Stratus Consulting, Boulder, CO** – Review of data on potential injury of PCBs to Bald Eagles breeding along the Hudson River.

2004 **Workshop on mass balance of PCBs and PBDEs in the Strait of Georgia, Vancouver BC -** Invited speaker and participant, expenses paid.

2005 **Western Canadian Wildlife Rehabilitators Conference. Campbell River, B.C.** Invited speaker, panel member, all expenses paid.

2005 **US Fish and Wildlife Service,** Guest lecturer on pesticide impacts on birds of prey at Pesticide Training Course, Fort Worden, WA, expenses paid.

2006 **US Department of Interior, Albany NY; New York State Attorney General, New York, NY; Stratus Consulting, Boulder CO** – Review for Trustees: Investigations of PCBs in kingfisher, sandpiper and tree swallow on Hudson River.

2006 **New Mexico Office of the Natural Resources Trustee, Albuquerque, NM, Stratus Consulting, Boulder CO** – Data review, development of a reasonable worst case scenario model inputs for bird toxicity, presentation of results for: Impacts of acid mine drainage on migratory birds.

2006 **The Environment Agency, Wallingford, UK,** Review of: Investigations of heron deformities in North Nottinghamshire**.**

2007 **Wildlife Toxicology Workshop, sponsored by The Smithsonian Institute, March 13-15, 2007, Washington DC.** Invited participant (accepted) and plenary lecturer (declined due to time constraints), all expenses paid.

2007 **University of Cardiff, Wales, Department of Biology, May 27, 2007 -** Invited seminar (Reproductive dysfunction in wild birds).

2007 **Centre for Reproductive Biology, Uppsala University, Uppsala, Sweden, post graduate course in 'Reproductive Endocrinology in Vertebrates' Jun 3-8, 2007 -** Invited guest lecturer on 'reproductive dysfunction in wildlife'.

2008 **USGS, Upper Midwest Environmental Sciences Center, La Crosse WI -** Review of report on effects of PCBs on spotted sandpipers on the Hudson River, NY. June 2008.

2006-2008 **State of Colorado Attorney General, Denver CO; Stratus Consulting, Boulder, CO –** Review data prepare reports and prepare as expert witness for an assessment of injury to wildlife resources from contaminants at the Rocky Mountain Arsenal.

2009 **USGS, Upper Midwest Environmental Sciences Center, La Crosse WI -** Review of report of effects of PCBs on tree swallows and kingfishers on the Hudson River, NY.

2009 **University of Coimbra, Coimbra, Portugal**. Expert advice and site assessment for design of wildlife ecotoxicology study at Mina de Sao Domingos contaminated site.

2010 **Fisheries and Oceans Canada** – Invited speaker and participant in workshop, "Do sediment quality guidelines protect marine mammal health?" Mar 2009.

2010 **Cohen Commission on the Decline of the Fraser River Sockeye Salmon.** Appointed expert witness on avian predation.

2010 **Stratus Consulting, Boulder CO, on behalf of US National Oceanic Atmospheric Administration**. Review of biomarker options for monitoring injury to wildlife at the Gulf of Mexico oil spill.

2010 **University of Coimbra, Coimbra, Portugal.** Expert advice and site assessment for design of wildlife ecotoxicology study at contaminated coastal lagoons.

2011 **U.S. Department of Interior, Columbia, MO**. Member, expert panel for Natural Resource Damage Assessment and Restoration (NRDAR) of a contaminated site at Anniston, Alabama, and recommendation of protocols for assessing wildlife toxicity for the NRDA process.

2011 **U.S. Environmental Protection Agency, Duluth, MN**. Invited speaker and panel member, Special Session on Pesticides and Raptors, Raptor Research Foundation Meeting, Duluth MN.

2011 **U.S. Environmental Protection Agency, Washington, DC**, Member, Science Advisory Panel on: Notice of Intent to Cancel 21 domestic use rodenticide products.

2011-12 **National Science and Engineering Research Council (NSERC Canada)**, M ember, Ecology and Evolution Review Panel.

2012 **Leipniz Institute for Wildlife and Zoo Research**, Berlin, Germany. Invited seminar on Wildlife Ecotoxicology: early developmental effects in birds, site and field visit.

2012 **U.S. Geological Survey, Fort Collins, CO**. Invited participant, Western North America Mercury Synthesis. Canadian Representative on Tri-national Initiative.

2012 **PICES (The North Pacific Marine Science Organization)**. Invited speaker and panel member. Special Symposium: Environmental contaminants in marine ecosystems: Seabirds and marine mammals as sentinels of ecosystem health. Hiroshima, Japan, Oct 15-19, 2012.

2012 **California Department of Pesticide Regulation (DPR).** Peer review of an assessment of the hazard posed by second generation anticoagulant rodenticides to non-target wildlife.

2013 **Health and Environmental Sciences Institute (HESI), Washington DC**, Terrestrial Bioaccumulation Workshop, invited expert. Jan 8-10, 2013, Miami, FL.

2013 **US Department of Interior, Albany NY; New York State Attorney General, New York, NY; Industrial Economics, Harvard MA.** Expert advice on development of an avian injury determination plan.

Session chair, convener, workshop leader:

2014	Co-Chair, Planning committee. SETAC 2014. Vancouver, BC, Canada. Nov 2014.
2014	Session co-chair and organizer. Wildlife Ecotoxicology. SETAC Europe 2014. Basel, Switzerland, May 11-14, 2014 (proposed).
2013	Session co-chair and organizer. Wildlife Ecotoxicology, SETAC 2013, Nashville TN, Nov 17-21, 2013.
2013	Session co-chair and organizer. Wildlife Ecotoxicology. SETAC Europe 2013. Glasgow, Scotland, May 12-16, 2013.
2012	Session chair and organizer. Rodenticides: ongoing problems with contamination and toxicity to non-target wildlife. SETAC 2012, Long Beach California, Nov 11-15, 2012-06-28.
2012	Session chair and organizer. Rodenticides: a nagging problem for non-target wildlife. SETAC Europe 2012, Berlin, Germany, May 20-24, 2012.

2012	Session chair and organizer. Extrapolation within wildlife toxicology. SETAC Europe 2012, Berlin, Germany, May 20-24, 2012.
2012	Session chair and co-organizer. Wildlife Ecotoxicology. SETAC Pacific Northwest. Vancouver, BC, April 26-28, 2012.
2011	Session chair and organizer, Wildllife Ecotoxicology: forensic approaches. SETAC ’11, Boston, MA, Nov 13-17, 2011.
2011	Session co-chair and organizer, The conservation imperative for marine birds and mammals of the Salish Sea. Salish Sea Conference. Vancouver, B.C. Oct 25-27 2011.
2010	Session chair and organizer, Wildlife Ecotoxicology (platform and poster sessions). SETAC ’10, Portland, OR, Nov 8-11, 2010.
2010	Invited session co-chair and organizer. Wildlife Ecotoxicology (platform and poster sessions), SETAC Europe 2010, Seville, Spain, May 23-27, 2010.
2009	Session Chair and Organizer, Wildlife Ecotoxicology (platform and poster session), SETAC ’09, New Orleans, LO, Nov 18-23 2009.
2008	Session Chair and Organizer, Wildlife Ecotoxicology (2 platform, 1 poster sessions) SETAC ’08, Tampa, FL, Nov 16-20, 2008.
2007	Session Chair and Organizer. Wildlife Ecotoxicology. SETAC ’07, Milwaukee, WI, Nov 11-15, 2007.
2007	Session Organizer and Co-Chair. Reproductive, developmental and behaviour toxicology. Platform and poster sessions. SETAC Europe ’07. Porto, Portugal, May 20-24, 2007.
2007	Session Chair. Toxic Chemicals II. Puget Sound – Georgia Basin Research Conference, Vancouver, BC. March 26-30, 2007
2006	Session Organizer and Co-chair, Wildlife Ecotoxicology, Part 1, General. Platform session. SETAC ’06. Montreal QU. Nov 15-20, 2006.
2006	Session Organizer and Co-chair, Wildlife Ecotoxicology: Part 2, Population Level Assessment. Platform and poster sessions. SETAC ’06. Montreal QU. Nov 15-20, 2006.
2006	Session Co-chair. Vertebrate Wildlife Toxicology – platform and poster sessions. SETAC Europe ‘06. The Hague, Netherlands, May 7-11, 2006.
2005	Session Organizer and Co-chair. Wildlife Ecotoxicology, SETAC ’05. Baltimore, MD, Nov 11-15, 2005
2005	Session Co-chair. Contaminant impacts on migrating wildlife. Wildlife Society Meeting, Northwest Chapter, Fort Worden, WA, Apr 20, 2005.

2004	Session Organizer and Co-chair, Wildlife Toxicology: Forensic Approaches. SETAC ’04, Portland, OR, Nov 14 – 18, 2004.
2004	Session Organizer and Co-chair, Wildlife Ecotoxicology. *SETAC ’04*, Portland, OR, Nov 14 – 18, 2004.
2004	Session Organizer and Co-chair, Vertebrate Wildlife Toxicology. SETAC Europe ’04, Prague Czech Republic, April 18 – 22, 2004.
2003	Session Organizer and Co-chair, Contaminant Effects on Raptor Populations. *6th World Conference on Birds of Prey and Owls.* Budapest, Hungary, May 18-23, 2003
2002	Session Organizer and Co-chair, Wildlife Toxicology, SETAC ‘02, Salt Lake City, UT, Nov 16- 20, 2002.
2002	Session Organizer and Co-chair, Amphibian and Wildlife Toxicology, *Aquatic Toxicity Workshop*, Whistler, BC, Oct 21 -23, 2002.
2001	Session Co-chair, Wildlife Toxicology, SETAC ‘01, Baltimore MD, Nov 11-15, 2001.
1999	Meeting Chair, Wildlife Toxicology Program, National Science ’99 Meeting, Ottawa, ON, Oct. 4-6, 1999.
1997	Session chair for ‘Marine Reserves II: Problems and Perspectives’’, *Annual Meeting of the Society for Conservation Biology*, Victoria, BC, June 6-9, 1997.
1995	Session chair and convener for `Wildlife Toxicology' (1 platform and 2 poster sessions) at SETAC '95, Vancouver, BC, Nov. 5-9, 1995.
1993	Workshop leader on `Investigating wildlife mortality incidents' at the W*orkshop on Wildlife Mortality Reporting*, Burlington, ON, Jan. 20-23, 1993.
1991	Session chair and convener for a Special Symposium `Dioxins in the marine environment: the Strait of Georgia as a case study'. SETAC '91, Seattle, WA, Nov. 3-7, 1991.
1986	Session chair on `Terrestrial Monitoring Programs' at the *9th US-German Seminar on Environmental Specimen Banking*, Gloucester, VA, Oct. 6-8, 1986.
1983	Workshop leader on `Uses of specimen bank materials' at the *International Review of Environmental Specimen Banking*, Washington, DC, Sept. 21-23, 1983.

CONSERVATION ACTIVITIES:

1989 -	**British Columbia Waterfowl Society**, member
1992 – present	**Vancouver Natural History Society**, member of Birder's Group and conservation Committee.
2006 -	**Director**, Little Campbell River Watershed Society
2010 -	**Director** – Campbell Valley Park Association

COMMUNITY ACTIVITIES:

1985 – 2005	Coached minor hockey, ringette and soccer during many years.

OTHER SKILLS:

Languages: English, French and German.

S.C.U.B.A. certification (1980).

Avian field identification.

BIBLIOGRAPHY

Theses:

Elliott, J.E. 1995: Environmental contaminants in bald eagles on the coast of British Columbia: exposure and biological effects. PhD Thesis, University of British Columbia.

Elliott, J.E. 1989. Toxicological investigation of polychlorinated biphenyl (PCB) congeners in Japanese quail: induction of drug metabolizing enzymes and porphyria. M.Sc. Thesis, University of Ottawa.

Elliott, J.E. 1979 Use of macro-invertebrate diversity for the biological assessment of a stream receiving impounded urban runoff. Bsc Honours Thesis, Carleton University.

Publications

Career Summary (1980 to present)

Primary peer-reviewed journal articles	137
Books edited	1
Book Chapters (peer reviewed)	18
Authoritative Reviews	10
Conference proceedings, technical reports and notes	44

Non-refereed professional publications, workshop proceedings and government reports	20
Internet publishing	5
Peer-reviewed articles submitted or in preparation	14
Unpublished technical reports	19
Published abstracts	243

Refereed journals (137):

Hindmarch S, Krebs EA, **Elliott** JE, Green DJ. 2014. Urban development influences the breeding success of barn owls in the Fraser Valley, British Columbia, Canada. *Condor* (accepted).

Eng ML, Williams TD, Letcher RJ, **Elliott** JE. 2014. Assessment of concentrations and effects of organohalogen contaminants in a terrestrial passerine, the European starling**.** *Science of the Total Environment* (on-line).

Elliott KH, **Elliott**, JE. 2014. Equations for lipid normalization of carbon stable isotope ratios in aquatic bird eggs. *PLOS ONE* 9. e83597.

Elliott JE, Hindmarch S, Albert CA, Emery J, Maisonneuve F, Mineau P. 2014. Exposure pathways of anticoagulant rodenticides to non-target wildlife. *Environmental Monitoring and Assessment* 186:895-906.

Chen D, Martin PA, Burgess NB, Champoux L, **Elliott** JE, Forsyth D, Idrissi A, Letcher RJ. 2013. European starlings (*Sturnus vulgaris*) indicate that landfills are an important source of bioaccumulative flame retardants to Canadian Terrestrial ecosystems. *Environmental Science & Technology* 47:12238-12247.

Currier H A., Letcher R J, Williams T D, **Elliott** J E. 2013. An Assessment of In Ovo Toxicity of the Flame Retardant 1, 2-Dibromo-4-(1, 2-Dibromoethyl) Cyclohexane (TBECH) in the Zebra Finch. *Bulletin of Environmental Contamination and Toxicology* 1-5. DOI 10.1007/s00128-013-1070-z.

Elliott JE, Elliott KH. 2013. Tracking marine pollution *Science* 340: 556-558. (invited).

Eng ML, Williams TD, **Elliott** JE. 2013. Early exposure to BDE-99: an assessment of effects on growth, physiology and reproduction in the zebra finch. *Environmental Pollution* 178: 343-349.

Eng ME, **Elliott** JE, Letcher RJ, Williams TD. 2012. Individual variation in body burden, lipid status and reproductive investment affects maternal transfer of a brominated diphenyl ether (BDE-99) to eggs in the zebra finch. *Environmental Toxicology and Chemistry* 32: 345–352,

Winter V, Williams TD, **Elliott** JE. 2013. A three-generational study of *in ovo* exposure to PBDE-99 in the zebra finch. *Environmental Toxicology and Chemistry* 32:562-568.

Eens M, Jaspers VL, Steen E van den, Bateson M, Carere C, Clergeau P, Costantini D, Dolenec Z, **Elliott** JE, Flux J, Gwinner H, Halbrook R, Heeb P, Mazgajsk T, Moksnes A, Polo V, Soler JJ, Sinclair R, Veiga JP, Williams TD, Covaci A, Pinxten R. 2013. Can starling eggs be useful as a biomonitoring tool to study organohalogenated contaminants on a worldwide scale? *Environment International* 51:141-149.

Elliott JE, Levac J, Guigeno MF, Wayland M, Shaw P, Morrissey CA, Muir DEG, Elliott KH. 2012. Factors influencing legacy pollutant accumulation in alpine osprey: biology, geography or melting glaciers? *Environmental Science & Technology* 46:9681-9689.

Winter V, **Elliott** JE, Letcher RJ, Williams TD. 2012. Validation of an egg-injection method for embryotoxicity studies in a small, model songbird, the zebra finch (*Taeniopygia guttata*). *Chemosphere* 90:125-131.

Hindmarch S, Krebs EA, **Elliott** JE, Green DJ. 2012. Do landscape features predict the presence of barn owls in a rapidly changing agricultural landscape? *Landscape and Urban Planning* 107:255-262.

Chen D, Letcher RJ, Burgess NB, Champoux L, **Elliott** JE , Hebert CE, Martin PA, Wayland M, Weseloh DV, Wilson LK. 2012. Flame retardants in eggs of four gull species (*Laridae*) from breeding sites spanning Atlantic to Pacific Canada. *Environmental Pollution* 168:1-9.

Guertin, D.A., M. Ben-David, A. Harestad, and J. E. **Elliott**. Fecal Genotyping Reveals Demographic Variation in River Otters Inhabiting a Contaminated Environment. 2012. *Journal of Wildlife Management* 76:1540–1550.

Eng ML, **Elliott** JE, MacDougall-Shackleton SA. Letcher RJ, Williams TD. 2012. Early exposure to 2,2'4,4',5-Pentabromodiphenylether (BDE-99) affects mating behaviour of zebra finches. *Toxicological Sciences* 127:269-276.

Morrissey CA, Pollet I, Ormerod SJ, **Elliott** JE. 2012. American dippers indicate contaminant biotransport by Pacific salmon. *Environmental Science & Technology* 46:1153-1162.

Guigueno MF, Elliott KH, Wayland M, Levac J, Wayland M, **Elliott** JE. 2012. Differential exposure of alpine ospreys to mercury: melting glaciers, hydrology or deposition patterns? *Environment International* 40:24-32.

Elliott KH, **Elliott**, JE, Wilson LK, Jones I, Stenersen K. 2011. Bald eagles and chum salmon: reduced winter survival and breeding success leads to density-dependence. *Journal of Wildlife Management* 75:1688-1699.

Gebbink WA, Letcher RJ, Burgess NM, Champoux L, **Elliott** JE, Hebert CE, Martin PA, Wayland M, Weseloh DV, Wilson, L. 2011. Perfluoroalkyl carboxylates and sulfonates and precursors in relation to dietary source tracers in eggs of four species of gulls (*Larids*) from breeding sites spanning Atlantic to Pacific Canada. *Environment International* 37:1175-1182.

Ashpole SL, Bishop CA, **Elliott** JE*.* 2011. Unexplained die-off of larval barred Tiger Salamanders (Ambystoma mavrtium) in an agricultural pond in the South Okanagan Valley, British Columbia , Canada. *Northwestern Naturalist* 9:221-224*.*

Hipfner JM, Hobson KA, **Elliott** JE. 2011. Ecological and behavioral factors differentially affect mercury levels in two sympatric marine bird species. *Science of the Total Environment* 409:1328–1335.

Thomas PH, Mineau P, Shore RF, Champoux L, Martin PA, Wilson LK, Fitzgerald G, **Elliott** JE. 2011. Second generation anticoagulant rodenticides in predatory birds: probabilistic characterization of toxic liver concentrations and implications for predatory bird populations in Canada. *Environment International*. 37:914-920.

Wilson LK, Harris ML,Trudeau S, Ikonomou MG, **Elliott** JE. 2010. Properties of blood, porphyrins and exposure to organic contaminants in surf scoters (*Melanitta perspicillata*) over-wintering on the south coast of British Columbia, Canada. *Archives Environmental Contamination and Toxicology.* 59:322-353.

Best, D.A., K.H. Elliott, W.W. Bowerman, M. Shieldcastle, S. Postupalsky, P.E.Nye, T.J. Kubiak, D.E. Tillitt and J.E. **Elliott**. 2010. Productivity, embryo and eggshell characteristics and contaminants in bald eagles from the Great Lakes, U.S.A. *Environmental Toxicology & Chemistry* 29:1581-1592.

Cesh L, Elliott KH, McKinney M, Quade S, Maisonneuve F, Garcelon DK, Sandau CD, Letcher RJ, Williams TD, **Elliott** JE. 2010. Polyhalogenated aromatic hydrocarbons and Metabolites: Relation to Circulating Thyroid Hormone and Retinol in Nestling Bald Eagles (Haliaeetus leucocephalus). *Environmental Toxicology and Chemistry* 29:1301-1311.

Guertin, D.A., A. Harestad, and J. E. **Elliott**. 2010. Summer feeding ecology of river otters inhabiting coastal southern Vancouver Island, British Columbia, Canada . *Northwest Science* 84:1-8*.*

Morrissey CA, **Elliott** JE, Ormerod SJ. 2010. Local to Continental Influences on Nutrient and Contaminant Sources to River Birds. *Environmental Science & Technology* 44:1860-1867.

Bishop CA, Ashpole SL, Edwards AM, VanAggelen G, **Elliott** JE. 2010. Hatching success and pesticide exposures in amphibians living in agricultural habitats of the South Okanagan valley, British Columbia, Canada (2004-2006). *Environmental. Toxicology and Chemistry* 29:!593-1603.

Westman A, **Elliott** JE, Cheng K, VanAggelen, G, Bishop CA. 2010. Effects Of environmentally relevant concentrations of endosulfan, azinphos-methyl and diazinon on Great Basin Spadefoot (Spea Intermontana) and Pacific Treefrog (Pseudacris Regilla). *Environmental Toxicology and Chemistry* 29:1604-1612.

Albert CA, Mineau P, Wilson LK, Trudeau S, **Elliott** JE. 2010. Rodenticide residues and autopsy data for three owl species from British Columbia, Canada. *Archives Environmental Contamination and Toxicology* 58:451-459.

Morrissey CA, **Elliott** JE, Ormerod SJ. 2010. Diet shifts during egg laying : implications for measuring contaminants in bird eggs. *Environmental Pollution* 158:447-454.

Enrique PL, Cheng KM, **Elliott** JE. 2010. The 'near threatened' bearded screech owl, *Megascops barbarus*: diet pattern and trophic assessment using $\delta^{13}C$ and $\delta^{15}N$ stable istopes. *Bird Conservation International* 20:25-33.

Guertin, D.A., M. Ben-David, A. Harestad, K.G. Drouillard, and J.E. **Elliott**. 2010. Non-invasive fecal sampling links individual river otters to chlorinated hydrocarbon contaminant exposure. *Environmental Toxicology and Chemistry* 29:275-284.

Elliott KH, Dooley J, Cesh L, Letcher R, **Elliott** JE. 2009. PCB and DDE, but not PBDEs, increase with trophic level and marine input in British Columbian bald eagles. *Science of the Total Environment*. 407:3867-3875.

Elliott JE, Guertin DA, Balke JME. 2008. Chlorinated hydrocarbon contaminants in faeces of coastal marine river otters. *Science of the Total Environment*. 397:58-71.

Albert CA , Williams TD, Cullen WR, Lai V, Morrissey C, **Elliott** JE. 2008. Tissue uptake, mortality and sub-lethal effects of monomethylarsonic acid (MMA (V)) in nestling Zebra Finches (*Taeniopygia guttata*). *Journal of Toxicology and Environmental Health*: 71: 353–360.

Cesh, L.S., T.D. Williams, D. K. Garcelon, and J.E. **Elliott**. 2008. Spatial and Temporal Trends of Chlorinated Hydrocarbons in Nestling Bald Eagle (*Haliaeetus leucocephalus*) Plasma in British Columbia and Southern California. *Archives of Environmental Contamination & Toxicology* (On line first).

Scheuhammer, A.M., N.Basu, N.M. Burgess, J.E. **Elliott**, G.D. Campbell, M. Wayland, L. Champoux, and J. Rodrigue. 2008. Relationships among mercury, selenium, and neurochemical parameters in common loons (*Gavia immer*) and bald eagles (*Haliaeetus leucocephalus)*. *Ecotoxicology*. 17:93-101.

Elliott, J.E., A. Birmingham, L.K. Wilson, M. McAdie, and P. Mineau. 2008. Fonofos poisons raptors and waterfowl several months after labelled application. *Environmental Toxicology & Chemistry.* 27:452-460.

Albert CA , Williams TD, Cullen WR, Lai V, Morrissey C, **Elliott** JE. 2008. Dose dependant uptake, elimination and toxicity of monosodium methanearsonate (MSMA) in adult Zebra Finches (*Taeniopygia guttata*). *Environmental Toxicology and Chemistry.* 27:605-611.

Morrissey CA, PL Dods, JE **Elliott**. 2008. Pesticide treatments affect mountain pine beetle abundance and woodpecker foraging behaviour. *Ecological Applications*. 18:172-194.

Harris ML, LK Wilson, S Trudeau, JE **Elliott**. 2007. Vitamin A and contaminant concentrations in surf scoters (*Melanitta perspicillata*) wintering on the Pacific coast of Canada. *Science of the Total Environment.* 378:366-375.

Elliott, J.E., C.M. Morrissey, L.K. Wilson, P. Shaw, S. Lee, C.J. Henny. 2007. Use of satellite telemetry to track southward migration of Pacific Northwest ospreys and their exposure to contaminants on the wintering grounds. *Ecological Applications* 17:1223-1233.

Loveridge, A.R., C.A. Bishop, J.E. **Elliott**, and C.J. Kennedy. 2007. Polychlorinated biphenyls and organochlorine pesticides bioaccumulated in Green Frogs, *Rana clamitans*, from the lower Fraser Valley, British Columbia, Canada. *Bulletin of Environmental Contamination and Toxicology* 79:315-318 (grad student paper).

Morrissey, C.A., C.A. Albert, P.L. Dods, W.R. Cullen, V.W.-M. Lai, and J.E. **Elliott**. 2007. Arsenic accumulation in bark beetles and forest birds occupying mountain pine beetle infested stands treated with monosodium methanearsonate (MSMA). *Environmental Science & Technology* 41:1494-1500 (grad student paper).

Elliott, J.E., M.L. Harris, L.K. Wilson, B.D. Smith, J. Maguire. 2007. Butyltins, trace metals and morphological variables in surf scoter (*Melanitta perspicillata*) wintering on the south coast of British Columbia, Canada. *Environmental Pollution* 149:114-124.

Basu, N., A.M. Scheuhammer, S.J. Bursian, J.E. **Elliott**, K. Rouvinen-Watt, H.M. Chan. 2007. Mink as a sentinel species in environmental health. *Environmental Research* 103:130-144.

McKinney, M.A., L.S. Cesh, J.E. **Elliott**, T.D. Williams, D.K. Garcelon, R.J. Letcher. 2006. Novel brominated and chlorinated contaminants and hydroxylated analogues among North American west coast populations of bald eagles (*Haliaeetus leucocephalus*). *Environmental Science & Technology* 40:6275-6281 (grad student paper).

Iwaniuk, A.N., D.T. Koperski, K.M. Cheng, J.E. **Elliott**, L.K. Smith, L.K. Wilson, and D.R.W. Wylie. 2006. The effects of environmental exposure to DDT on the brain of a songbird: Changes in structure associated with mating and song. *Behavioural Brain Research* 173:1-10.

Elliott, K.H., S.L. Lee, J.E. **Elliott**. 2006. Foraging ecology of bald eagles at an anthropogenic food source: does the Vancouver landfill affect local populations? *Wilson Bulletin* 118:380-390.

Weech, S.A., A.M. Scheuhammer, J.E. **Elliott**. 2006. Mercury exposure and reproduction in fish-eating birds breeding in the Pinchi Lake Region, British Columbia, Canada. *Environmental Toxicology and Chemistry* 25:1433-1440. (grad student paper).

Morrissey, C.A., L.I. Bendell-Young, and J.E**. Elliott** 2005. Contributions from atmospheric deposition to food chain biomagnification in the Chilliwack watershed of British Columbia, Canada. *Environmental Science and Technology* 39:8090-8098 (grad student paper).

Dods, P.L., E. Birmingham, T.D. Williams, M. Iknonomou and J.E.**Elliott**. 2005. Exposure of breeding tree swallows (*Tachycineta* bicolour) to nonyl-phenol at a

wastewater treatment plant. *Environmental Toxicology & Chemistry* 24:3106-3112 (grad student paper).

Elliott, J.E. 2005. Trace metals, stable isotope ratios, and trophic status in seabirds from the North Pacific Ocean. *Environmental Toxicology and Chemistry* 24:3099-3105.

Harris, M.L., L. K. Wilson and J. E. **Elliott**. 2005. An assessment of chlorinated hydrocarbon contaminants in eggs of double-crested (*Phalacrocorax auritus*) and pelagic (*P. pelagicus*) cormorants from the west coast of Canada,1970 to 2002. *Ecotoxicology* 14:607-625.

Elliott, J.E., L.K. Wilson, B. Wakeford. 2005. Polybrominated diphenyl ether trends in eggs of marine and freshwater birds from British Columbia, Canada, 1979-2002. *Environmental Science and Technology* 39:5584-5591.

Morrissey, C.A., L.I. Bendell-Young, J.E. **Elliott**. 2005. Assessing trace-metal exposure to American dippers in mountain streams of southwestern British Columbia, Canada. *Environmental Toxicology and Chemistry* 24:836-845.

Elliott, J.E. Chlorinated hydrocarbon contaminants and stable isotope ratios in pelagic seabirds from the North Pacific Ocean. 2005. *Archives of Environmental Contamination and Toxicology* 49:89-96.

Christensen, J.R., J.S. Richardson, C.A. Bishop, B. Pauli, J.E. **Elliott**. 2005. Effects of nonylphenol on tail resorption and metamorphic staging of bullfrog (*Rana catesbeiana*) tadpoles. *Journal of Toxicology and Environmental Health* 68:557-572 (grad student paper).

Elliott, J.E., M.J. Miller and L.K. Wilson. 2005. Assessing breeding potential of peregrine falcons based on chlorinated hydrocarbon concentrations in prey. *Environmental Pollution* 134:353-361.

Elliott, K.H., C.E. Gill, J.E. **Elliott**. 2004. The influence of tide and weather on provisioning rates of chick-rearing bald eagles in Vancouver Island, British Columbia *Journal of Raptor Research* 39:1-10 (grad student paper).

Morrissey, C.A., L.I. Bendell-Young, and J.E. **Elliott**. 2004. Seasonal trends in population density, distribution and movement of American dippers within a watershed of southwestern British Columbia, Canada. *Condor* 106:815-825 (grad student paper).

Christensen, J.R., C. A. Bishop, J.S. Richardson, B. Pauli J.E. **Elliott**. 2004 Validation of an amphibian sperm inhibition toxicological test (ASITT) method using zinc. *Environmental Toxicology & Chemistry* 23: 2950-2955 (grad student paper).

Gill, H., J.E. **Elliott**, C.A. Bishop, K.M. Cheng, T.D. Williams. 2004. Effects of azinphos-methyl on cholinergic responses and general health of Zebra Finches (*Taeniopygia guttata*) following prior treatment with *p,p'*-DDE. *Archives of Environmental Contamination and Toxicology* 48:118-126 (grad student paper).

Elliott, K.H., C.L. Struik and J.E. **Elliott**. 2004. Bald eagles *Haliaeetus leucocephalus*) feeding on spawning plainfin midshipman (*Porichthys notatus*) at Crescent Beach, British Columbia. *Canadian Field Naturalist* 117:601-604 (student paper).

Christensen, J.R., B.D. Pauli, J.S. Richardson, C.A. Bishop and J.E. **Elliott** .2004. Effects of pH and osmolality on *Xenopus laevis* sperm motility. *Canadian Journal of Zoology* 82:555-563 (grad student paper).

Morrissey, C.A., J.E. **Elliott** and L.I. Bendell-Young. 2004. Linking contaminant profiles to the diet and breeding location of American dippers using stable isotope analysis. *Journal of Applied Ecology* 41-502-512 (grad student paper).

Weech, S.A., A.M. Scheuhammer, K.M. Cheng and J.E. **Elliott**. 2004. Mercury in fish from the Pinchi Lake Region, British Columbia, Canada. *Environmental Pollution* 131-275-386 (grad student paper).

Weech, S.A., J.E. **Elliott** and L.K. Wilson. 2003. Mercury residues in livers of bald eagles (*Haliaeetus leucocephalus*) found dead or dying in British Columbia (1987-1994). *Archives of Environmental Contamination & Toxicology* 45:562-569 (grad student paper).

Wayland, M., L.K Wilson, J.E. **Elliott**, M.J.R. Miller, T. Bollinger, M. McAdie, K. Langelier J. Keating and J.M.W. Froese. 2003. Lead poisoning and other causes of mortality and debilitation in bald and golden eagles in western Canada, 1986-98. *Journal of Raptor Research* 37: 8 -18.

Gill, C.E. and J.E. **Elliott**. 2003. Influence of food supply and chlorinated hydrocarbon contaminants on breeding success of bald eagles. *Ecotoxicology* 12:95-112 (grad student paper).

Gill, H., L.K. Wilson, K.M. Cheng and J.E. **Elliott**. 2003. Effects of organochlorine compounds on the reproductive success of American robins (*Turdus migratorius*) breeding in fruit orchards. *Ecotoxicology* 12:113-124 (grad student paper).

Harris, M.L., L.K. Wilson, R.J. Norstrom and J.E. **Elliott**. 2003. Egg concentrations of polychlorinated dibenzo-*p*-dioxins and dibenzofurans in double-crested (*Phalacrocorax auritus*) and pelagic (*Phalacrocorax pelagicus*) cormorants from the Strait of Georgia, Canada, 1973 to 1998. *Environmental Science & Technology* 37:822-831.

Kennedy, S.W., S.P. Jones and J.E. **Elliott**. 2003. Sensitivity of bald eagle *Haliaeetus leucocephalus*) hepatocyte cultures to cytochrome P4501A induction and porphyrin accumulation by 2,3,7,8-tetrchlorodibenzo-*p*-dioxin. *Ecotoxicology* 12:163-170.

Hughes, M.R., D.C. Bennett, D.A. D.A. Gray, P. Sharp, A.M. Scheuhammer, J.E. **Elliott.** 2003. Effects of cadium ingestion on plasma and osmoregulatory hormone concentrations in male and female Pekin ducks. *Journal of Toxicology and Environmental Health* 66:565-579.

Henny, C.J., J.L. Kaiser, R.A. Grove. V. Raymond Bentley and J.E. **Elliott**. 2003. Biomagnification factors (fish to osprey eggs from Willamette River, Oregon, U.S.A.) for PCDDs, PCDFs, PCBs and OC pesticides. *Environmental Monitoring & Assessment* 84:275-315.

Harris, M.L., J.E. **Elliott**, L.K. Wilson and R.W. Butler. 2003. Reproductive success and chlorinated hydrocarbon contamination of resident great blue herons (*Ardea herodias*) from coastal British Columbia, Canada, 1977 to 1998. *Environmental Pollution* 121: 207-227.

Barjactarovic, L., J.E. **Elliott**, A.M. Scheuhammer. 2002. Heavy metals and metallotheionein in surf scoters from British Columbia and the Yukon. *Archives of Environmental Contamination & Toxicology* 43(4): 486-491.

Elliott, J.E. and M.L. Harris. 2001/2002. An ecotoxicological assessment of chlorinated hydrocarbon effects on bald eagle populations. *Reviews in Toxicology* 4:1-60.

De Solla, S.R., K.E. Pettit, C.A. Bishop, K.M. Cheng and J.E. **Elliott**. 2002. Effects of agricultural runoff on hatching success, deformities and survivorship of native amphibians in the Lower Fraser Valley, British Columbia, Canada, 1997-8. *Environmental Toxicology & Chemistry* 21:353-360 (grad student paper).

Wilson, L.K., J.E. **Elliott**, R.S. Vernon and S.Y. Szeto. 2002. Retention of the active ingredients in granular phorate, terbufos, fonofos, and carbofuran in soils of the Lower Fraser Valley and their implications for wildlife poisoning. *Environmental Toxicology & Chemistry* 21:260-268.

De Solla, C.A. Bishop and J.E. **Elliott**. 2002. Organochlorine pesticides and polychlorinated biphenyls (PCBs) in eggs of red-legged frogs (*Rana aurora*) and northwestern salamanders (*Ambystoma gracile*) in an agricultural landscape. *Chemosphere* 46:1027-1032 (grad student paper).

Elliott, J.E., M.L. Harris, L.K. Wilson, P.E. Whitehead and R.J. Norstrom. 2001. Monitoring great blue heron (*Ardea herodias*) coastal breeding colonies in British Columbia, Canada, 1983 to 1998: temporal and spatial trends in polychlorinated dibenzo-*p*-dioxins and dibenzofurans in eggs. *Ambio* 30:416-428.

Wilson, L.K., P.A. Martin J.E. **Elliott**, P. Mineau and K.M. Cheng. 2001. Exposure and effects of organophosphate insecticides on California quail breeding in fruit orchards in the Okanagan Valley of British Columbia. *Ecotoxicology* 10:79-90.

Peterson, C.A., S.L. Lee and J.E. **Elliott**. 2001. Scavenging of waterfowl carcasses by avian predators in the Fraser River delta of British Columbia. *Journal of Field Ornithology* 72: 150-159 (Intern paper).

Elliott, J.E., L.K. Wilson, C.J. Henny, S. Trudeau, F.A. Leighton, S.W. Kennedy, K.M. Cheng. 2001. Assessment of biological effects of chlorinated hydrocarbons in osprey chicks. *Environmental Toxicology & Chemistry* 20(4): 866-879.

Hughes, M.R., J. Smits, J.E. **Elliott** and D.C. Bennett. 2000. Morphological, physiological and pathological effects of simultaneous ingestion of cadmium and

saline by Pekin ducks. *Journal of Toxicology and Environmental Health* Part A 61:591-608.

Gill, H., L.K. Wilson, K.M. Cheng, S. Trudeau and J.E. **Elliott**. 2000. Effects of azinphos-methyl on American robins breeding in fruit orchards. *Bulletin of Environmental Contamination and Toxicology* 65:756-763 (Grad student paper).

Harris, M.L. L.K. Wilson, J.E. **Elliott**, C.A. Bishop, A.D. Tomlin and K.V. Henning. 2000. Transfer of DDT and metabolites from fruit orchard soils to American robins (*Turdus migratorius*) twenty years after agricultural use of DDT in Canada. *Archives of Environmental Contamination and Toxicology* 39:205-220.

Harris, M.L. and J.E. **Elliott**. 2000. Reproductive success and chlorinated hydrocarbon contamination of tree swallows (*Tchycineta bicolor*) nesting along rivers receiving pulp and paper mill effluent discharges. *Environmental Pollution* 110:307-320.

Bennett, D.C., M.R. Hughes, J.E. **Elliott**, A.M. Scheuhammer and J. Smits. 2000. Effects of cadmium on Pekin duck total body water, water flux, renal filtration and salt gland function. *Journal of Toxicology and Environmental Health* Part A 59:43-56.

Elliott, J.E., L.K. Wilson, C.J. Henny and M.M. Machmer. 2000. Contaminants in ospreys from the Pacific Northwest: II. Patterns and trends in organochlorine pesticides, polychlorinated biphenyls and mercury in eggs and plasma. *Archives Environmental Contamination and Toxicology* 38:93-106.

Howald, G.R., P. Mineau, J.E. **Elliott** and K.M. Cheng. 1999. Brodifacoum poisoning of avian scavengers during rat control on a seabird colony. *Ecotoxicology* 8:429-446 (Grad student paper).

Tittlemeier, S.A., R.J. Norstrom, M. Simon, W.M. Jarman and J.E. **Elliott**. 1999. Identification and distribution of a novel brominated chlorinated heterocyclic compound in seabird eggs. *Environmental Science & Technology* 33:26-33.

Mineau, P. M.R. Fletcher, L.C. Glaser, N.J. Thomas, C. Brassard, LK. Wilson, J.E. **Elliott**, L.A. Lyon, C.J. Henny, T. Bollinger and S.L. Porter. 1999. Poisoning of raptors with organophosphate and carbamate pesticides with emphasis on Canada, the United States and the United Kingdom. *Journal of Raptor Research* 33: 1-37.

Elliott, J.E., C.J. Henny, M.L. Harris, L.K. Wilson and R.J. Norstrom. 1999. Chlorinated hydrocarbons in livers of American mink (*Mustela vison*) and river otter (*Lontra canadensis*) from the Columbia and Fraser River basins, 1990-91. *Environmental Monitoring and Assessment* 57: 229-252.

Harding, L., M.L. Harris, C.R. Stephen and J.E. **Elliott**. 1999. Reproductive and morphological condition of wild mink (*Mustela vison*) and river otter (*Lutra canadensis*) in relation to chlorinated hydrocarbon contamination. *Environmental Health Perspectives* 107: 141-147.

Harding, L., M.L. Harris, and J.E. **Elliott**. 1998. Heavy trace metals in wild mink (*Mustela vison*) and river otter (*Lutra canadensis*) captured on rivers receiving metals discharges. *Bulletin of Environmental Contamination and Toxicology* 61:600-607.

Elliott, J.E., M. Machmer, C.J. Henny, P.E. Whitehead and R.J. Norstrom. 1998. Contaminants in ospreys from the Pacific Northwest: I. Trends and patterns in polychlorinated dibenzo-*p*-dioxins and dibenzofurans in eggs and plasma. *Archives of Environmental Contamination and Toxicology* 35: 620-631.

Elliott, J.E. and P.A. Martin. 1998. Chlorinated hydrocarbons in grebes and seaducks wintering on the coast of British Columbia, Canada, 1988-93. *Environmental Monitoring and Assessment* 53:337-362.

Kannan, K., K. Senthilkumar, J.E. **Elliott**, L.A. Feyk and J.P. Giesy. 1998. Occurrence of butyltin compounds in tissues of waterbirds and seaducks from the United States and Canada. *Archives of Environmental Contamination and Toxicology* 35:64-69.

Wilson, L.K., J.E. **Elliott**, K.E. Langelier, A.E. Scheuhammer and V. Bowes. 1998. Lead poisoning in swans from British Columbia. *Canadian Field Naturalist* 112:204-211.

Elliott, J.E. and R.J. Norstrom. 1998. Chlorinated hydrocarbon contaminants and productivity of bald eagles on the Pacific coast of Canada. *Environmental Toxicology and Chemistry* 17:1142-1153.

Elliott, J.E., I.E. Moul and K.M. Cheng. 1998. Variable reproductive success of bald eagles on the British Columbia coast. *Journal of Wildlife Management* 62:518-529.

Elliott, J.E., S.W. Kennedy and A. Lorenzen. 1997. Comparative effects of Polychlorinated biphenyls on Japanese quail and American kestrels. *Journal of Toxicology and Environmental Health* 51:57-75.

Elliott, J.E. and A.M. Scheuhammer. 1997. Heavy metal and metallothionein concentrations in Pacific Canadian seabirds. *Marine Pollution Bulletin* 34:794-801.

Elliott, J.E., L.K. Wilson, K.E. Langelier, P. Mineau and P.H. Sinclair. 1997. Poisoning of birds of prey by the organophosphorus insecticide, Phorate. *Ecotoxicology* 6:219-231.

Henshel, D.S., J.W. Martin, R.J. Norstrom, J.E. **Elliott**, K.M. Cheng and J.C. DeWitt. 1997. Morphometric brain abnormalities in double-crested cormorant chicks exposed to polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls. *Journal of Great Lakes Research* 23:11-26.

Elliott, J.E., L.K. Wilson, K.E. Langelier and R.J. Norstrom. 1996. Bald eagle mortality and chlorinated hydrocarbons in liver samples from the Pacific coast of Canada, 1988-1995. *Environmental Pollution* 94:9-18.

Elliott, J.E., R.J. Norstrom and G.E.J. Smith. 1996. Patterns, trends and toxicological significance of chlorinated hydrocarbons and mercury in bald eagle eggs. *Archives of Environmental Contamination and Toxicology* 31:354-367.

Elliott, J.E., K.E. Langelier, P. Mineau and L.K. Wilson. 1996. Poisoning of bald eagles and red-tailed hawks by carbofuran and fensulfothion in the lower Fraser valley of British Columbia, Canada. *Journal of Wildlife Diseases* 32:486-491.

Elliott, J.E., R.J. Norstrom, A. Lorenzen, L.E. Hart, H. Phillibert, S.W. Kennedy, J.J. Stegeman, G.D. Bellward and K.M Cheng. 1996. Biological effects of polychlorinated dibenzo-*p*-dioxin, dibenzofurans and biphenyls in bald eagle (*Haliaeetus leucocephalus*) chicks. *Environmental Toxicology and Chemistry.* 15:782-793.

Bonin, J., J.-L. Desgranges, C.A. Bishop, J. Rodrigue, A. Gendron and J.E. **Elliott**. 1995. Comparative study of contaminants in mudpuppy (*Amphibia*) and common snapping turtle (*Reptilia*) from the St. Lawrence River, Canada. *Archives of Environmental Contamination and Toxicology* 28:184-194.

Elliott, J.E., and P.A. Martin. 1994. Chlorinated hydrocarbons and shell thinning in eggs of *Accipiter* hawks in Ontario, 1986-1989. *Environmental Pollution* 86:189-200.

Elliott, J.E., P.A. Martin, T.W. Arnold & P.H. Sinclair. 1994. Organochlorines and reproductive success of birds in orchard and non-orchard areas of central British Columbia, 1990-91. *Archives of Environmental Contamination and Toxicology* 26:435-443.

Moul, I.E. & J.E. **Elliott**. 1994. The bird community found on golf courses in British Columbia. *Northwest Naturalist* 75:88-96.

Sanderson, J.T., J.E. **Elliott**, R.J. Norstrom, P.E. Whitehead, L.E. Hart, K.M. Cheng & G.D. Bellward. 1994. Monitoring biological effects of polychlorinated dibenzo-p-dioxins, dibenzofurans and biphenyls in great blue heron chicks. *Journal of Toxicology and Environmental Health* 41:435-450.

Sanderson, J.T., R.J. Norstrom, J.E. **Elliott**, L.E. Hart, K.M. Cheng and G.D. Bellward. 1994. Biological effects of polychlorinated dibenzodioxins, dibenzofurans and biphenyls in double-crested cormorant chicks. *Journal of Toxicology and Environmental Health* 41:247-265.

Struger, J., J.E. **Elliott**, C.A. Bishop, M. E. Obbard, R.J. Norstrom, D.V. Weseloh, M. Simon & P. Ng. 1994. Environmental contaminants in eggs of the common snapping turtle (*Chelydra s. serpentina*) from the Great Lakes-St.Lawrence River Basin of Ontario, Canada (1981, 1984). *Journal of Great Lakes Research* 19:681-694.

Elliott, J.E. and L. Shutt. 1993. Monitoring organochlorines in blood of Sharp-shinned hawks (*Accipiter striatus*) migrating through the Great Lakes. *Environmental Toxicology and Chemistry* 12:241-250.

Vermeer, K., W.J. Cretney, J.E. **Elliott**, R.J. Norstrom & P.E. Whitehead. 1993. Elevated polychlorinated dibenzodioxin and dibenzofuran concentrations in grebes, ducks and their prey near Port Alberni, British Columbia, Canada. *Marine Pollution Bulletin* 26:431-35.

Elliott, J.E., A.M. Scheuhammer, F.A. Leighton and P.A. Pearce. 1992. Heavy Metal and Metallotheionein levels in Atlantic Canadian Seabirds. *Archives of Environmental Contamination and Toxicology* 22:63-73.

Elliott, J.E., S.W. Kennedy, D. Jeffrey and L. Shutt. 1991. Polychlorinated Biphenyl (PCB) effects of hepatic mixed function oxidases and porphyria in birds: II. American Kestrels. *Comparative Biochemistry and Physiology* 99C:141-145.

Noble, D.G. and J.E. **Elliott**. 1990. Environmental contaminants in Canadian raptors. 1966-1988: effects and temporal trends. *Canadian Field-Naturalist* 104:222-243.

Peakall, D.B., D.G. Noble, J.E. **Elliott**, J.D. Somers and G. Erickson. 1990. Environmental contaminants in Canadian peregrine falcons: a toxicological assessment. *Canadian Field-Naturalist* 104:244-254.

Baril, A., J.E. **Elliott**, J.D. Somers and G. Erickson. 1990. Residue levels of environmental contaminants in the prey of the peregrine falcon *Falco peregrinus* in Canada. *Canadian Field-Naturalist* 104:273-284.

Elliott, J.E., S.W. Kennedy, D.B. Peakall and H. Won. 1990. Polychlorinated Biphenyl (PCB) effects on hepatic mixed function oxidases and porphyria in birds: I. Japanese Quail. *Comparative Biochemistry and Physiology* 96C:205-210.

Bellward, G.D., R.J. Norstrom, P.E. Whitehead, J.E. **Elliott**, S.M. Bandiera, C. Dworschak, T. Cheng, S. Forbes, B. Cadario, L.E. Hart and K.M. Cheng. 1990. Comparison of Dibenzodioxin (PCDD) and Dibenzofuran (PCDF) levels with mixed-function oxidase induction in Great Blue Herons. *Journal of Toxicology and Environmental Health* 30:33-52.

Bellward, G.D., R.J. Norstrom, P.E. Whitehead, J.E. **Elliott**, S.M. Bandiera, C. Dworschak, T. Cheng, S. Forbes and B. Cadario. 1990. Correlation of polychlorinated dibenzodioxin levels with hepatic mixed function oxidase induction in great blue herons. *Chemosphere* 20:1087-1090.

Elliott, J.E., R.W. Butler, R.J. Norstrom and P.E. Whitehead. 1989. Environmental contaminants and reproductive success of Great Blue Herons *Ardea herodias* in British Columbia, 1986-87. *Environmental Pollution* 59:91-114.

Elliott, J.E., P.E. Whitehead, D.G. Noble and R.J. Norstrom. 1989. Organochlorine contaminants in seabird eggs from the Pacific coast of Canada, 1971-1986. *Environmental Monitoring and Assessment* 12:67-82.

Pearce, P.A., J.E. **Elliott**, D.B. Peakall and R.J. Norstrom. 1989. Environmental contaminants in seabirds from the western north Atlantic, 1968-1984. *Environmental Pollution* 56:217-235.

Elliott, J.E., R.J. Norstrom and J.A. Keith. 1988. Organochlorines and eggshell thinning in gannets *(Sula bassana)* from eastern Canada, 1968-1984. *Environmental Pollution* 52:1-23.

Proulx, G., D.V. Weseloh, J.E. **Elliott**, S. Teeple, P.A.M. Angehrn and P. Mineau. 1987. Organochlorine and PCB residues in Lake Erie Mink populations. *Bulletin of Environmental Contamination and Toxicology* 39:939-944.

Struger, J., J.E. **Elliott** and D.V. Weseloh. 1987. Metals and essential elements in herring gulls from the Great Lakes 1983. *Journal of Great Lakes Research* 13:43-55.

Books edited (1)

Elliott JE, Bishop CA, Morrissey CA. *Wildlife Ecotoxicology: Forensic Approaches*. 2012. 15 Chapters on forensic investigations and regulatory outcomes of wildlife contamination and poisoning issues. Springer, New York.

Refereed chapters in books, and review articles (17):

Elliott, JE, Bishop CA, Morrissey CA. 2011. Wildlife Ecotoxicology: forensic approaches. *In* **Elliott** JE, Bishop CA, Morrissey CA. *Wildlife Ecotoxicology: Forensic Approaches*. Springer, New York, pp. 1-9.

Elliott JE. Dioxins, wildlife and the forest industry n British Columbia, Canada. *In* **Elliott** JE, Bishop CA, Morrissey CA. *Wildlife Ecotoxicology: Forensic Approaches*. Springer, New York, pp.11-44.

Elliott JE, Wilson, LK, Vernon RS. Controlling wireworms without killing wildlife in the Fraser River Delta. *In* **Elliott** JE, Bishop CA, Morrissey CA. *Wildlife Ecotoxicology: Forensic Approaches*. Springer, New York, pp. 213-238.

Edson JT, Holmes JV, **Elliott** JE, Bishop CA. The rocky mountain arsenal: from environmental catastrophe to urban wildlife refuge. *In* Elliott JE, Bishop CA, Morrissey CA. *Wildlife Ecotoxicology: Forensic Approaches*. Springer, New York, pp 93-152.

Morrissey CA, **Elliott** JE. Toxic trees : arsenic pesticides, woodpeckers and the mountain pine beetle. *In* Elliott JE, Bishop CA, Morrissey CA. *Wildlife Ecotoxicology: Forensic Approaches*. Springer, New York.

Rattner B, Scheuhammer A, **Elliott** JE. The History of Wildlife Toxicology. *In* Beyer WN, Meador J. eds. Environmental contaminants in wildlife - Interpreting tissue concentrations. CRC Press, New York, NY, USA, pp. 9-44.

Elliott JE, Bishop CA. Cyclodienes and other organochlorine pesticides in birds. *In* Beyer WN, Meador J, eds *Environmental Contaminants in Wildlife — Interpreting Tissue Concentrations*. CRC Press, New York, NY, USA, pp 441-469.

Harris ML, **Elliott** JE. Polychlorinated biphenyls, dibenzo-*p*-dioxins and dibenzofurans and polybrominated diphenyl ethers in birds. *In* Beyer WN, Meador J, eds *Environmental Contaminants in Wildlife — Interpreting Tissue Concentrations*. CRC Press, New York, NY, USA, pp 471-522.

Henny, C.J. and J.E. **Elliott**. 2007. Chapter 18, Toxicology. *Raptor Research and Management Techniques*. Second Edition. D.M Bird and K.L. Bildstein (eds.). Raptor Research Foundation. Hancock House Publishing. Surrey, BC, Canada, 351-364.

Morrissey, C.A., G. Kardosi, L.K. Wilson, J.E. **Elliott**. 2004. Effect of food supply and contaminants on osprey productivity in southern British Columbia, Canada. In Chancellor, R.D and B.-U. Meyburg (eds.). *Raptors Worldwide*. Proceedings 6th World Conference on Birds of Prey and Owls. Budapest, Hungary, May 18-23, 2003.

Henny, C.J., P. Mineau, J.E. **Elliott** and B. Woodbridge. 1998. Raptor pesticide poisonings and current insecticide use: what do isolated kill reports mean to populations? In: N. Adams and R. Slowtow (eds.). Proceedings 22nd International Ornithological Congress, Aug 16-22, 1998, Durban, South Africa (Invited).

Henshel, D.S., J.W. Martin, D. Best, K.M. Cheng, J.E. **Elliott**, D. Rosenstein and J. Sikarskie. (1997) Evaluating gross brain assymetry as a potential biomarker for 2,3,7,8-tetrachlorodebenzo-*p*-dioxin-related neurotoxicity. 1997. In: D.A. Bengston and D.S. Henshel (eds.). Environmental Toxicology and Risk Assessment: Biomarkers and Risk Assessment (5th Volume). American Society for Testing Materials, Philadelphia, *ASTM STP* 1306.

Elliott, J.E., P.E. Whitehead, P.A. Martin, G.D. Bellward and R.J. Norstrom. 1996. Persistent pulp mill pollutants in wildlife. In: M.E. Servos, K.R. Munkittrick, J.H. Carey and G.J. Van Der Kraak (eds.). Fate and Effects of Pulp and Paper Mill Effluents. St. Lucie Press, Boca Raton, Florida, USA. pp. 297-314.

Elliott, J.E. and Noble, D.G. 1993. Chlorinated hydrocarbon contaminants in marine birds of the temperate North Pacific. In K. Vermeer, K.T. Briggs, and K.H. Morgan (Eds.) *The status, ecology and conservation of marine birds of the North Pacific*, Can. Wildl. Serv. Spec. Publ., Ottawa, 241-253.

Elliott, J.E., D.G. Noble, R.J. Norstrom, P.E. Whitehead, M. Simon, P.A. Pearce, D.B. Peakall. 1992. Patterns and trends of organic contaminants in Canadian seabirds, 1968-1990. In *Persistent Pollutants in the Marine Environment*, C.H. Walker and D.R. Livingston (Eds.) Pergamon Press. Oxford, pp. 181-194.

Gilbertson, M., J.E. **Elliott** and D.B. Peakall. 1987. Seabirds as indicators of marine pollution. In: Diamond, A.W. and F. Fillion (eds.). *The Uses of Birds*. ICBP Technical Publication, Cambridge, No. 6, 231-248.

Elliott, J.E. 1984. Collecting and archiving wildlife specimens in Canada. In: R.A. Lewis, N. Stein, and C.W. Lewis (eds.). *Environmental Specimen Banking and Monitoring as Related to Banking*, Martinus Nijhoff, The Hague, pp. 45-64.

Conference proceedings, technical reports and notes (44):

McKinney, M.A., L.S. Cesh, J.E. **Elliott**, D.K. Garcelon and R.J. Letcher. 2006. Brominated flame retardants and hydroxylated and methoxylated organohalogen contaminants in plasma of North American west coast bald eagles (*Haliaeetus leucocephalus*). DIOXIN'2006, Aug. 21-26, Oslo, Norway; Organohalogen Compounds, 68:736-739.

Morrissey, C.A., P.L. Dods, C.A. Albert, W. Cullen, V. Lai, T.D. Williams, J.E. **Elliott** . 2006. Assessing forest bird exposure and effects from Monosodium Methanearsonate (MSMA) during the Mountain Pine Beetle epidemic in British Columbia. Canadian Wildlife Service, Technical Report Series No. 460, 89 pp.

Morrissey, C.A., R.W. Elner, and J.E. **Elliott**. Assessment of chlorinated hydrocarbon contaminants in mammals from agricultural areas of Delta, British Columbia. 2004. Canadian Wildlife Service, Technical Report Series, No. 420. 23 pp.

Elliott, J.E. P. Shaw, M. Wayland, L. Wilson`, G. Kardosi, and D. Muir. 2003. Chlorinated hydrocarbons in fish and osprey from alpine and reference areas of British Columbia. Science Highlights from: *Ecological and Earth Sciences in Mountain Areas*, Banff, AB, Sept 6-10, 2002.

Elliott, J.E. and L.K. Wilson. 2003. Chlorinated hydrocarbons and heavy metals in river otter and mink samples from coastal British Columbia. Proceedings, Georgia Basin/Puget Sound Research Conference, Vancouver, BC, Mar 31 – Apr 3, 2003.

Elliott, J.E., L.K.Wilson, R.J. Norstrom and M.L. Harris. 2003. Chlorinated contaminant trends in indicator species, great blue herons and double-crested cormorants in the Strait of Georgia, 1973-2000. Proceedings, Georgia Basin/Puget Sound Research Conference, Vancouver, BC, Mar 31 – Apr 3, 2003.

Wilson, L.K. and J.E. **Elliott**. Contaminants in surf scoters wintering in the Strait of Georgia, BC, Canada. 2003. Proceedings, Georgia Basin/Puget Sound Research Conference, Vancouver, BC, Mar 31 – Apr 3, 2003.

Wakeford, B.J., M.J. Simon, J.E. **Elliott** and B. M. Braune. 2002. Analysis of polybrominated biphenyl ethers (BDEs) in wildlife tissues. Proceedings, 4Th Annual Workshop on Brominated Flame Retardants in the Environment. Burlington, ON, June 17-18, 2002.

Newson, S.C., C.D. Sandau, J.E. Elliott, S.B. Brown and R.J. Norstrom. PCBs and hydroxylated metabolites in bald eagle plasma - Comparison of thyroid hormone and retinol levels. 2000. Proceedings, International Conference on Environmental Chemistry, Ottawa ON, May 7-11, 2000.

Braune BM, Malone BJ, Burgess NM, **Elliott** JE, Garrity N, Hawkings J, Hines J, Marshall H, Marshall WK, Rodrigue, J, Wakeford B, Wayland M, Weseloh DV, Whitehead PE. 1999. Chemical Residues in Waterfowl and Gamebirds

Harvested in Canada, 1987-95. Canadian Wildlife Service, Technical Report Series, No 326, Ottawa ON, Canada.

Wilson, L.K., M.L. Harris and J.E. **Elliott**. 1998. Contaminants in wildlife indicator species from the Fraser Basin. Pp. 161-188 In: Gray, C. and Tuominen, T. *Health of the Fraser River Aquatic Ecosystem*. Environment Canada, Vancouver, BC. DOE FRAP 1998-11, Vol. 1.

Wilson, L.K., M.L. Harris and J.E. **Elliott**. 1998. Impact of agricultural pesticides on birds of prey in the Lower Fraser Valley. Pp. 101-108 In: Gray, C. and Tuominen, T. *Health of the Fraser River Aquatic Ecosystem*. Environment Canada, Vancouver, BC. DOE FRAP 1998-11, Vol. 2.

Nichol, L., L.K. Wilson, K. Moore and J.E. **Elliott**. 1998. Marine and estuarine birds of the Georgia Basin: Chemical Contamination. *Georgia Basin Booklet*, Canadian Wildlife Service, Delta, BC. Cat. No. CW66-186/2000E. 15 pp.

Tittlemeier, S.A., R.J. Norstrom and J.E. **Elliott**. 1998. Levels of a possible naturally-produced bromo/chloro compound in bird eggs from the Great Lakes, Atlantic, and Pacific coastal areas. *Organohalogen Compounds*, 39:117-120.

Wilson, L.K., J.E. **Elliott** and M. McAdie. 1997. Barbituate poisoning of bald eagles. *Canadian Cooperative Wildlife Health Centre Newsletter* No. 5(1):8.

Kaiser, G.W., R.H. Taylor, P.D. Buck, J.E. **Elliott**, G.R. Howald and M.C. Drever. 1997. The Langara Island Seabird Habitat Recovery Project: Eradication of Norway Rats - 1993-1997. *Canadian Wildlife Service, Technical Report Series*, Delta, BC, No. 304, 96 pp.

Wilson, L.K. and J.E. **Elliott**. 1996. Diazinon poisoning of American wigeon. *Canadian Cooperative Wildlife Health Centre Newsletter* No. 4(6):7.

Elliott, J.E., L.K. Wilson, K.M. Langelier and P. Mineau. Secondary poisoning of birds of prey by anticholinesterase insecticides. 1996. In: Proceedings, 3rd FRAP Research Workshop. Environment Canada, Vancouver, DOE FRAP 1996-10, pp. 175-176.

Whitehead, P.E., J.E. **Elliott**, J. Van Oostdam and R.J. Norstrom. PCDDs and PCDFs in eagle and osprey eggs near kraft pulp mills in British Columbia, Canada. 1996. In: Proceedings, 3rd FRAP Research Workshop. Environment Canada, Vancouver, DOE FRAP 1996-10, pp. 177-178.

Wilson, L.K., J.E. **Elliott** and P.E. Whitehead. 1996. Chlorinated compounds in wildlife in the Fraser River basin. *Canadian Wildlife Service, Technical Report Series*, Delta, BC, No. 251, 73 pp.

Harding, L., J.E. **Elliott**, C.R. Stephen. 1996. Contaminants and biological measurements in mink (*Mustela vison*) and river otter (*Lontra canadensis*). *Organohalogen Compounds*, 29:102-107.

Elliott, J.E., L.K Wilson, K.E. Langelier and R.J. Norstrom. 1995. Chlorinated hydrocarbon residues and autopsy data from British Columbia bald eagles (*Haliaeetus leucocephalus*), 1986-1993. *Organohalogen Compounds*. 24: 401-404.

Whitehead, P.E., J.E. **Elliott**, J. Van Oostdam and R.J. Norstrom. 1995. PCDDs and PCDFs in eagle and osprey eggs near kraft pulp mills in British Columbia, Canada. *Organohalogen Compounds*. 24: 233-238.

Wilson, L.K., I.E. Moul, K.E. Langelier and J.E. **Elliott**. 1995. Summary of bird mortalities in British Columbia and Yukon, 1963-1994. *Canadian Wildlife Service, Technical Report Series*, Delta, BC, No. 249, 205 pp.

Harfenist, A., P.E. Whitehead, W.J. Cretney & J.E. **Elliott**. 1995. Food chain sources of polychlorinated dioxins and furans to Great Blue Herons (*Ardea herodias*) foraging in the Fraser River estuary, British Columbia. *Canadian Wildlife Service, Technical Report Series*, Delta, BC, No. 169, 26 pp.

Harding, L., G. Kaiser, P. Whitehead, R.W. Butler, J.E. **Elliott**. 1995. Factors affecting hatching, fledging and survival of offshore birds and inshore coastal birds. Proc.2nd Korea-Canada Symposium. National Fisheries University of Pusan. Pusan, Korea, 24-25 Oct 1995.

Whitehead, P.E., J.E. **Elliott**, R.J. Norstrom, M.M. Machmer, J.C. Van Oostdam & G.E.J. Smith. 1993. Chlorinated hydrocarbon levels in eggs of osprey nesting near pulp mills in British Columbia, Canada. *Organohalogen Compounds*. 12:231-234.

Sinclair, P.H. & J.E. **Elliott**. 1993. Birds and Pesticides in orchards of the south Okanagan/Similkameen region of British Columbia. *Canadian Wildlife Service, Technical Report Series*, No. 185, 54 pp.

Moul, I.E. & **Elliott**, J.E. 1993. The bird community found on British Columbia golf courses. *U.S. Golf Association, Green Section Record*, July/August, 1993, pp. 13-15.

Noble, D.G., J.E. **Elliott** and J.L. Shutt. 1993. Environmental contaminants in Canadian raptors, 1967-1989: levels and effects. *Canadian Wildlife Service, Technical Report Series*, Ottawa, ON, No. 91, 225 pp.

Elliott, J.E., K.M. Langelier, A.M. Scheuhammer, P.H. Sinclair, P.E. Whitehead. 1992. Incidence of lead poisoning in Bald Eagles and lead shot in waterfowl gizzards from British Columbia, 1988-91. *Canadian Wildlife Service, Progress Notes*, Ottawa, ON, No. 200, 7pp.

Moul, I.E. and J.E. **Elliott**. 1992. A survey of pesticide use and bird activity on selected golf courses in British Columbia. *Canadian Wildlife Service, Technical Report Series*, Delta, BC, No. 163. 103 pp.

Whitehead, P.E., R.J. Norstrom & J.E. **Elliott**. 1992. Dioxin levels in eggs of great blue herons (*Ardea herodias*) decline rapidly in response to process changes in a nearby kraft pulp mill. *Organohalogen Compounds*. 9:325-328.

Whitehead, P.E., A. Harfenist, J.E. **Elliott** & R.J. Norstrom, 1992. Levels of polychlorinated dibenzodioxins and polychlorinated dibenzofurans in waterbirds of Howe Sound, British Columbia. In: C.D. Levings, R.B. Turner & B. Ricketts (eds.) Proceedings of the Howe Sound Environmental Science Workshop, *Canadian Technical Reports in Fisheries and Aquatic Science*, No. 1879, pp. 229-238.

Moul, I.E. and J.E. **Elliott**. 1991. Birds, golf courses and turf care chemicals: preliminary results. *Proc. Western Canada Turfgrass Association Conference*, Feb. 27, 1991. Vancouver.

Whitehead, P.E., J.E. **Elliott**, R.J. Norstrom and K. Vermeer. 1990. PCDD and PCDF contamination of waterfowl in the Strait of Georgia, British Columbia, Canada, 1989-90. *Organohalogen Compounds*. 1:459-462.

Baril, A., J.E. **Elliott**, J.D. Somers, G. Erickson. 1989. Contaminants in the prey of the Peregrine Falcon in Canada. *Canadian Wildlife Service, Technical Report Series*, Ottawa, ON, No. 62, 89 pp.

Elliott, J.E., R.W. Butler, R.J. Norstrom and P.E. Whitehead. 1988. Levels of polychlorinated dibenzodioxins and polychlorinated dibenzofurans in eggs in Great Blue Herons (*Ardea herodias*) in British Columbia, 1983-87: possible effects on reproductive success. *CWS Progress Notes*, Ottawa, ON, No. 176, 7 pp.

Elliott, J.E., R.J. Norstrom, S.W. Kennedy and G.A. Fox. 1988. Trends and effects of environmental contaminants determined from analysis of archived wildlife samples. In: *Progress in Environmental Special Banking*, ed. by S.A. Wise, R. Zeisler, G.M. Goldstein. National Bureau of Standards (U.S.), Special Publication No. 740, 131-142.

Elliott, J.E., W.J. Learning and Y. Ouellette. 1987. A guide to the Canadian Wildlife Service National Registry of Toxic Chemical Residues. *Canadian Wildlife Service, Technical Report Series*, Ottawa, ON, No. 27, 57 pp.

Weseloh, D.V., J.E. **Elliott** and J.H. Olive. 1987. Herring Gull surveillance program. International Joint Commission, Windsor, ON.

Noble, D.G. and J.E. **Elliott**. 1986. Environmental contaminants in Canadian seabirds, 1967-1985: trends and effects. *Canadian Wildlife Service, Technical Report Series*, Ottawa, ON, No. 13. 275 pp.

Elliott, J.E. 1985. Specimen banking in support of monitoring for toxic contaminants in Canadian wildlife. In: S.A. Wise and R. Zeisler (eds.). *An International Review of Environmental Specimen Banking*. National Bureau of Standards (U.S.), Special Publications No. 657, 4-12.

Non-refereed professional publications, workshop proceedings and government reports (18):

Morrissey, C.A., P. Dods, C. Albert, L.K. Wilson, W. Cullen, T.D. Williams, J.E. **Elliott**. 2004. Assessing avian exposure to monosodium methanearsonate (MSMA) as used for bark beetle control in British Columbia forests. Pesticide Information Exchange. Environment Canada, Reg Prog Rep 04-02.

Wilson, L.K., S. Lee, J.E. **Elliott**. 2004. Raptor and waterfowl exposure to pesticides in agricultural ecosystems of southwestern BC. Pesticide Information Exchange. Environment Canada, Reg Prog Rep 04-02.

Elliott, J.E., L. Wilson, C. Morrissey. 2003. Assessing the impacts of MSMA (monosodium methanearsonate) for bark beetle control to forest birds in B.C. Pesticide Information Exchange. Environment Canada, Reg Prog Rep 03-01.

Wilson, L.K., S. Lee, J.E. **Elliott**. 2004. Raptor and waterfowl exposure to pesticides in agricultural ecosystems of southwestern BC. Pesticide Information Exchange. Environment Canada, Reg Prog Rep 03-01.

Ashpole, S., C.A. Bishop, B. Pauli, J.E. **Elliott**. 2004. Assessment of pesticide exposure and effect in amphibians using agricultural habitat, South Okanagan, British Columbia. Pesticide Information Exchange. Environment Canada, Reg Prog Rep 03-01.

Wilson, L.K., **Elliott** J.E and Lee, S. 2002. Pesticide poisoning of birds of prey. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 03-01.

Elliott, J.E., P. Shaw, M. Wayland, L.K. Wilson, G. Kardosi and D.C.G. Muir. 2002. Chlorinated hydrocarbons in fish and osprey from alpine and reference areas of British Columbia. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 03-01.

Gill, H., L.K. Wilson, K.M. Cheng and J.E. **Elliott**.2002. Effects of DDT and other chlorinated compounds on the reproductive success of American robins (*Turdus migratorius*) breeding in fruit orchards. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 03-01.

Christensen, J.R., C.A. Bishop, J.S. Richardson and J.E. **Elliott**. 2002. The amphibian sperm inhibition toxicological test method. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 03-01.

Gill, H., **Elliott**, J.E., C.A. Bishop, K.M. Cheng and T.D. Williams. 2002. Effects of DDE and current use pesticides on reproduction and health in zebra finches. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 03-01.

Wilson, L.K., **Elliott** J.E. 2001. Pesticide poisoning of raptors. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 02-01.

Wilson, L.K., **Elliott** J.E. 2000. Poisoning of raptors wintering on the Fraser River delta by anticholinesterase insecticides and related studies examining the process of secondary poisoning. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 01-01.

Gill, H., L,K. Wilson, K.M. Cheng, and J.E. **Elliott** J.E. 2000 . Effects of azinphos-methyl on American robins breeding in fruit orchards. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 01-01.

Elliott J.E., L.K. Wilson, L. Barjaktarovic. 1999. Pesticide poisoning of raptors in B.C. 1990-1999. *Pesticide Information Exchange Proceedings*, Environment Canada, North Vancouver, Regional Program Report 00-01.

Elliott, J.E. 1997. Chlorinated hydrocarbon contaminants in seabird eggs from the Pacific coast of Canada - evidence of atmospheric sources? *Air Quality and the Health of our Ecosystem Workshop*. Environment Canada, North Vancouver, pp 185-187.

Wilson, L.K. and J.E. **Elliott**. 1997. Poisoning of raptors wintering in the Fraser delta by anticholinesterase insecticides and conservation efforts to reduce the impact of avian species in agro-ecosystems. *Pesticide Workshop Proceedings*, Environment Canada, North Vancouver, Regional Program Report 97-05.

Water Quality Branch. 1981. MacKenzie River Basin, Water Quality Data, 1960-1979. Inland Waters Directorate, Ottawa, ON, 1080 pp.

Watt, E.R. and J.E. **Elliott**. 1981. A compendium of surveillance field procedures. International Joint Commission, Windsor, ON, 134 pp.

INTERNET PUBLISHING:

Wilson, L.K. and J.E. **Elliott**. PCB trends in cormorants. Pacific and Yukon Region Environmental Indicators. Environment Canada. Http://www.ecoinfo.org.

Wilson, L.K. and J.E. **Elliott**. Dioxin trends in great blue herons. Pacific and Yukon Region Environmental Indicators. Environment Canada. Http://www.ecoinfo.org.

Wilson, L.K. and J.E. **Elliott**. Pesticide poisoning of raptors in the Fraser Valley. Pacific and Yukon Region Environmental Indicators. Environment Canada. Http://www.ecoinfo.org.

Elliott, J.E. Population trends of bald eagles. Pacific and Yukon Region Environmental Indicators. Environment Canada. Http://www.ecoinfo.org.

Elliott, J.E. and L.K.Wilson. Contaminant trends in ospreys. Pacific and Yukon Region Environmental Indicators. Environment Canada. Http://www.ecoinfo.org.

MANUSCRIPTS SUBMITTED AND IN PREPARATION:

Hindmarch S, **Elliott**, JE. Diet of the Barred Owl in a hetrogenous urban-rural landscape. *Journal of Raptor Research*. (in review).

Rattner BA, Lazarus RS, Elliott JE, Shore RF, van den Brink N. Adverse Outcome Pathway and Risks of Anticoagulant Rodenticides to Predatory Wildlife. *Environmental Science & Technology*. (In prep).

Elliott JE, Rattner B, Shore R, van den Brink N. Rodenticides: another contaminant conundrum. *Science* (invited, in preparation).

Miller AA, **Elliott** JE, Elliott KH, Guigueno MF, Wilson LK, Lee S, Idrissi A. Spatial and Temporal trends in Brominated Flame Retardants in Seabirds from the Pacific Coast of Canada (in preparation).

Elliott JE, Elliott KH, Brogan J, Lee S, Drouillard KG. Trophic level and carbon source as factors in accumulation of PBDEs and legacy POPs in urban raptors. (in preparation).

van den Brink NW, Arblaster JA, Bowman SR, Conder JM, Drost W, **Elliott** JE, Johnson MS, Muir DCG, Natal da Luz TM, Rattner BA, Sample B, Shore RF. Use of terrestrial field studies in the derivation of bioaccumulation potential of chemicals (in preparation).

Elliott JE, Hindmarch S, Wilson LK, Maisonneuve F, Bowes V, Clegg R, Maisonneuve F. Second Generation Anti-coagulant Rodenticides are Pervasive Contaminants of Terrestrial Birds of Prey. (in preparation).

Webster KH, Harr KE, Williams TD, Albert CA, Cheng KM, Bennett DC, **Elliott** JE. The Effect Of Brodifacoum On The Prothrombin Time Of Japanese Quail: A Potential Model For Estimating Exposure In Wild Birds *Ecotoxicology* (submitted).

Wong IL, Labrecque MP, Ibuki N, Cox ME, **Elliott** JE, Beischlag TV. *p,p'*-DDT and *p,p'*-DDE repress prostate specific antigen levels in human LNCaP prostate cancer cells: potential confounding factor in clinical PSA screens. *Environmental Health Perspectives* (In review).

Currier HA, Drouillard KG, Williams TD, **Elliott** JE. Long-term environmental exposure to persistent organic pollutants in a municipal landfill, and their effects on the breeding success in European starlings (*Sturnus vulgaris*). *Environmental Toxicology and Chemistry* (in review).

Scherr H, Wong L, Williams T, Beischlag T, Letcher R, **Elliott**, JE. In Vitro Screening, and use of an Animal Model, the Zebra Finch, to Identify Possible Endocrine Disrupting Effects of TBECH. *Toxicological Science* (in preparation).

Eng, ML, **Elliott** JE, Letcher RJ, Williams TD. Methods for assessing developmental toxicity in a free-living passerine: BDE-99 effects on growth, physiology,

neuroanatomy, and photo-induced reproductive development in the European starling. *Environmental Science & Technology* (in review).

Elliott JE, Kirk D, Elliott KH, Ruelas-Inzunza E, Scheuhammer A. Mercury in forage fish and risk to fish-eating birds from sites in Mexico and Central America (in preparation).

Gill, H, T. D. Williams, C. A. Bishop, J.E. **Elliott**. Effects of *p,p'*-DDE and current-use pesticides on egg production and yolk precursor levels in the Zebra finch (*Taeniopygia guttata*) (in preparation).

Elliott, J.E. and C.J. Henny. Risky business: avian scavengers in a toxic environment. (in preparation).

Nye PE, Elliott KH, **Elliott** JE. Contaminants and reproductive success of bald eagles breeding on the Hudson River, New York (in preparation).

Harris ML, Wilson WK, Smith L, **Elliott** JE, Cheng KM. Effects of early exposure to DDT on thyroid hormones, immunity, and stress response in American robins. (in preparation).

UNPUBLISHED REPORTS:

Elliott JE, Bishop CA. 2008. Rocky Mountain Arsenal Natural Resource Damage Assessment Plan and Phase I Injuries to Wildlife, Part 2. Prepared for the State of Colorado Office of the Attorney General, Denver CO. June 2008 68 pp.

Elliott JE, Bishop CA. 2007. Rocky Mountain Arsenal Natural Resource Damage Assessment Plan and Phase I Injuries to Wildlife. Prepared for the State of Colorado Office of the Attorney General, Denver CO. June 2007. 89 pp.

Elliott, J.E., K. Grasman, S.W. Kennedy, Stratus Consulting. Evaluation of injury from PCBs to avian resources of the Hudson River: options for further study. For: The Hudson River Trustees. Albany, NY. 62 pp.

Elliott, J.E., T.W. Custer, C.M. Custer, C.A. Bishop and Stratus Consulting. Options to Investigate the Effects of Polychlorinated Biphenyls on Belted Kingfisher (Ceryle alcyon) and Spotted Sandpiper (Actitus macularia) Populations on the Hudson River, New York. For: The Hudson River Trustees. Albany NY.

Elliott, J.E. 2001. Using blood samples to monitor chlorinated hydrocarbon exposure of bald eagles on the Northern Channel Islands of California. A report to the Science Review Panel for the Northern Channel Island Bald Eagle Feasibility Study. Available on internet: http://www.darrp.noaa.gov/southwest/montrose/pdf/mon-srbelliott.pdf

Coker, C., L.K. Wilson and J.E. **Elliott**. 1997. Waterfowl mortality in agricultural fields treated with Dyfonate G (fonofos): Preliminary Report. Canadian Wildlife Service, Delta, BC, October 1997.

Wilson, L.K., S. Szeto, J.E. **Elliott** and R. Vernon. 1996. Persistence of four granular insecticides in Fraser delta soils: implications for wildlife. Canadian Wildlife Service and Agriculture Canada, Vancouver, BC, 24 pp.

Harding, L., C. Stephen and J.E. **Elliott**. 1995. Organochlorine and trace metal contaminants in mink (*Mustela vison*) and otter (*Lontra canadensis*) in the Columbia and Fraser River systems. Canadian Wildlife Service, Progress Report to the Fraser River Action Plan, 13 pp.

Elliott, J.E and I. Moul. 1994. Nest locations, breeding success and blood sampling of bald eagles on Langara Is., British Columbia, 1994. Progress Report for the Nestucca Fund. 33 pp.

Elliott, J.E., P.A. Martin, P.E. Whitehead and J.C. Van Oostdam. 1994. Chlorinated hydrocarbons in waterfowl wintering on the coast of British Columbia. 16 pp.

Elliott, J.E. and L. Wilson. Pesticide poisoning of birds of prey wintering in the Lower Fraser Valley, British Columbia. Canadian Wildlife Service (unpubl. rep.). 33pp.

Elliott, J.E., P.E. Whitehead and R.J. Norstrom. 1991. Chlorinated hydrocarbons in bald eagle eggs from the Strait of Georgia, 1990. Canadian Wildlife Service (unpubl. rep.), 5 pp.

Elliott, J.E. and P.E. Whitehead. 1989. A risk assessment for mink breeding downstream of a proposed bleached kraft pulp mill in Alberta. Prepared for the ALPAC environmental review board. 11 pp.

Whitehead, P.E., J.E. **Elliott** and R.J. Norstrom. 1989. Update on studies of dioxins in wildlife in the Strait of Georgia, 1989. Canadian Wildlife Service (unpubl. rep.), 7 pp.

Elliott, J.E. and A. Macaulay. 1984. Environmental Specimen Banking in the Great Lakes: Involvement of Canadian Agencies. Canadian Wildlife Service, Ottawa, 18 pp.

Elliott, J.E. 1983. National Specimen Bank and National Registry of Toxic Chemical Residues: Policy and Procedures. Canadian Wildlife Service. 31 pp.

Elliott, J.E., W.J. Learning, Y. Ouellette and P. Angehrn. 1981. Criteria for selecting of specimens for the National Tissue Bank. Canadian Wildlife Service, Ottawa. 20 pp.

Elliott, J.E. 1981. The Canadian Wildlife Service National Tissue Bank: Overview Report. Canadian Wildlife Service, Ottawa, 27 pp.

Nutrients Task Force. 1980. Nutrients task force report on water quality in the Ottawa River. Water Quality Branch, Inland Waters Directorate, Ottawa, 106 pp.

ABSTRACTS (243)

Elliott JE, Elliott KH, Guigueno MF, Lee S. Temporal and spatial trends in POPs in seabird eggs from the north Pacific ocean. SETAC 2013, Nashville TN, Nov 17-21, 2013.

Eng M, Williams TD, **Elliott** JE. In ovo exposure to an organophosphate flame retardant, tris(2-butoxyenthyl) phosphate, in zebra finches. SETAC 2013, Nashville TN, Nov 17-21, 2013

Eng M. Williams TD, **Elliott** JE. In ovo exposure to BDE-99 affects the song control system of a model songbird species, the zebra finch. SETAC 2013, Nashville TN, Nov 17-21, 2013

Reers, A., Beischlag, T. **Elliott** JE. The effects of organophosphate flame retardants on endocrine signaling pathways in human cancer cell lines. SETAC 2013, Nashville TN, Nov 17-21, 2013

Conder JM, van den Brink NW, Muir DCG, Arblaster JA, Bowman SR, Drost W, **Elliott** JE, Johnson MS, Natal da Luz TM, Rattner BA, Sample B, Shore RF. Use of terrestrial field data to address chemical bioaccumulation potential: Advice and research needs. SETAC 2013, Nashville TN, Nov 17-21, 2013.

Rattner B. Lazarus RA, van den Brink N. Shore R. **Elliott** JE An adverse outcome pathway for secondary poisoning of non-target wildlife by anticoagulant rodenticides. SETAC 2013, Nashville TN, Nov 17-21, 2013

Elliott JE. Raptor Ecotoxicology from Peregrines to Vultures and Beyond. Raptor Research Foundation Annual Meeting, Bariloche, Argentina, Oct 20-24 2013. Invited Plenary Address.

Elliott JE, Lee S, Wilson LK, Hindmarch S, Maisonneuve F. Terrestrial birds of prey are widely contaminated with anticoagulant rodenticides. Raptor Research Foundation Annual Meeting, Bariloche, Argentina, Oct 20-24 2013.

Elliott KH, **Elliott** JE. Foraging ecology of ospreys in British Columbia revealed by stable isotope analysis. Raptor Research Foundation Annual Meeting, Bariloche, Argentina, Oct 20-24 2013.

Hindmarch, S. **Elliott**, JE, Lee, S. (W)Hoooo Can Be Hurt by Rat Poisons… Are Metropolitan Owls At Greater Risk Than Their Rural Counterparts? Raptor Research Foundation Annual Meeting, Bariloche, Argentina, Oct 20-24 2013.

Huang AC, **Elliott** JE, Martin K. Hindmarch S. SARA-listed (Species At Risk Act) Barn Owls (*Tyto alba*) in British Columbia: Genetic Diversity, Connectivity, and Divergence**.** Raptor Research Foundation Annual Meeting, Bariloche, Argentina, Oct 20-24 2013.

Brogan

Eagles-Smith C, Evers D, Marvin-DiPasquale M, Wiener J, Ackerman J, Aiken G, Alpers C, Cline C, Dassuncao C, Davis J, Eckley C, **Elliott** JE, Flanagan C, Fleck J, Gustin M, Jackson A, Jaffe D, Johnson P, Krabbenhoft D, Lepak J, Luengen A, Morris K, Siitari K, Steffen S, Stewart R, Sunderland E, Turnquist M,

Villatoro F, Webster J, Wilson A, Wright G. Mercury Cycling, Bioaccumulation, and Risk across Western North America: a landscape scale synthesis**.** 11th International Conference on Mercury as a Global Pollutant, Glasgow, UK. Jul 28 – Aug 2, 2013.

Erratico C, Currier H, Szeitz A, Bandiera S, Covaci A, **Elliott** JE. Levels and profiles of PBDEs, PCBs, HO-PCBs, in plasma of starlings from British Columbia, Canada: first insghts into PBDEs into PBDEs oxidative metabolism by avian liver microsomes. BFR 2013, San Francisco CA, April 7-10, 2013.

Bishop CA, **Elliott** JE. Impact assessment of a rodenticide containing strychnine on wild gopher snakes (*Pituophis cotenifer deserticola*). SETAC EU 2013, Glasgow, Scotland, May 12-16, 2013.

Elliott JE, Elliott KH, Guigueno MF, Wilson LK, Lee S, Cyr F. Spatial and Temporal Patterns and Trends of Perfluorinated Carboxylates and Sulfonates in Seabird eggs from the Pacific Coast of Canada**.** SETAC EU 2013, Glasgow, Scotland, May 12-16, 2013.

Elliott JE, Elliott KH, Guigueno MF, Wilson LK, Lee S, Idrissez A. Brominated Flame Retardants: Spatial and Temporal Patterns and Trends in Seabird eggs from the Pacific Coast of Canada. SETAC EU 2013, Glasgow, Scotland, May 12-16, 2013.

Elliott JE, Wilson LK, Hindmarch S, Maisonneuve F. Anticoagulant rodenticides are pervasive contaminants of terrestrial birds of prey**.** SETAC 2012, Long Beach, CA, Nov 11-15, 2012.

Eng, M.L., Williams, T.D., MacDougall-Shackleton, S.A., Letcher, R.J., and **Elliott**, J.E. (2012). Effects of early exposure to a brominated flame retardant on development and behaviour in birds. *SETAC 2012*, Long Beach, CA, Nov 11-15, 2012.

Hindmarch SR, **Elliott** JE. Investigating the Risk of Secondary Rodenticide Poisoning to Owls Inhabiting and Foraging in Urban Landscapes of the Lower Mainland, British Columbia. *SETAC 2012*, Long Beach, CA, Nov 11-15, 2012.

Scherr H, Wong L, Williams T, Beischlag T, Letcher R, **Elliott**, JE. In Vitro Screening, and use of an Animal Model, the Zebra Finch, to Identify Possible Endocrine Disrupting Effects of TBECH. SETAC 2012, Long Beach, CA, Nov 11-15, 2012.

Wong L, **Elliott** JE, Scherr H, Beischlag TV. The effects of DDT, DDE and 1,2-dibromo-4-(1,2 dibromoethyl)cyclohexane on androgen receptor signalling in the human LNCaP prostate cancer cells. SETAC 2012, Long Beach, CA, Nov 11-15, 2012

Elliott JE, Wilson LK, Hindmarch S, Maisonneuve F. Anticoagulant rodenticides are pervasive contaminants of terrestrial birds of prey**.** SETAC 2012, Long Beach, CA, Nov 11-15, 2012.

Elliott JE, Elliott KH, Guigueno MF, Wilson LK, Lee S, Idrissi A. Seabirds are Indicators of Persistent Contaminants in the Marine Environment: Examples from the

Pacific Coast of Canada. PICES 2012 Annual Meeting. Hiroshima, Japan. Oct 12-21, 2012.

Elliott JE, Wilson LK, Hindmarch S, Maisonneuve F. Rodenticides: ongoing problems with contamination and poisoning of birds of prey. 5th North American Ornithological Conference (NAOC), Vancouver, B.C. August 14-18 2012.

Eng, M.L., Williams, T.D., MacDougall-Shackleton, S.A., Letcher, R.J., and **Elliott**, J.E. (2012). Effects of early exposure to a brominated flame retardant on behaviour and development in birds. 5th North American Ornithological Conference (NAOC), Vancouver, B.C. August 14-18 2012.

Davis, M.L., J.E., **Elliott,** and T.D., Williams. 2012. Temporal and spatial variation in the diet of Glaucous-winged Gulls (Larus glaucescens): implications for ecotoxicology monitoring. 5th North American Ornithological Conference (NAOC). Vancouver, BC, August 14-18, 2012

Nelson, C. Guertin, D., Drouillard, K.G., Cheng, K. **Elliott**, JE. Assessing the exposure and effects of persistent contaminants in river otters (*Lontra candensis*) in Victoria Harbour, Vancouver Island, British Columbia. SETAC Europe 2012. Berlin, Germany, May 20-24, 2012.

Hindmarch SR, **Elliott** JE. SETAC Europe 2012. Investigating the potential risk of secondary rodenticide poisoning to urban owls inhabiting and foraging in urban landscapes of the Lower Mainland, British Columbia. SETAC Europe, Berlin, Germany, May 20-24, 2012.

Eng, M.L., Williams, T.D., MacDougall-Shackleton, S.A., Letcher, R.J., and Elliott, J.E. (2012). Early Exposure to 2,2′,4,4′,5-Pentabromodiphenyl Ether (BDE-99) affects neuroanatomy and behavior of zebra finches. SETAC Pacific Northwest Annual Meeting, Vancouver, B.C. April 26-28 2012.

Hindmarch SR, **Elliott** JE. Investigating the potential risk of secondary rodenticide poisoning of barn owls (Tyto alba). SETAC Pacific Northwest 2012, Vancouver, British Columbia, 26-28 April 2012.

Elliott JE, Brogan J, Wilson LK, Drouillard KG. Accumulation of PBDEs and legacy POPs in urban raptors from British Columbia, Canada, 1999-2009. SETAC Pacific Northwest 2012, Vancouver, British Columbia, 26-28 April 2012.

Winter V, Williams TD, Letcher RJ, **Elliott** JE A three-generational study of *in ovo* exposure to PBDE-99 in the zebra finch. SETAC Pacific Northwest Chapter meeting, Vancouver, British Columbia, 26-28 April 2012.

Scherr, H., **Elliott**, J., Beischlag, T.V., and T.D. Williams. 2012 .In Vitro Screening, and Use of an Animal Model the Zebra Finch, to Identify Possible Endocrine Disrupting Effects of TBECH. SETAC Pacific Northwest Chapter meeting, Vancouver, British Columbia, 26-28 April 2012 .

Nelson, C. Guertin, D., Drouillard, K.G., Cheng, K. **Elliott**, J. 2012. Contaminant Exposure in Marine Foraging River Otters. SETAC, Pacific Northwest Chapter meeting. Vancouver, BC, 26-28 April 2012.

Davis, M.L., J.E., **Elliott,** and T.D., Williams. 2012. Dietary trends of the Glaucous-winged Gull: A context for interpretation of contaminant-monitoring data. Talk. SETAC, Pacific North West Chapter meeting. Vancouver, BC, April 26-28, 2012

Elliott JE, Guigeno MF, Elliott KH, Wayland M, Levac J, Shaw P, Morrissey CA, Muir DEG. Bioaccumulation of POPs in ospreys from alpine regions of western Canada. SETAC 2011, Boston MA, Nov 13-17, 2011.

Elliott JE, Bishop CA, Morrissey CA. What is forensic ecotoxicology? SETAC 2011, Boston MA, Nov 13-17, 2011.

Eng, M.L., Williams, T.D., Letcher, R.J., and **Elliott**, J.E. Impact of exposure level and condition on the maternal transfer of PDE-99 in Zebra finches (2012). SETAC 2011, Boston, MA, Nov 13-17, 2011.

Scherr, H., **Elliott,** J., Beischlag, T.V., and T.D. Williams. In Vitro Screening of TBECH in Multiple Human Cancer Cell Lines for Possible Endocrine Disrupting Effects. SETAC 2011, Boston, MA, Nov 13-17, 2011.

Holmes JV, Edson JT, Bishop CA, **Elliott** JE. The Rocky Mountain Arsenal : from environmental catastrophe to urban wildlife refuge. SETAC 2011, Boston MA, Nov 13-17, 2011.

Morrissey CA, **Elliott** JE. Toxic trees: arsenic pesticides, woodpeckers, and the Mountain Pine Beetle. SETAC 2011, Boston MA, Nov 13-17, 2011.

Davis, M.L., J.E., Elliott, and T.D., Williams. 2011. Dietary trends of the Glaucous-winged Gull on the Canadian West Coast. Talk. Salish Sea Ecosystem Conference, Vancouver, BC, October 2011.

Elliott KH, **Elliott** JE, Wilson LK, Stenerson K. Density-dependence in reproduction and survival of Bald Eagles: linkages with chum salmon. Salish Sea Ecosystem Conference, Vancouver, BC, October 2011.

Nelson, C. Guertin, D., Drouillard, K.G., Cheng, K.M., **Elliott**, J. 2011. Contaminant Exposure in Marine Foraging River Otters. Salish Seas Ecosystem Conference. October 2011.

Elliott JE, Cesh L, Elliott KH, McKinney M, Quade S, Maisonneuve F, Garcelon DK, Sandau CD, Letcher RJ, Williams TD. Polyhalogenated aromatic hydrocarbons and Metabolites: Relation to Circulating Thyroid Hormone and Retinol in Nestling Bald Eagles (Haliaeetus leucocephalus). Salish Seas Ecosystem Conference. October 2011.

Davis, M.L., J.E., Elliott, and T.D., Williams. 2011. Dietary trends of the Glaucous-winged Gull on the Canadian West Coast. Talk. Salish Sea Ecosystem Conference, Vancouver, BC, October 2011.

Elliott JE, Wilson LK**.** Insecticide Poisoning of raptors in agricultural areas of southwestern British Columbia**.** Raptor Research Foundation, Annual Meeting, Duluth, MN, USA, Oct 4-8, 2011.

Hindmarch SR, **Elliott** JE. Investigating the potential risk of secondary rodenticide poisoning of barn owls (Tyto alba) inhabiting and foraging in urban landscapes of the Lower Mainland, British Columbia. Raptor Research Foundation, Annual Meeting, Duluth, MN, USA, Oct 4-8, 2011.

Elliott KH, **Elliott** JE, Wilson LK, Stenerson K. Density-dependence in reproduction and survival of Bald Eagles: linkages with chum salmon. Raptor Research Foundation, Annual Meeting, Duluth, MN, USA, Oct 4-8, 2011.

Elliott JE, Guigeno MF, Elliott KH, Wayland M, Levac J, Shaw P, Morrissey CA, Muir DG. Are melting glaciers increasing exposure of alpine wildlife to contaminants? SETAC Europe 2011. Milan, Italy, May 15-19, 2011.

Elliott JE, Wilson LK, Drouillard KG. PBDEs and legacy POPs in hawk and falcon species from urban environments n British Columbia, Canada, 1999-2009. SETAC Europe 2011. Milan, Italy, May 15-19, 2011.

Eng ML, Williams TD, Letcher RJ, **Elliott** JE. Neuroanatomical and behavioural effects of early exposure to BDE-99 in an integrated avian laboratory and field model system. SETAC Europe 2011. Milan, Italy, May 15-19, 2011.

Eng, M.L., Williams, T.D., Letcher, R.J., and **Elliott**, J.E. (2011). Effects of early exposure to BDE-99 on growth, physiology and reproduction in zebra finches and European starlings. SETAC Europe, Milan, Italy, May 15-19 2011.

Elliott JE, Wilson LK, Drouillard KG. Accumulation of PBDEs and legacy POPs in urban raptors from British Columbia, Canada, 1999-2009. SETAC 2010, Portland OR, Nov 7-11, 2010.

Eng M, Letcher RJ, **Elliott** JE. Concentrations and effects of persistent organic pollutants in a terrestrial avian monitoring species, the European starling. SETAC 2010, Portland OR, Nov 7-11, 2010.

Lord SI, Schindler DW, **Elliott** JE. Persistent organic pollutants in common loons in western Alberta, Canada. SETAC 2010, Portland OR, Nov 7-11, 2010.

Khamzina V, Williams TD, Letcher RJ, **Elliott** JE. Validation of an egg-injection method for testing *in ovo* toxicity in a small songbird model species, the zebra finch (*Taeniopygia guttata*). SETAC 2010, Portland OR, Nov 7-11, 2010.

Elliott JE, Wilson LK, Drouillard KG. Accumulation of PBDEs and legacy POPs in urban raptors from British Columbia, Canada, 1999-2009. Raptor Research Foundation Annual Meeting, Ft Collins, CO, Sept 22-25, 2010.

Hindmarch SR, **Elliott** JE. Investigating the potential risk of secondary rodenticide poisoning of barn owls (Tyto alba) inhabiting and foraging in urban landscapes of

the Lower Mainland, British Columbia. Raptor Research Foundation Annual Meeting, Ft Collins, CO, Sept 22-25, 2010.

Davis M, **Elliott** JE, Williams TD. Spatial and historical comparisons of diet in the Glaucous-winged gull on the West Coast of Canada: context for interpretation of contaminant-monitoring data. 1st International Seabird Conference. Victoria, BC, Sept 7-11, 2010.

Elliott JE, Cesh L, Elliott KH, McKinney M, Quade S, Maisonneuve F, Garcelon DK, Sandau CD, Letcher RJ, Williams TD. Polyhalogenated aromatic hydrocarbons and Metabolites: Relation to Circulating Thyroid Hormone and Retinol in Nestling Bald Eagles (Haliaeetus leucocephalus). SETAC Europe '10, Seville, Spain, 2009. 23-27 May, 2010.

Webster KH, Harr KE., Albert CA, Williams TD, **Elliott** JE. Brodifacoum exposure in Japanese quail: the use of prothrombin time to estimate oral exposure in barn owls. SETAC '09, New Orleans, LO, 11-15 November, 2009.

Lord SI, **Elliott** JE, Scheuhammer AM, Schindler DW. Patterns of mercury contamination in Common Loons and fishes near a local source of mercury pollution. SETAC '09, New Orleans, LO, 11-15 November, 2009.

Elliott JE, Henny CJ. Risky business: avian scavengers in a toxic environment. SETAC '09, New Orleans, LO, 11-15 November, 2009.

Elliott JE, Cesh L, Elliott KH, McKinney M, Quade S, Maisonneuve F, Garcelon DK, Sandau CD, Letcher RJ, Williams TD. Polyhalogenated aromatic hydrocarbons and Metabolites: Relation to Circulating Thyroid Hormone and Retinol in Nestling Bald Eagles (Haliaeetus leucocephalus). SETAC '09, New Orleans, LO, 11-15 November, 2009.

Eng ML, Williams TD, Letcher RJ, **Elliott** JE. Long term effects of early exposure to a brominated flame retardant (PBDE-99) in birds. SETAC '09, New Orleans, LO, 11-15 November, 2009.

Gebbink WA, Burgess N, Champoux L, **Elliott** JE, Martin P, Wayland M, Weseloh C, Wilson L and Letcher RJ. 2009. Perfluoroalkyl Compounds in the Eggs of Four Species of Gulls (*Larids*) From Breeding Sites Spanning Atlantic to Pacific Canada. SETAC '09, New Orleans, LO, 11-15 November, 2009.

Martin P, Burgess N, Champoux L, **Elliott** JE, Wayland M, Weseloh C, Wilson L and Letcher RJ. 2009. Brominated Flame Retardants in the Eggs of Four Species of Gulls and two terrestrial sentinel species Spanning Atlantic to Pacific Canada. Aquatic Toxicology Workshop, La Malbaie, Quebec, 27-30 September, 2009.

Martin P, Burgess N, Champoux L, **Elliott** JE, Wayland M, Weseloh C, Wilson L and Letcher RJ. 2009. Brominated Flame Retardants in the Eggs of Four Species of Gulls and two terrestrial sentinel species Spanning Atlantic to Pacific Canada. 11th Annual Workshop on Brominated Flame Retardants, Ottawa, Ontario 19-20, 2009.

Elliott JE, Iwaniuk A. **I**mpact of contaminants in birds subsequent to early developmental exposure: a recent example of effects on the brain from field exposure to DDT. SETAC Europe '09, Goteborg Sweden, May 29 – Jun 2 2009.

Elliott JE, Guertin, D. Assessing the exposure and effects of persistent contaminants in river otters (*Lontra candensis*) using non-intrusive sampling. SETAC Europe '09, Goteborg Sweden, May 29 – Jun 2 2009.

Webster KH, Harr K E., Albert C A, Williams T D, Elliott J E. Brodifacoum exposure in Japanese quail: the use of prothrombin time to estimate oral exposure in barn owls. SETAC Pacific Northwest. April 2009.

Elliott JE, Harris ML. Polychlorinated biphenyls, dibenzo-*p*-dioxins and dibenzofurans in wild birds. *SETAC '08*. Tampa FL, Nov 16-20, 2008.

Guertin D, Harestad A, Ben-David M, **Elliott** JE. Combining non-invasive techniques to investigate the exposure of individual river otters to environmental contaminants. *SETAC '08*. Tampa FL, Nov 16-20, 2008.

Bishop CA, De Jong Westman A, Ashpole S, McKibben R, VanAggelen G, McNeil K, Cheng K, Edwards M, **Elliott** JE. Is amphibian survival probable in agricultural landscapes? How we continue to stress amphibian populations in beautiful British Columbia and the rest of the world. *SETAC '08*. Tampa FL, Nov 16-20, 2008.

Henny CJ, **Elliott** JE. Toxicology, Raptor Research and Management Techniques. *Raptor Research Foundation Annual Meeting*. Sept 24-28, Missoula MN.

Elliott JE, Iwaniuk A. Early exposure and later effects of contaminants in birds with an example of DDT effects on the brain of a songbird. AOU '08, Portland OR, Aug 2008.

Elliott, JE, CM Morrissey, P. Dods, C. Albert, W. Cullen, TD Williams. Risk to insectivorous birds inhabiting forested areas treated with MSMA (monosodium methanearsonate) for mountain pine beetle suppression in British Columbia. *SETAC Europe '08*, Warsaw, May 25-29, 2008.

Elliott, J.E., C.A. Morrissey, M. Wayland, L.K. Wilson, S. Lee, D.C.G. Muir. Bioaccumulation of chlorinated hydrocarbons in relation to productivity and dietary factors in ospreys breeding in alpine regions of western Canada. *SETAC '07*, Milwaukee, WI, Nov 11-15, 2007.

Wilson, L.K., M.L. Harris, J.E. **Elliott**. Properties of Blood, Porphyrins and Exposure to Organic Contaminants in Surf Scoters (*Melanitta perspicillata*) Over-wintering on the South Coast of British Columbia, Canada. *SETAC '07*, Milwaukee, WI, Nov 11-15, 2007.

Best, D.A., K.H. Elliott, W.W. Bowerman, M. Shieldcastle, S. Postupalsky, P.E. Nye, T.J. Kubiak, D. Tillitt, J.E. **Elliott**. Assessing reproductive success, developmental anomalies, eggshell parameters, and environmental contaminants in Bald Eagles

(*Haliaeetus leucocephalus*) from the Great Lakes, USA. *SETAC Europe* '06, Porto, Portugal, May 20-24, 2007.

Elliott, JE, LK Wilson. Pesticide poisoning of wildlife in the Fraser River Delta. *North West Vertebrate Society Meeting*. Victoria, BC. Mar 7-8 2007.

Elliott, J.E., M.L. Harris, L.K. Wilson, B.D. Smith, J. Maguire. Butyltins, trace metals and morphological variables in surf scoter (*Melanitta perspicillata*) wintering on the south coast of British Columbia, Canada. *Puget Sound Georgia Basin Research Conference*, Vancouver BC, Mar 26-30, 2007.

Guertin, D., A. Harestad, J.E. **Elliott**, Use of faecal samples for sampling contaminants and populations of river otter in the Georgia Basin. *Puget Sound Georgia Basin Research Conference*, Vancouver BC, Mar 26-30, 2007.

Wilson, L.K. J.E. **Elliott**, A. Birmingham, M. McAdie, and P. Mineau. Fonofos poisons raptors and waterfowl several months after labelled application. *Puget Sound Georgia Basin Research Conference*, Vancouver BC, Mar 26-30, 2007.

Best, D.A., K.H. Elliott, W.W. Bowerman, M. Shieldcastle, S. Postupalsky, P.E. Nye, T.J. Kubiak, D. Tillitt, J.E. **Elliott**. Reproductive success, developmental anomalies, eggshell parameters, and environmental contaminants in Bald Eagles (*Haliaeetus leucocephalus*) from the Great Lakes, USA. *SETAC '06*. Montreal QU, Nov 15-20, 2006.

Ashpole, S., C.A. Bishop, J.E. **Elliott**. Pesticides and amphibian populations in the south Okanagan region of British Columbia, Canada. *SETAC '06*. Montreal QU, Nov 15-20, 2006.

Wilson, L.K. J.E. **Elliott**, A. Birmingham, M. McAdie, and P. Mineau. Fonofos poisons raptors and waterfowl several months after labelled application. *SETAC '06*. Montreal QU, Nov 15-20, 2006.

Albert, C., C.M. Morrissey, W. Cullen, V. Lai, T.D. Williams, J.E. **Elliott**. Assessing woodpecker exposure and risk from arsenic based pesticide (monomethanearsonate) during the mountain pine beetle epidemic in British Columbia. *SETAC '06*, Montreal, QU, Nov 5-9, 2006.

Elliott, J.E., C.M. Morrissey, C.J. Henny, E.I. Ruelas, P. Shaw. Use of Satellite telemetry to track migrating ospreys and exposure to contaminants on the wintering grounds. *AOU '06*, Veracruz, Mexico, Oct 3-7, 2006.

Albert, C., C.M. Morrissey, P. Dods, C. Albert, W. Cullen, T.D. Williams, J.E. **Elliott**. Woodpecker exposure to monomethanearsonate (MSMA) for bark beetle control in British Columbia pine forests. *AOU '06*, Veracruz, Mexico, Oct 3-7, 2006.

Albert, C., J.E. **Elliott**, J.E., T.D. Williams, C.M. Morrissey, W. Cullen. Assessing the uptake, elimination and toxicity of monomethanearsonate (MSMA) use for bark beetle control in adult and nestling zebra finches. *SETAC Europe 2006*, The Hague, May 7-11, 2006.

Elliott, J.E., C.M. Morrissey, P. Dods, C. Albert, W. Cullen, T.D. Williams. Woodpecker exposure to monomethanearsonate (MSMA) for bark beetle control in British Columbia pine forests. *SETAC Europe 2006*, The Hague, May 7-11, 2006.

Elliott, J.E., C.M. Morrissey, C.J. Henny, E.I. Ruelas, P. Shaw. Satellite telemetry of migrating ospreys and exposure to contaminants on the wintering grounds. *SETAC '05*, Baltimore, MD, Nov 11-15, 2005.

Cesh, L.S. J.E. **Elliott**, T.D. Williams, D.K. Garcelon, R.J. Letcher. The effects of PCBs, PBDEs and their hydroxylated metabolites on thyroid function and vitamin A levels in bald eagles. *SETAC '05*, Baltimore, MD, Nov 11-15, 2005.

Albert, C.A., C.A. Morrissey, J.E. **Elliott**, W.R. Cullen, T.D. Williams. Assessing uptake, elimination and toxicity of monosodium methanearsonate (MSMA) used for bark beetle control in adult and nestling zebra finches. *SETAC '05*, Baltimore, MD, Nov 11-15, 2005.

Morrissey, C.A., P.L Dods, T.D. Williams, W.R. Cullen, J.E. **Elliott**. Woodpecker exposure to monosodium methanearsonate (MSMA) as used for bark beetle control in British Columbia pine forests. *SETAC '05*, Baltimore, MD, Nov 11-15, 2005.

Elliott, J.E., L.K. Wilson, K.H. Elliott. Raptor poisonings in southwestern British Columbia: lessons for species conservation elsewhere. *American Ornithologist Union 2005*. Santa Barbara CA, Aug 23-27, 2005.

Morrissey, C.M., P. Dods, C. Albert, T.D. Williams, W. Cullen, J.E. **Elliott**. Woodpecker foraging behaviour in bark beetle infested forests – effects of pesticide treatment. *American Ornithologist Union 2005*. Santa Barbara CA, Aug 23-27, 2005.

Elliott, J.E., L.K. Wilson, P. Mineau, M. McAdie. An assessment of the role of poisoning in mortality of birds of prey in south western British Columbia. *Wildlife Society, Northwest Chapter*, Fort Worden, WA, Apr 20, 2005.

Elliott, J.E., L.K. Wilson and B. Wakeford. Trends in polybrominated diphenyl ethers in eggs of aquatic and marine birds from British Columbia, Canada, 1979-2002. *Puget Sound Georgia Basin Research Conference*. Seattle WA, Mar 29-31, 2005.

Elliott, J.E., L.K. Wilson, P. Mineau, M. McAdie. Poisoning of birds of prey by anti-cholinesterase insecticides in agricultural areas of south western British Columbia. *Puget Sound Georgia Basin Research Conference*. Seattle WA, Mar 29-31, 2005.

Elliott, J.E., P.A. Martin, L.K. Wilson, B.D. Smith. An assessment of chlorinated hydrocarbon effects on development of wild mink (*Mustela vison*) from Ontario and British Columbia, Canada. *Puget Sound Georgia Basin Research Conference*. Seattle WA, Mar 29-31, 2005.

Cesh, L.S. J.E. **Elliott**, T.D. Williams, D.K. Garcelon, R.J. Letcher. The effects of PCBs, PBDEs and their hydroxylated metabolites on thyroid function and vitamin A

levels in bald eagles. *Puget Sound Georgia Basin Research Conference*. Seattle WA, Mar 29-31, 2005.

Elliott, J.E., L.K. Wilson, P. Mineau, M. McAdie. An analysis of poisoning in raptors. *Wildlife Rehabilitators of British Columbia, AGM*, Campbell River, BC, Mar 19, 2005.

Elliott, J.E., L.K. Wilson, P. Mineau, M. McAdie. Poisoning of birds of prey by anti-cholinesterase insecticides in agricultural areas of south western British Columbia. *Setac '04*. Portland OR, Nov 14-16, 2004.

P.A. Martin, L.K. Wilson, B.D. Smith, J.E. **Elliott**,. Chlorinated hydrocarbon effects on development of wild mink (*Mustela vison*) from Ontario and British Columbia, Canada. *Setac '04*. Portland OR, Nov 14-16, 2004.

Morrissey, C.A., T.D. Williams, C. Albert, J.E. **Elliott**. Effects of MSMA on cavity nesting birds in British Columbia. *Setac '04*. Portland OR, Nov 14-16, 2004

Cesh, L., D. Garcelon, T.D. Williams, J.E. **Elliott**. Effects of plasma lipid content on chlorinated hydrocarbon concentrations in bald eagles. *Setac '04*. Portland OR, Nov 14-16, 2004.

Elliott, J.E., M.J. Miller, L.K. Wilson. An assessment of chlorinated hydrocarbon concentrations in prey of peregrine falcons. *SETAC-Europe 14th Annual Meeting*. Prague, Czech Republic. Apr 18-22, 2004.

Elliott, J.E., P.A. Martin, L.K. Wilson, B.D. Smith. An assessment of chlorinated hydrocarbon effects on development of wild mink (*Mustela vison*) from Ontario and British Columbia, Canada. *SETAC-Europe 14th Annual Meeting*. Prague, Czech Republic. Apr 18-22, 2004.

Ashpole, S.L., C. A. Bishop, J.E. **Elliott** and L.K. Wilson. Pesticide exposure and effect in amphibians using agricultural habitat, South Okanagan, British Columbia. *Species at Risk 2004*. Victoria BC, Mar 1-5, 2004 .

Elliott, J.E., L.K.Wilson, R.J. Norstrom and M.L. Harris. 2003. Chlorinated contaminant trends in indicator species, great blue herons and double-crested cormorants in the Strait of Georgia, 1973-2000. *SETAC '03*. Austin, TX, Nov 9 – 13, 2003.

Elliott, J.E. Chlorinated hydrocarbons, trace elements and stable isotope ratios in pelagic seabirds from the North Pacific Ocean. *SETAC '03*. Austin, TX, Nov 9 - 13, 2003.

Gill, H., **Elliott**, J.E., C.A. Bishop, K.M. Cheng and T.D. Williams. Effects of DDE and current use pesticides on reproduction and health in zebra finches (*Taeniopygia guttata*). *SETAC '03*. Austin, TX, Nov 9 -13, 2003.

Morrissey, C.A., L.I. Bendell-Young and J.E. **Elliott.** Assessing exposure to trace metals in mountain streams using the diet, feathers and feces of the American Dipper. *SETAC '03*. Austin, TX, Nov. 8-13, 2003.

Morrissey, C.A., L.I. Bendell-Young and J.E. **Elliott**. Contributions from atmospheric deposition to food chain biomagnification in the Chilliwack watershed of British Columbia, Canada. *Setac '03*, Austin, Texas. Nov. 8-13, 2003.

Elliott, J.E., L.K.Wilson, R.J. Norstrom and M.L. Harris. 2003. Chlorinated contaminant trends in indicator species, great blue herons and double-crested cormorants in the Strait of Georgia, 1973-2000. *SETAC ASIA-PACIFIC '03*. Christchurch, New Zealand. Sept 28 – Oct 1, 2003.

Elliott, J.E. Chlorinated hydrocarbons, trace elements and stable isotope ratios in pelagic seabirds from the North Pacific Ocean. *SETAC ASIA-PACIFIC*, Christchurch, New Zealand, Sept 28 – Oct 1, 2003.

Ashpole, S.L.,C. A. Bishop, B. Pauli and J.E. **Elliott**. Assessment of pesticide exposure and effect in amphibians using agricultural habitat, South Okanagan, British Columbia. *Canadian Amphibian and Reptile Conservation Network Annual Meeting*. Pele Island, ON. Sept 18-21, 2003.

Elliott, J.E., P. Shaw, S. Lee, M. Wayland, L. Wilson and D. Muir. An assessment of sources and effects of contaminants in osprey (*Pandion Haliaetus*) breeding in alpine areas of western Canada. *6th World Conference on Birds of Prey and Owls.* Budapest, Hungary, May 18-23, 2003.

Dods, P., E. Birmingham, M.G. Ikonomou, T. Williams and J. **Elliott**. Exposure of tree swallows (*Tachycineta bicolour*) to nonylphenol ethoxylates and other contaminants at a wastewater treatment plant. *Georgia Basin/Puget Sound Research Conference*, Vancouver, BC, Mar 31 – Apr 3, 2003.

Christensen, J.R., C.A. Bishop, J.S. Richardson and J.E. **Elliott**. The amphibian sperm inhibition toxicological test method. *Georgia Basin/Puget Sound Research Conference*, Vancouver, BC, Mar 31 – Apr 3, 2003.

Elliott, J.E. and L.K. Wilson. 2003. Chlorinated hydrocarbons and heavy metals in river otter and mink samples from coastal British Columbia. *Georgia Basin/Puget Sound Research Conference*, Vancouver, BC, Mar 31 – Apr 3, 2003.

Elliott, J.E., L.K.Wilson, R.J. Norstrom and M.L. Harris. 2003. Chlorinated contaminant trends in indicator species, great blue herons and double-crested cormorants in the Strait of Georgia, 1973-2000. *Georgia Basin/Puget Sound Research Conference*, Vancouver, BC, Mar 31 – Apr 3, 2003.

Morrissey, C.A., L.I. Bendell-Young and J.E. **Elliott**, Migration within a watershed influences contaminant profiles of American dippers. *Georgia Basin/Puget Sound Research Conference*, Vancouver, BC, Mar 31 – Apr 3, 2003.

Wilson, L.K. and J.E. **Elliott**. Contaminants in surf scoters wintering in the Strait of Georgia, BC, Canada. *Georgia Basin/Puget Sound Research Conference*, Vancouver, BC, Mar 31 – Apr 3, 2003.

Elliott, J.E., P. Shaw, M. Wayland, L.K. Wilson, G. Kardosi and D.C.G. Muir. Chlorinated hydrocarbons in fish and osprey from alpine and reference areas of British Columbia. *SETAC 'O2*. Salt Lake City, UT, Nov 16-20, 2002.

Morrissey, C.A., L.I. Bendell-Young and J.E. **Elliott**, Migration within a watershed influences contaminant profiles of American dippers. *SETAC 'O2*. Salt Lake City, UT, Nov 16-20, 2002.

Weech, S.A., A.M. Scheuhammer and J.E. **Elliott**. Blood mercury levels and reproductive success of bald eagles (*Haliaeetus leucocephalus*) breeding in the Pinchi Lake region of British Columbia, Canada, 2000 and 2001. *SETAC 'O2*. Salt Lake City, UT, Nov 16-20, 2002.

Christensen, J.R., J.S. Richardson, C.A. Bishop, and J.E. Elliott. Effects of nonylphenol and tail resorption and metamorphic staging in *Rana catasbeiana* tadpoles. *SETAC 'O2*. Salt Lake City, UT, Nov 16-20, 2002.

Gill, H. J.E. **Elliott**, C.A. Bishop and T.D. Williams. Effects of exposure to *p,p'*-DDE and azinphos methyl on brain and plasma cholinesterase activity and general health in zebra finches (*Taeniopygia guttata*). *SETAC 'O2*. Salt Lake City, UT, Nov 16-20, 2002.

Smith, L.K., J.E. Elliott, L.K. Wilson, K.M. Cheng. Effects of DDT exposure on immune response in American Robins from orchards of the Okanagan Valley, British Columbia. *Setac '03*. Salt Lake City, UT, Nov 16-20, 2002.

Elliott, J.E. and L.K. Wilson, Contaminants in surf scoters wintering in the Strait of Georgia. International Seaduck Conference. Victoria, BC. Nov 6 -9, 2002.

Elliott, J.E., P. Shaw, M. Wayland, L.K. Wilson, G. Kardosi and D.C.G. Muir. Chlorinated hydrocarbons in fish and osprey from alpine and reference areas of British Columbia. *29th Aquatic Toxicity Workshop*, Whistler, BC, Oct 21-23, 2002.

Morrissey, C.A., L.I. Bendell-Young and J.E. **Elliott**, Migration within a watershed influences contaminant profiles of American dippers. *29th Aquatic Toxicity Workshop*, Whistler, BC, Oct 21-23, 2002.

Weech, S.A., A.M. Scheuhammer and J.E. **Elliott**. Blood mercury levels and reproductive success of bald eagles (*Haliaeetus leucocephalus*) breeding in the Pinchi Lake region of British Columbia, Canada, 2000 and 2001. *29th Aquatic Toxicity Workshop*, Whistler, BC, Oct 21-23, 2002.

Christensen, J.R., C.A. Bishop, J.S. Richardson and J.E. **Elliott**. The amphibian sperm inhibition toxicological test method.*29th Aquatic Toxicity Workshop*, Whistler, BC, Oct 21-23, 2002.

Dods, P., E. Birmingham, M.G. Ikonomou, T. Williams and J. **Elliott**. 2002. Exposure of tree swallows (*Tachycineta bicolour*) to nonylphenol ethoxylates and other contaminants at a wastewater treatment plant. *29th Aquatic Toxicity Workshop*, Whistler, BC, Oct 21-23, 2002.

Elliott, J.E. Chlorinated hydrocarbons in otter (*Lutra canadensis*) scats from the coast of British Columbia. PCBs in Puget Sound and Georgia Basin Ecosystem. Friday Harbour, WA, Sept 16 – 19, 2002.

Elliott, J.E., L.K.Wilson, R.J. Norstrom and M.L. Harris. 2003. Chlorinated contaminant trends in indicator species, great blue herons and double-crested cormorants in the Strait of Georgia, 1973-2000. PCBs in Puget Sound and Georgia Basin Ecosystem. Friday Harbour, WA, Sept 16 – 19, 2002.

Elliott, J.E. P. Shaw, M. Wayland, L. Wilson`, G. Kardosi, and D. Muir. Chlorinated hydrocarbons in fish and osprey from alpine and reference areas of British Columbia. *Ecological and Earth Sciences in Mountain Areas*, Banff, AB, Sept 6-10, 2002. (Invited plenary lecture).

Elliott, J.E. and M.L. Harris. An Ecotoxicological assessment of chlorinated hydrocarbon effects on bald eagle populations. *Setac '01*, Baltimore, MD, Nov 11-15, 2001.

Wilson, L.K. L. Barjactarovic, J.E. **Elliott**, A.M. Scheuhammer. Heavy metals and metallotheionein in surf scoters from British Columbia and the Yukon. *Setac '01*, Baltimore, MD, Nov 11-15, 2001.

Smith, L.K., J.E. **Elliott**, L.K. Wilson and K.M. Cheng. Effects of DDT exposure on American robins from orchards of the Okanagan valley, British Columbia. *Setac '01*, Baltimore, MD, Nov 11-15, 2001.

Christensen, J.R., C.A. Bishop, J.S. Richardson, J.E. Elliott, K.J. Hall and G.C. van Aggelen. Amphibian sperm inhibition toxicological test method. *Setac '01*, Baltimore, MD, Nov 11-15, 2001.

Gill, H., L.K. Wilson, K.M. Cheng and J.E. **Elliott**. An assessment of DDT and other chlorinated compounds on the reproductive success of American robins (*Turdus migratorius*) breeding in fruit orchards. (in preparation) (grad student paper). *Setac '01*, Baltimore, MD, Nov 11-15, 2001.

Weech, S.A., J.E. **Elliott** and L.K. Wilson. Mercury residues in bald eagles found dead or dying in British Columbia. (grad student paper). *Setac '01*, Baltimore, MD, Nov 11-15, 2001.

Elliott, J.E. and L.K. Wilson. Contaminants in surf scoters wintering in the Strait of Georgia, British Columbia, Canada. *North Pacific Marine Science Organization (PICES) 10th Annual Meeting*. Victoria, BC, Oct 5-13, 2001.

Elliott, J.E. and M.L. Harris. An Ecotoxicological assessment of chlorinated hydrocarbon effects on bald eagle populations. *4th Eurasian Conference on Raptors*, Seville, Spain, Sept 25-29, 2001.

Henny, C.J., J.L. Kaiser, R.A. Grove. V. Raymond Bentley and J.E. **Elliott**. Biomagnification factors (fish to osprey eggs) for PCDDs, PCDFs, congener-specific PCBs and OC pesticides. *4th Eurasian Conference on Raptors*, Seville, Spain, Sept 25-29, 2001.

Elliott, J.E. Chlorinated hydrocarbons in otter (*Lutra canadensis*) scats from the coast of British Columbia. *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Gill, H., L.K. Wilson, K.M. Cheng, J.E. **Elliott.** Effects of Azinphos-Methyl on American Robins Breeding in Fruit Orchards. *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Wilson, L.K., J.E. **Elliott**, S. Trudeau, S.M. Scheuhammer. Contaminants in seaducks wintering in the Strait of Georgia. *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Loveridge, R., J.E. **Elliott**, C.A. Bishop, C.J. Kennedy. The effects of agricultural effluents on red-legged frog (*Rana aurora*) in the Fraser Valley. *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Smith, L.K., K.M. Cheng, L.K. Wilson, J.E. **Elliott**. Effects of DDT exposure on behaviour and reproduction in the American robin (*Turdus migratorius*). *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Peterson, C.A., L.I. Bendell-Young, J.E. **Elliott**, Trace Metals and organic contaminants from atmospheric deposition in American dippers and their diet. *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Bennett, D.C., M.R. Hughes, J.E. **Elliott**, A.M. Scheuhammer, J.E. Smits. Sex influences effects of cadmium on osmoregulation in pekin ducks. *SETAC 2000*, Nashville, TN, Nov 12-16, 2000.

Newson, S.C., C.D. Sandau, J.E. **Elliott**, S.B. Brown and R.J. Norstrom. PCBs and hydroxylated metabolites in bald eagle plasma - Comparison of thyroid hormone and retinol levels. International Conference on Environmental Chemistry, Ottawa ON, May 7-11, 2000.

Wilson, L.K. and J.E. **Elliott**. Waterfowl mortality in agricultural fields treated with dyfonate G (fonofos) and risk of poisoning to raptors. *SETAC '99*, Philadelphia PA, Nov 14-18, 1999.

Peterson, C.A., J.E. **Elliott** and S.L. Lee. Raptor scavenging on waterfowl carcasses in the Fraser delta of British Columbia. *SETAC '99*, Philadelphia PA, Nov 14-18, 1999.

Sandau, C.D., S.C. Newson, J.E. **Elliott**, and R.J. Norstrom. PCBs and their hydroxylated metabolites in bald eagle plasma and comparison to thyroid hormone levels. *SETAC '99*, Philadelphia PA, Nov 14-18, 1999.

Elliott, J.E., L.K. Wilson, C.J. Henny, S. Trudeau, S.W. Kennedy, F.A. Leighton, K.M. Cheng. Biological effects of chlorinated hydrocarbon contaminants in osprey chicks. *Raptor Research Foundation Annual Meeting*, La Paz, MX, Nov 3-7, 1999.

Wilson, L.K. and J.E. **Elliott**. Waterfowl mortality in agricultural fields treated with dyfonate G (fonofos) and risk of poisoning to raptors. *Raptor Research Foundation Annual Meeting*, La Paz, MX, Nov 3-7, 1999.

Peterson, C.A., J.E. **Elliott** and S.L. Lee. Raptor scavenging on waterfowl carcasses in the Fraser delta of British Columbia. *The Wildlife Society 6th Annual Conference*. Austin TX, Sept 7-11, 1999.

Wilson, L.K. and J.E. **Elliott**. Waterfowl mortality in agricultural fields treated with dyfonate G (fonofos) in the Fraser delta. *The Wildlife Society 6th Annual Conference*. Austin TX, Sept 7-11, 1999.

Peterson, C.A., J.E. **Elliott** and S.L. Lee. Raptor scavenging on waterfowl carcasses in the Fraser delta of British Columbia. *Raptor Research Foundation Annual Meeting*, La Paz, MX, Nov 3-7, 1999.

Elliott, J.E., L.K. Wilson, C.J. Henny, S. Trudeau, S.W. Kennedy, F.A. Leighton, K.M. Cheng. Biological effects of chlorinated hydrocarbon contaminants in osprey chicks. *SETAC '98*. Charlotte, NC, Nov 15-20, 1998.

Wilson, L.K., J.E. **Elliott**, M.M. Machmer, C.J. Henny and R.J. Norstrom. Trends and patterns of chlorinated hydrocarbon contaminants in ospreys from the Pacific Northwest. *SETAC '98*. Charlotte, NC, Nov 15-20, 1998.

Elliott, J.E. and C.E. Gill. Influence of contaminants and food supply on productivity of bald eagles. *International Ornithological Congress*. Durban, South Africa, Aug. 16-22, 1998.

Henny, C.J., P. Mineau, J.E. **Elliott** and B. Woodbridge. Bird poisonings and current insecticide use - what do isolated kill reports mean to populations? *International Ornithological Congress*. Durban, South Africa, Aug. 16-22, 1998.

Elliott, J.E. and C.E. Gill. Environmental contaminants in fish-eating birds from the Strait of Georgia, BC. *Environmental contaminants and marine mammal health: research applications*. Sidney, BC, April 17-19, 1998.

Gill, C.E., J.E. **Elliott** and L. Bendell-Young. Environmental contaminants, food availability, and reproduction of bald eagles on Vancouver Island, BC. *North American Ornithological Congress*. St. Louis, MO, April 8-12, 1998.

Elliott, J.E., C.J. Henny, M.L. Harris, L.K. Wilson and R.J. Norstrom. Chlorinated hydrocarbons in livers of mink and river otter from the Columbia and Fraser Rivers, 1990-91. *SETAC '97*. San Francisco, Ca., Nov. 16-20, 1997.

Martin, P.A., L.K. Wilson, J.E. **Elliott**, P. Mineau and K. Cheng. Exposure of California quail to Organophosphorus insecticides in orchards in the Okanagan Valley, BC. *SETAC '97*. San Francisco, Ca., Nov. 16-20, 1997.

Wilson, L.K. and J.E. **Elliott.** Monitor avian wildlife mortality - A case study: British Columbia and Yukon, Canada, 1963-94. *SETAC '97.* San Francisco, Ca., Nov. 16-20, 1997.

Gill, C.E., J.E. **Elliott** and L. Bendell-Young. Effects of contaminants and food availability on bald eagles nesting near a bleached kraft pulp mill. *SETAC '97*. San Francisco, Ca., Nov. 16-20, 1997.

Pettit, K.E., J.E. **Elliott**, C.A. Bishop and K. Cheng. Effects of agricultural pollutants on herptiles breeding in the lower Fraser valley of BC. *SETAC '97*. San Francisco, Ca., Nov. 16-20, 1997.

Pettit, K.E., J.E. **Elliott**, C.A. Bishop and K. Cheng. Effects of agricultural pollutants on herptiles breeding in the lower Fraser valley of BC. *Canadian Reptile and Amphibian Conservation Network*, 2nd Annual Meeting, Wolfville NS, Oct. 3-6.

Elliott, J.E and L.K. Wilson. Raptors, pesticides and farmland conservation issues in the Fraser delta. *Soc. Conserv. Biol. Annual Meeting*, Victoria, BC, June 6-9, 1997.

Elliott, J.E., L.K. Wilson, K.E. Langelier and P. Mineau. Effects of anticholinesterase insecticides on raptors wintering in southwestern British Columbia, Canada. *2nc Internat. Confer. on Raptors*, Urbino, Italy, Oct. 2-5, 1996.

Mineau, P., J.E. **Elliott**, L.K. Wilson, C.A. Brassard, L.A. Lyon, M.R. Fletcher, S.L. Porter, T. Bollinger, N.J. Thomas, L.C. Glaser, C.J. Henny and D.A. Kirk. Birds of prey and cholinesterase inhibiting pesticides - an emerging global concern. *2nd Internat. Confer. on Raptors*, Urbino, Italy, Oct. 2-5, 1996.

Elliott, J.E. and R.J. Norstrom. Influence of contaminants and food supply on bald eagle reproductive success. *SETAC '96*, Washington, DC, Nov. 1996.

Martin, P.M. and J.E. **Elliott**. Levels of chlorinated hydrocarbon contaminants in waterfowl of British Columbia and the Yukon territory: 1982-1992. *SETAC '96*, Washington, DC, Nov. 1996.

Wilson, L.K., J.E. **Elliott**, S. Szeto, R. Vernon. Degradation of four granular insecticides in Fraser delta soils and implications for wildlife poisonings. *SETAC '96*, Washington, DC, Nov. 1996.

Harding, L., J.E. **Elliott** and C. Stephen. Contaminants and biological measurements in mink (*Mustela vison*) and river otter (*Lontra canadensis*). *DIOXIN '96*. Amsterdam, Netherlands. Aug. 1996.

Elliott, J.E., L.K. Wilson, K.E. Langelier and P. Mineau. Secondary poisoning of raptors by anticholinesterase insecticides in southwestern British Columbia. *AOU/RRF Annual Meeting*, Boise, ID, Aug. 13-17, 1996.

Mineau, P., J.E. **Elliott**, C. Brossard, N.J. Thomas, L.C. Glazer, L.A. Lyon, M.R. Fletcher, S.L. Porter, R. Bollinger, C.J. Henny, D.A. Kirk. Assessing the vulnerability of birds of prey to cholinesterase-inhibiting pesticides. *AOU/RRF Annual Meeting*, Boise, ID, Aug. 13-17, 1996.

Howald, G.R., P. Mineau, J.E. **Elliott** and K.M. Cheng. Risk of wildlife poisoning from brodifacoum control of Norway rats in a seabird colony. *AOU/RRF Annual Meeting*, Boise, ID, Aug. 13-17, 1996. (graduate student paper).

Tomlin, A., R. Protz, R. Martin, J.E. **Elliott**, C.A. Bishop, L.K. Wilson, K. Henning and J. Johnston. Heavy metal and pesticide sequestration and transport by earthworms in soil ecosystem compartments of Canadian orchards. *Ecological Monitoring and Assessment Network 2nd National Science Meeting*, Halifax NS, Jan. 17-20, 1996.

Elliott, J.E., L.K. Wilson, K.M. Langelier, P. Mineau. Secondary poisoning of birds of prey by anticholinesterase insecticides. *SETAC '95*, Vancouver, BC, Nov. 5-9, 1995.

Wilson, L.K., J.E. **Elliott**, C.A. Bishop, A. Tomlin. Soil persistence and food chain transfer of DDT to American robins. *SETAC '95*, Vancouver, BC, Nov. 5-9, 1995.

Howald, G.R.H., J.E. **Elliott**, P. Mineau, K.M. Cheng. Risk of wildlife poisoning by brodifacoum control of rats on a seabird colony. *SETAC '95*, Vancouver, BC, Nov. 5-9, 1995. (graduate student paper).

Hooper, M.J., K.A. Hunt, K. Johnson, D.M. Bird, C.A. Bishop and J.E. **Elliott**. Organophosphate insecticides in American kestrels (*Falco sparverius*): toxicity, toxicokinetics and field applications. *Raptor Research Foundation Annual Meeting*, Duluth MS, Nov. 1-4, 1995.

Harding, L., G. Kaiser, P. Whitehead, R.W. Butler and J.E. **Elliott**. Factors affecting hatching, fledging and survival of offshore and inshore coastal birds. 2nd Korea-Canada symposium on marine environment and marine resources. Seoul, Korea, Oct. 24-25, 1995.

Elliott, J.E., L.K Wilson, K.E. Langelier and R.J. Norstrom. Chlorinated hydrocarbon residues and autopsy data from British Columbia bald eagles (*Haliaeetus leucocephalus*), 1986-1993. *DIOXIN '95*, Edmonton, AB, Aug. 20-24, 1995.

Whitehead, P.E., J.E. **Elliott**, J. Van Oostdam and R.J. Norstrom. PCDDs and PCDFs in eagle and osprey eggs near kraft pulp mills in British Columbia, Canada. *DIOXIN '95*, Edmonton, AB, Aug 20-24, 1995.

Mineau, P., J.E. **Elliott**, C. Brossard, N. Thomas, M.R. Fletcher, S.L. Porter, L.A. Lyon and T. Bollinger. Birds of prey and cholinesterase-inhibiting pesticides - a priority for research. SETAC Europe, Copenhagen, Denmark, Aug. 1995.

Elliott, J.E. R.J. Norstrom, A. Lorenzen, L.E. Hart, H. Phillibert, S.W. Kennedy, J.J. Stegeman, G.D. Bellward and K.M Cheng. Biological effects of polychlorinated dibenzo-*p*-dioxin, dibenzofurans and biphenyls in bald eagle chicks. *8th International Symposium on Response of Marine Organisms to Pollutants*. Asilomar, CA, Apr. 2-5, 1995.

Whitehead, P.E., J.E. **Elliott**, J. Van Oostdam and R.J. Norstrom. PCDDs and PCDFs in eagle and osprey eggs near kraft pulp mills in British Columbia, Canada. *DIOXIN '95*, Edmonton, AB, Aug. 20-24, 1995. (invited).

Elliott, J.E., P.E. Whitehead and R.J. Norstrom. Persistent pulp mill pollutants in wildlife. *2nd International Conference on Environmental Fate and Effects of Bleached Pulp Mill Effluents*. Vancouver, BC, Nov. 6-9, 1994. (invited plenary lecture).

Elliott, J.E. R.J. Norstrom, A. Lorenzen, L.E. Hart, H. Phillibert, S.W. Kennedy, J.J. Stegeman, G.D. Bellward and K.M Cheng. Biological effects of polychlorinated dibenzo-*p*-dioxin, dibenzofurans and biphenyls in bald eagle (*Haliaeetus leucocephalus*) chicks. *2nd International Conference on Environmental Fate and Effects of Bleached Pulp Mill Effluents*. Vancouver, BC, Nov. 6-9, 1994.

Elliott, J.E. R.J. Norstrom, A. Lorenzen, L.E. Hart, H. Phillibert, S.W. Kennedy, J.J. Stegeman, G.D. Bellward and K.M Cheng. Biological effects of polychlorinated dibenzo-*p*-dioxin, dibenzofurans and biphenyls in bald eagle (*Haliaeetus leucocephalus*) chicks. *SETAC '94*, Denver, CO, Oct. 29 - Nov. 2, 1994.

Elliott, J.E., L.K Wilson, K.E. Langelier and R.J. Norstrom. Chlorinated hydrocarbon residues and autopsy data from British Columbia bald eagles (*Haliaeetus leucocephalus*), 1986-1993, *SETAC '94*, Denver, CO, Oct. 29 - Nov. 2, 1994.

Wilson, L.K., J.E. **Elliott**, K.E. Langelier, A.E. Scheuhammer and V. Bowes. Lead poisoning in swans from British Columbia. *SETAC '94.* Denver, CO, Oct. 29 - Nov. 2, 1994.

Moul, I.E. and J.E. **Elliott**. Wildlife habitat and bird communities on golf courses in the Lower Fraser Valley, British Columbia. *Urban Landscapes for People and Wildlife*, Seattle. WA, Sept., 1994.

Elliott, J.E. Use of fish-eating birds for monitoring marine and aquatic ecosystems. *1st International Symposium on Ecosystem Health and Medicine*, Ottawa, ON, June 19-23, 1994.

Elliott, J.E. Seabirds as indicators of persistent contaminants in the marine environment. *Marine Ecosystem Health Workshop*, Sidney, BC, April 3-5, 1994.

Elliott, J.E., P.A. Martin, T.W. Arnold, P.H. Sinclair & L. Wilson. Organochlorines and reproductive success of birds in orchard and non-orchard areas of central British Columbia, 1990-91. *SETAC '93*, Houston TX, Nov. 15-18, 1993.

Elliott, J.E., P.E. Whitehead, R.J. Norstrom, M.M. Machmer, J.C. Van Oostdam. Chlorinated hydrocarbons in ospreys breeding near pulp mills in British Columbia. *SETAC '93*, Houston, TX, Nov. 15-18, 1993.

Mineau, P., J.E. **Elliott** & L.K. Wilson. The hazard of granular insecticides in soils prone to puddling or seasonal flooding - the British Columbia experience. *SETAC '93*, Houston, TX, Nov. 15-18, 1993.

Whitehead, P.E., J.E. **Elliott**, R.J. Norstrom, M.M. Machmer, J.C. Van Oostdam & G.E.J. Smith. Chlorinated hydrocarbon levels in eggs of osprey nesting near pulp mills in British Columbia, Canada. *DIOXIN '93*, Vienna, Austria.

Elliott, J.E. A Wildlife Kill Network for British Columbia. *Workshop on Wildlife Mortality Reporting*. Burlington Ontario, Jan, 20-22, 1993. (invited).

Elliott, J.E., J.T. Sanderson, R.J. Norstrom, P.E. Whitehead, L.E. Hart, K.M. Cheng and G.D. Bellward. Monitoring biological effects of dioxin exposure in great blue heron embryos. *SETAC '92*, Cincinnati, OH, Nov. 8-12, 1992.

Elliott, J.E. and P.A. Martin. Chlorinated hydrocarbons and shell thinning in eggs of *Accipiter* hawks in Ontario, 1986-1989. *SETAC '92*, 1992, Cincinnati, OH, Nov. 8-12, 1992.

Whitehead, P.E., R.J. Norstrom & J.E. **Elliott**. Dioxin levels in eggs of great blue herons (*Ardea herodias*) decline rapidly in response to process changes in a nearby kraft pulp mill. *DIOXIN '92*, Tampere, Finland, Aug. 24-28, 1992.

Elliott, J.E., K.M. Langelier, A.M. Scheuhammer, P.H. Sinclair and P.E. Whitehead. Lead poisoning of bald eagles from British Columbia. *SETAC '91*, Seattle, WA, Nov. 3-7, 1991.

Moul, I.E. and J.E. **Elliott**. Turf care chemical use and hazard to birds in British Columbia. *SETAC '91*, Seattle, WA, Nov. 3-7, 1991.

Norstrom, R.J., M. Simon, C. Macdonald, P.E. Whitehead, J.E. **Elliott**, D.C.G. Muir, C. Ford and K.M. Langelier. Food-chain transfer and sources of PCDDs, PCDFs and PCBs in the Strait of Georgia Marine Ecosystem. *SETAC '91*, Seattle, WA, Nov. 3-7, 1991. (invited)

Whitehead, P.E., A. Breault and J.E. **Elliott**. Levels and effects of chlorinated hydrocarbons in fish-eating birds in the Strait of Georgia. *SETAC '91*, Seattle, WA, Nov. 3-7, 1991. (invited)

Elliott, J.E., P.E. Whitehead, R.J. Norstrom, S.M. Bandiera, L.E. Hart, K.M. Cheng and G.D. Bellward. Monitoring chlorinated hydrocarbon exposure in Great Blue Herons using mixed function oxidases. *Sixth International Symposium on responses of marine organisms to pollutants*. Woods Hole, Ma., USA, Apr. 24-26, 1991.

Elliott, J.E. Pesticides and metal residues in marine birds. *Symposium on Ecology and Status of Marine and Shoreline Birds on the West Coast of Vancouver Island*. Sidney, B.C., April 8, 1991. (invited).

Elliott, J.E. Dioxins in wildlife in the Strait of Georgia. *Western Canada Wildlife Health Workshop*. Victoria, B.C. Feb. 15-16, 1991. (invited).

Elliott, J.E., A.M. Scheuhammer, F.A. Leighton and P.A. Pearce. Heavy metal and metallothionein levels in Atlantic Canadian seabirds. *SETAC '90*. Washington, D.C., Nov. 11-15, 1990.

Elliott, J.E., R.J. Norstrom, P.E. Whitehead and P.A. Pearce. Monitoring organic contaminants in Canadian seabirds, 1968-1990. *17th Annual Aquatic Toxicity Workshop*, Vancouver, B.C., Nov. 5-7, 1990. (invited).

Elliott, J.E. and P.E. Whitehead. PCDDs and PCDFs in marine birds from British Columbia: evidence of health effects? *Dioxin Workshop*, Vancouver, B.C., Oct. 17-18, 1990. (invited).

Whitehead, P.E., J.E. **Elliott**, R.J. Norstrom and K. Vermeer. PCDD and PCDF contamination of waterfowl in the Strait of Georgia, British Columbia, Canada, 1989-90. *DIOXIN '90*. Bayreuth, Germany, Sept. 20-25, 1990.

Elliott, J.E., R.J. Norstrom, D.B. Peakall and P.A. Pearce. Patterns and trends of organic contaminants in Canadian seabirds, 1968-1988. *SETAC '89*. Toronto, ON, Oct. 28-Nov. 2, 1989. (invited).

Shutt, L. and J.E. **Elliott**. Monitoring organochlorines in blood of sharp-shinned hawks migrating through the Great Lakes. *SETAC '89*. Toronto, ON, Oct. 28-Nov. 2, 1989,

Peakall, D.B., J.E. **Elliott**, A.J. Gaston and D.N. Nettleship. Organochlorines in Arctic seabirds: changes over the last decade. *SETAC '89*. Toronto, ON, Oct. 28-Nov. 2, 1989. (invited)

Elliott, J.E., S.W. Kennedy. A comparative study of PCB toxicity in Japanese quail and American kestrels: induction of mixed function oxidases and porphyria. *SETAC '89*. Toronto, ON, Oct. 28-Nov. 2, 1989.

Bishop, C.A., J.E. **Elliott**, R.J. Norstrom, R.J. Brooks, J.H. Carey, D. Tillitt. Relationship among organochlorine levels, AHH-induction, hatching success and deformities in Ontario Snapping Turtles. *SETAC '89*. Toronto, ON, Oct. 28-Nov. 2, 1989.

Elliott, J.E., R.J. Norstrom, D.B. Peakall, P.A. Pearce. Marine environmental monitoring and specimen banking. *Canadian Specimen Banking Seminar*, Ottawa, ON, Sept. 25-28, 1989. (invited).

Elliott, J.E., R.J. Norstrom, P.E. Whitehead and R.W. Butler. PCDDs, PCDFs and reproductive success of fish-eating birds from British Columbia. *DIOXIN '89*, Toronto, ON, Sept. 17-22, 1989.

Bellward, G.E., R.J. Norstrom, P.E. Whitehead, J.E. **Elliott**, S.M. Bandiera, C. Dworschak, T. Chang, S. Forbes, B. Cadario, L.E. Hart and K.M. Cheng. Comparison of polychlorinated dibenzodioxin (PCDD) and Dibenzofuran (PCDF) levels with hepatic mixed-function oxidase induction in Great Blue Herons. *DIOXIN '89*, Toronto, ON, Sept. 17-22, 1989.

Elliott, J.E., R.J. Norstrom, P.E. Whitehead and R.W. Butler. Environmental contaminants and reproductive success of Great Blue Herons (*Ardea herodias*) in British Columbia. *SETAC '88*. Arlington, Va, Oct. 13-17, 1988.

Elliott, J.E. Environmental monitoring and specimen banking at the Canadian Wildlife Service - some recent activities. *11th U.S.-German seminar of state and planning on environmental specimen banking*, Bayreuth, FRG, May 1-3, 1988. (invited).

Elliott, J.E., R.J. Norstrom, and J.A. Keith. Environmental contaminants, eggshell thinning and productivity of northern gannets from eastern Canada, 1968-1988. *SETAC '87*. Pensacola, FL, Nov. 2-9, 1987.

Elliott, J.E. and R.J. Norstrom. Trends and effects of environmental contaminants as determined from analysis of Canadian wildlife samples. *9th U.S. - German Seminar on Environmental Specimen Banking*, Gloucester, VA, Oct. 6-8, 1986 (Invited)

Gilbertson, M., J.E. **Elliott** and D.B. Peakall. Seabirds as indicators of marine pollution. *International Conference of Bird Conservation*, Kingston, ON, June 10-16, 1986.

Struger, J., J.E. **Elliott**, M.E. Obbard and D.V. Weseloh. Organochlorine contaminants in snapping turtle eggs from Ontario. *29th Conference on Great Lakes Research*, Toronto, ON, May 26-29, 1986.

Elliott, J.E., D.B. Peakall and P.A. Pearce. Long term organochlorine trends in seabirds from the western north Atlantic. *Pacific Seabird Group* annual meeting, San Francisco, CA, Dec. 4-8, 1985.

Elliott, J.E. and R.J. Norstrom. Specimen banking and monitoring of contaminants in Great Lakes wildlife. *28th Conference on Great Lakes Research*, Milwaukee, WI, June 3-5, 1985.

Struger, J., J.E. **Elliott**, D.V. Weseloh. Heavy metals and essential elements in Herring Gulls from the Great Lakes, 1983. *28th Conference on Great Lakes Research*, Milwaukee, WI, June 3-5, 1985.

Elliott, J.E. Canadian Wildlife Service National Specimen Bank - current activities. Plenary lecture at the *International review of environmental specimen banking*, Washington, DC, Sept. 21, 1983. (invited).

Elliott, J.E. Collecting and archiving wildlife specimens in Canada. *Symposium on environmental specimen banking monitoring as related to banking*, Saarbruecken, FRG, May 10-15, 1982. (invited plenary lecture).

VITAE

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PROFESSIONAL EXPERIENCE:

2001-present. Senior Scientist for the California Condor and Aplomado Falcon programs of the Peregrine Fund, Boise, Idaho. Continued involvement in conservation research on the golden eagle population of the Diablo Mountains in California.

1993-2001. Principal investigator, population ecology of golden eagles in the Diablo Range of California, with emphasis on determining population effects of mortality associated with wind power generation.

1992-1993. Principal investigator, ecology of peregrine falcons on the Channel Islands in relation to DDE contamination.

1991-present. Biologist, Predatory Bird Research Group, University of California.

1986-1992. Principal investigator, ecology of bald eagles in Arizona.

1989-1993. Member of Peregrine Falcon Recovery Team, Western United States for the U.S. Fish and Wildlife Service

1988-89. Member of Board of Directors, Raptor Research Foundation

1983-1986. Principal investigator, ecology of bald eagles on the Pit River, Shasta County, California

1982-1984. Comparison of pesticide levels in Latin American migrant songbirds with those of non-migrants in the southwestern U.S.

1979-1983. Field research on the spring passage of arctic peregrine falcons at Padre Island, Texas, a migration and habitat study using radio telemetry.

1982. (spring). Nesting survey of peregrine falcons in western Texas.

1981. Analysis of migrations of North American peregrine falcons.

1979-1992. Ecologist for BioSystems Analysis, Inc.

1979-1981. Field research on the ecology of wintering bald eagles in northwestern Washington: an analysis of carrying capacity using radio-telemetry.

1979. Author of initial draft of "A Manual of Raptor Research Techniques" for the National Wildlife Federation.

1974-1978. Founding Director and Research Director of the Chihuahuan Desert Research Institute, Alpine, Texas.

1974-1978. Supervised the following field studies: status and ecology of golden eagles in western Texas; time-lapse photographic studies of nesting golden eagles; nesting behavior and ecology of zone-tailed hawks in western Texas; ... of Harris' hawks in western Texas; ...of aplomado falcons in Mexico, ...of bat falcons in southern Mexico; ... of peregrine falcons throughout the Chihuahuan Desert; pesticide contamination of red-shouldered hawks in southern Texas, raptor and prairie dog survey in western Wyoming. Co-supervised: study of insect fauna of locoweed and threadleaf groundsel in western Texas; radio-tracking coyotes in the Big Bend National Park.

1976. Taught Genetics at Sul Ross State University, Alpine, Texas.

1973-1984. Member of Board of Directors of Chihuahuan Desert Research Institute.

1973-1982. Long-term study of the ecology of peregrine falcons in the Chihuahuan Desert and surrounding mountain ranges. Forty active eyries located (only four were known at beginning of survey); also, data on pesticide contamination in wildlife and sediments in Texas, New Mexico, and Mexico.

1971. Postdoctoral Research Associate: Studies of genetic variation in natural populations of land snails with Dr. Robert K. Selander, University of Texas, Austin.

1970. Taught Population Biology at University of Texas, Austin.

1968-1971. Investigation of protein variation and evolution in hybridizing subspecies of the house mouse in Denmark. University of Texas doctoral studies.

1969. Assistant Instructor in General Zoology, University of Texas, Austin.

1967. Laboratory technician (Biochemical Genetics) to Dr. Robert P. Wagner, University of Texas, Austin.

1966. Field Research Assistant (Ornithology) to Dr. Jon Barlow, University of Toronto, Canada.

1965. Attended international peregrine conference at the University of Wisconsin, Madison.

1957-1985. Independent investigation of migratory and foraging behavior of arctic peregrines on the Texas coast.

INTELLECTUAL AND TEACHING INTERESTS:

Population biology, evolution, ecology, genetics, behavior, wildlife conservation, general biology

SKILLS:

Field research, radio-telemetry, data analysis, population genetics, population dynamics modeling, endangered species, time-lapse photography, small mammal trapping, captive breeding of raptors, proposal and report writing, bird banding, GIS, electrophoresis, herpetology, travel and research in Mexico. Commercial pilot, instrument rated.

ACADEMIC BACKGROUND:

University of Texas, Austin, Texas.
Ph.D. (Zoology), December 1970
Dissertation Title: Biochemical variation in hybridizing subspecies of the house mouse (*Mus musculus*) in Europe.
Dissertation advisor: Dr. Robert K. Selander

Sul Ross State University, Alpine, Texas
B.S. (Biology), August 1964
M.A.(Biology), August 1966
Thesis Title: Observations of peregrine falcons on the Texas coast.

FELLOWSHIPS AND SCHOLARSHIPS:

National Institutes of Health Postdoctoral Traineeship in Behavior-Genetics and Evolution, 1971-1972.
National Institutes of Health Predoctoral Traineeship in Genetics, 1968-1970.
National Science Foundation Fellowship, 1968

GRANTS AND CONTRACTS (from):

U.S. Fish and Wildlife Service
World Wildlife Fund
U.S. Boundaries and Water Commission
National Park Service
National Wildlife Federation
Bureau of Land Management
Texas Parks and Wildlife Department
National Renewable Energy Laboratory
California Energy Commission
U.S. Forest Service
U.S. Department of Defense
National Audubon Society
National Geographic Society
U.S. Bureau of Reclamation
Seattle City Light Department
Pacific Gas and Electric Company
Kenetech Windpower, Inc.
The Hopi Tribe

PUBLICATIONS AND REPORTS:

Hunt, W.G. 1966. Observations of peregrines on the Texas coast. Master's Thesis, Sul Ross State University.

Selander, R.K, S.Y. Yang, and W.G. Hunt. 1969. Polymorphism in esterases and hemoglobin in wild populations of the house mouse (Mus musculus). Studies in Genetics 5 (University of Texas Publication 6918):271-338.

Selander, R.K., W.G. Hunt, and S.Y. Yang. 1969. Protein polymorphism and genetic heterozygosity in two European subspecies of the house mouse. Evolution 23:379-380.

Selander, R.K., S.Y. Yang, W.G. Hunt, L. Wheeler, and F.M. Johnson. 1969. Protein polymorphism in wild populations of the house mouse (Mus musculus). Isozyme Bulletin 2:24-25.

Hunt, W.G. and R.K. Selander. 1973. Biochemical genetics of hybridization in European house mice. Heredity 31(1):11-33.

Hunt, W.G. and R.R. Rogers. 1973. Texas peregrine falcons. Bulletin Texas Ornithological Society 6(2):14-16.

Hunt, W. G. 1974. Speculations on the natural regulation of population size in peregrines. Raptor Research Symposium, Nov. 1974.

Rogers, R.R. and W.G. Hunt. 1974. Padre Island, recent years. In J.R. Murphy, et al. (eds), Population status of raptors. Proceedings, Conference Raptor Conservation Techniques 1973. Raptor Research Foundation, University of South Dakota.

Hunt, W.G., R.R. Rogers, and D.J. Slowe. 1975. Migratory and foraging behavior of peregrines on the Texas coast. Canadian Field Naturalist. 89(2):111-123.

Hunt, W.G. 1977. The significance of wilderness ecosystems in western Texas and adjacent regions in the ecology of the peregrine. In: R.H. Wauer and D.H. Riskind (eds.), Transactions of the Symposium on the Biological Resources of the Chihuahuan Desert: United States and Mexico. National Park Service Transactions and Proceedings Series. No. 3.

Hunt, W.G. 1975. The Chihuahuan Desert peregrine falcon survey, 1975. Chihuahuan Desert Research Institute Contribution No. 9.

Hunt, W.G. 1976. The peregrine falcon population in the Chihuahuan Desert and surrounding mountain ranges: an evaluation through 1976. Chihuahuan Desert Research Institute Contribution no. 6.

Hunt, W.G. and B.S. Johnson (eds.). 1977. Nesting peregrine falcons in Texas, 1977. Chihuahuan Desert Research Institute Contribution no. 37.

Hunt, W.G., B.S. Johnson, and D..F. Whitacre. 1976. An opinion concerning the impact upon peregrine falcons of the proposed channelization of the Rio Grande in Presidio and Hudspeth Coun-

ties, Texas. Prepared for the U.S. Fish and Wildlife Service. Chihuahuan Desert Research Institute Contribution No. 11.

Hunt, W.G. 1976. Nesting peregrines in western Texas and northeastern Mexico. In R.W. Fyfe, S.A. Temple, and T.J. Cade, The 1975 North American peregrine falcon survey. Canadian Field Naturalist 90(3):228-273.

Hunt, W.G. and B.S. Johnson. 1976. Continuing studies of raptors in two national parks in western Texas, 1976. Chihuahuan Desert Research Institute Contribution No. 12.

Johnson, B.S., W.G. Hunt, and V.R. Wade. 1977. A study of nesting peregrine falcons between Presidio and Ft. Quitman, Texas: Data summary. Chihuahuan Desert Research Institute Contribution no. 8.

Porter, R. D., G. Craig, D. Ellis, J. Enderson, and W. Hunt. 1978. Status of the Peregrine Falcon in the Rocky Mountains and the southwestern United States, Baja, California and Mexico (south of Texas). Pp. 26-32 in Schaeffer, P. O., and S. M. Ehlers (eds.), The current status of Peregrine Falcon populations in North America. National Audubon Society, Western Education Center, Tiburon, CA.

Hunt, W.G. 1979. West Texas Peregrines. Texas Parks and Wildlife Magazine. February 1979.

Hunt, W.G. and B.S. Johnson. (1979) Migratory North American wildlife in changing Latin American environments. Manuscript twice rejected! Notice the date.

Hunt, W.G., B.S. Johnson, and F.P. Ward. 1979. Spring passage of arctic peregrine falcons at Padre Island: A migration and habitat study using radio telemetry. Chihuahuan Desert Research Institute Contribution No. 50.

Hunt, W.G., B.S. Johnson, and J.B. Bulger, and C. G. Thelander. 1980. Impacts of a proposed Copper Creek dam on bald eagles. BioSystems Analysis, Inc. environmental report for Seattle City Light, City of Seattle, Washington.

Hunt, W.G., F.P. Ward, B.S. Johnson, C.M. Anderson, and G.P. Vose. 1980. A study of the spring passage of peregrine falcons at Padre Island, Texas, using radio-telemetry. Report prepared for the U.S. Fish and Wildlife Service and the National Park Service.

Hunt, W.G., and B.S. Johnson. 1981. Impacts of a proposed Copper Creek Dam on bald eagles: second winter study. BioSystems Analysis, Inc. environmental report for Seattle City Light, City of Seattle, Washington.

Hunt, W.G., F.P. Ward, and B.S. Johnson. 1981. Peregrine falcon migrations: Continuing studies. Report to Department of Defense, Aberdeen Proving Ground, MD. Contract No. DAADO-81-C-0095. 43 pp.

Hunt, W.G. and B.S. Johnson. 1982. Nesting peregrine falcons and pesticides in western Texas, 1982. Report to the National Park Service and U.S. Fish and Wildlife Service.

Johnson, B.S. and W.G. Hunt. 1982. Agua Dulce peregrine falcon eyrie, Lincoln National Forest, 1982 survey. Report to the U.S. Forest Service.

Hunt, W.G. 1983. Aplomado falcon: Texas' lost legacy. Texas Parks and Wildlife Magazine. July, 1983.

Lanning, D.V., P.W. Lawson, and W.G. Hunt. 1985. Ecology of the peregrine falcon in northeastern Mexico. In Winfield Swanson (ed), National Geographic Society Research Reports 1977. 18:377-388.

Hunt. W.G., B.S. Johnson, C.G. Thelander, B.J. Walton, R.W. Risebrough, W.M. Jarman, A.M. Springer, W. Walker II, and J.G. Monk. 1986. Environmental levels of p,p'-DDE indicate multiple sources. Environmental Toxicology and Chemistry 5:21-27.

Risebrough, R.W., W.M. Jarman, A.M. Springer, W. Walker II, and W.G. Hunt. 1986. A metabolic derivation of DDE from Kelthane. Environmental Toxicology and Chemistry 5:13-19.

Hunt, W.G. 1987. Radio telemetry in the study of raptor habitat selection. Raptor Research 21(4):144-146.

Hunt, W.G. and F.P. Ward. 1988. Habitat selection by spring migrant peregrines at Padre Island, Texas. In: T.J. Cade, J.H. Enderson, C.G. Thelander, and C.M. White, (eds.), Peregrine Falcon Populations: Their Management and Recovery. The Peregrine Fund, Inc., 949 pp.

Hunt, W.G., J.H. Enderson, D.V. Lanning, M.A. Hitchcock, and B.S. Johnson. 1988. Nesting peregrines in Texas and northern Mexico. In: T.J. Cade, J.H. Enderson, C.G. Thelander, and C.M. White, (eds.), Peregrine Falcon Populations: Their Management and Recovery. The Peregrine Fund, Inc., 949 pp.

Skaggs, R.W., D.H. Ellis, W.G. Hunt, and T.H. Johnson. 1988. Peregrine Falcon. Pages 127-36 in R.L. Glinski, et al., eds. Proceedings of the Southwest Raptor Management Symposium and Workshop. National Wildlife Federation, Washington, D.C.

Hunt, W.G. 1988. The natural regulation of peregrine populations. In: T.J. Cade, J.H. Enderson, C.G. Thelander, and C.M. White, (eds.), Peregrine Falcon Populations: Their Management and Recovery. The Peregrine Fund, Inc., 949 pp.

Hunt, W.G., J. M. Jenkins, R.E. Jackman, C.G. Thelander, and A.G. Gerstell. 1992. Ecology of bald eagles on a regulated river. Raptor Research 26(4):243-256.

Hunt, W.G., R.E. Jackman, J. M. Jenkins, C.G. Thelander, and R.N. Lehman. 1992. A northward post-fledging migration of California Bald Eagles. Raptor Research 26(1): 19-23.

Hunt, W.G., B.S. Johnson, and R.E. Jackman. 1992. Carrying capacity for bald eagles wintering along a northwestern river. Raptor Research 26(2): 49-60.

Hunt, W.G., D.E. Driscoll, E.W. Bianchi, and R.E. Jackman. 1992. Ecology of bald eagles in Arizona. Report to the U.S. Bureau of Reclamation by BioSystems Analysis, Inc., Santa Cruz, California. Contract No. 6-CS-30-04470.

Jackman, R. E., W. G. Hunt, D. E. Driscoll, and J. M. Jenkins. 1993. A modified floating fish snare for capture of inland Bald Eagles. North American Bird Bander 18: 98-101.

Jackman, R.E., W.G. Hunt, D.E. Driscoll and F. Lapsansky. 1994. Refinements to selective capture techniques for bald and golden eagles: a radio-controlled bow net and power snare for bald and golden eagles. Raptor Research 28:268-273.

Jenkins, J.M., R.M. Jurek, D.K. Garcelon, R. Mesta, W.G. Hunt, R.E. Jackman, D.E. Driscoll, and R.W. Risebrough. 1994. DDE contamination and population parameters of bald eagles *Haliaeetus leucocephalus* in California and Arizona, USA. Pages 751-756 *in* B.-U. Meyburg and R.D. Chancellor, eds. Raptor Conservation Today. Proceedings, 4th World Conference, World Working Group on Birds of Prey, Berlin, 1992. The Pica Press. 799 pp.

Hunt, W.G. R.E. Jackman, T.L. Brown, J. G. Gilardi, D.E. Driscoll and L. Culp. 1995. A pilot golden eagle population study in the Altamont Pass Wind Resource Area, California. Report to National Renewable Energy Laboratory, Subcontract No. XCG-4-14200 to the Predatory Bird Research Group, University of California, Santa Cruz.

Hunt, W.G. R.E. Jackman, T.L. Brown, D.E. Driscoll and L. Culp. 1996. A Population study of golden eagles in the Altamont Pass Wind Resource Area: Second-year progress report. Report to National Renewable Energy Laboratory, Subcontracts XAT-5-15174-01 and XAT-6-16459-01 to the Predatory Bird Research Group, University of California, Santa Cruz.

Hunt, W.G. R.E. Jackman, T.L. Brown, and L. Culp. 1999. A population study of golden eagles in the Altamont Pass Wind Resource Area: Population trend analysis 1994-1997. Report to National Renewable Energy Laboratory, Subcontracts XAT-5-15174-01, XAT-6-16459-01 to the Predatory Bird Research Group, University of California, Santa Cruz.

Hunt, W. G. 1998. Raptor floaters at Moffat's equilibrium. Oikos 82:191-197.

Hunt, W.G. 1998. Bald eagle (chapter). Pages 50-54 in: R.L. Glinski (ed.), The raptors of Arizona. University of Arizona Press, Tucson. 220 pages.

Driscoll, D.E., R.E. Jackman, W.G. Hunt, G.L. Beatty, J.T. Driscoll, R.L. Glinski, T.A. Gatz, and R.I. Mesta. 1999. Status of nesting bald eagles in Arizona. Journal of Raptor Research 33:218-226.

Jenkins, J.M., R.E. Jackman, and W.G. Hunt. 1999. Survival and movements of immature bald eagles fledged in northern California. Raptor Research 33:81-86.

Jackman, R.E., W.G. Hunt, J.M. Jenkins, and P.J. Detrich. 1999. Prey of nesting bald eagles in northern California. Raptor Research 33:87-96.

Hunt, W.G. and P.R. Law. 2000. Site-dependent regulation of population size: comment. Ecology 81:1162-1165.

Hunt, W.G. 2002. Golden eagles in a perilous landscape; predicting the effects of mitigation for wind turbine blade-strike mortality. University of California, Santa Cruz, Santa Cruz, CA, California Energy Commission, Public Interest Energy Research (PIER) Program, Contract Number 500-97-4033, P500-02-043F.

Philips, J. R., W. G. Hunt, and S. W. Matteson. 2002. Arthropods from a Zone-tailed Hawk nest in Texas. Southwestern Entomologist 27: 293-295.

Hunt, W.G., R.E. Jackman, D.E. Driscoll, and E.W. Bianchi. 2002. Foraging ecology of nesting bald eagles in Arizona. Raptor Research 36:245-255.

White, C.M., N.J. Clum, T.J. Cade, and W.G. Hunt. 2002. Peregrine Falcon (*Falco peregrinus*). In A. Poole and F. Gill (eds), The Birds of North America, No. 660. The Birds of North America, Inc., Philadelphia, PA.

Jenny, P., W. Heinrich, A. Montoya, B. Mutch, C. Sandfort, and W.G. Hunt. 2004. Progress in restoring the aplomado falcon to southern Texas. Wildlife Society Bulletin 32:276-285.

Macias-Duarte, A. , A. B. Montoya, W.G. Hunt, A. Lafon-Terrazas, and R. Tafanelli. 2004. Reproduction, prey, and habitat of the Aplomado Falcon (Falcon femoralis) in desert grasslands of Chihuahua, Mexico. The Auk 121:1081-1093.

Brown, J.L., M.W. Collopy, E.J. Gott, P.W. Juergens, A.B. Montoya, and W.G. Hunt. 2006. Wild-reared aplomado falcons survive and recruit at higher rates than hacked falcons in a common environment. Biological Conservation 131:453-458.

Hunt W.G, W. Burnham, C.N. Parish K.K. Burnham, B. Mutch, and J.L. Oaks. 2006. Bullet fragments in deer remains: implications for lead exposure in avian scavengers. Wildlife Society Bulletin 34:167-170. DOI:10.4080/ilsa.2009.0123.

Parish, C.N., W.R. Heinrich, and W.G. Hunt. 2007. Lead exposure, diagnosis, and treatment in California condors released in Arizona. Pages 97-108 in California Condors in the 21st Century (A. Mee, L. S. Hall and J. Grantham, Eds.). Special Publication of the American Orinithologists' Union and Nuttall Ornithological Club.

Hunt WG, Parish CN, Farry SC, Sieg R, Lord TG. 2007. Movements of introduced California Condors in Arizona in relation to lead exposure. Pages 79-98 in California Condors in the 21st Century (A. Mee, L. S. Hall, and J. Grantham, Eds.). Special Publication of the American Ornithologists' Union and Nuttall Ornithological Club.

Green, R.E., W.G. Hunt, C.N. Parish, and I. Newton. 2008. Effectiveness of action to reduce exposure of free-ranging California Condors in Arizona and Utah to lead from spent ammunition.

PLoS ONE 3(12): e4022. DOI:10.1371/journal.pone.0004022

Stroud, R.K. and W.G. Hunt. 2009. Gunshot wounds: A source of lead in the environment. In R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). 2009. Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0109

Parish, C.N., W.G. Hunt, E. Feltes, R. Sieg, and K. Orr. 2009. Lead exposure among a reintroduced population of California Condors in northern Arizona and southern Utah. In R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0217

Hunt W.G., R.T. Watson, J.L. Oaks, C.N. Parish, K.K. Burnham, R.L. Tucker, J.R. Belthoff, and G. Hart G. 2009. Lead Bullet Fragments in Venison from Rifle-killed Deer: Potential for Human Dietary Exposure. PLoS ONE 4(4): e5330. doi:10.1371/journal.pone.0005330.

Hunt, WG, C.N. Parish, K. Orr, and R.F. Aguilar. 2009. Lead Poisoning and the Reintroduction of the California Condor in Northern Arizona. Journal of Avian Medicine and Surgery 23(2):145-150.

Macías-Duarte, A., A.B. Montoya, C.E. Méndez-González, J.R. Rodríguez-Salazar, W. G. Hunt, and P.G. Krannitz. 2009. Factors influencing habitat use by migratory grassland birds in Chihuahua, Mexico. The Auk 126:896-905.

Hunt, W.G. 2009. Golden Eagles and us: Reflections on a most remarkable raptor. Living Bird 29(2):20-29.

Hunt, W.G. 2009. Falcons and falconers: Reflections on an ancient relationship between man and bird. Journal North American Falconers' Association 47:28-31.

Hunt, W.G., D.E. Driscoll, R.I. Mesta, J.H. Barclay, and R.E. Jackman. 2009. Migration and survival of juvenile bald eagles from Arizona. Journal of Raptor Research 43(2):121-126.

Hunt, W.G. 2009. On being a bird in the arctic. In In R.T. Watson, T.J. Cade, M. Fuller, and W.G. Hunt (Eds.). Gyrfalcons and Ptarmigan in a Changing World. The Peregrine Fund, Boise, Idaho.

Hunt, W.G. 2011. Christi and the Cliff Swallow. Birdwatching. June 2011

Hunt, W.G. 2011. Silent alert. Living Bird 30(2):16-19.

Hunt, W.G. 2012. A Darwinian dance. Living Bird (Winter issue: 28-33).

Hunt, W. G. 2012. Implications of Sublethal Lead Exposure in Avian Scavengers. Raptor Research. 46:389-393.

Hunt, W.G., J. L. Brown, T.J. Cade, J. Coffman, M. Curti, E. Gott, W. Heinrich, J.P. Jenny, P. Juergens, A. Macias-Duarte, A.B. Montoya, B. Mutch, and C. Sandfort. 2013. Restoring aplomado

falcons to the United States. Journal of Raptor Research 47(4):335–351.

PROFESSIONAL BACKGROUND OF NOEL F.R. SNYDER March 2014

DATE of Birth: December 25, 1938

MARITAL STATUS: married to Helen A. Snyder since 1967

EDUCATION

Swarthmore College 1957-62, B.A. in 1962, major in biology.
Curtis Institute of Music 1956-62, B.M. in 1962, major in 'cello.
Cornell University, 1962-66, Ph.D. in 1966, major in evolutionary biology.

TEACHING EXPERIENCE

1965-66 Assistant in Introductory Biology, Cornell University
1966-67 Lecturer, Biology Department, Cornell University
1967-72 Assistant Professor of Biology, University of South Florida (courses in general biology, animal behavior, and conservation)

GOVERNMENT EMPLOYMENT

1972-76 Research Biologist in charge of Puerto Rican Field Station, Endangered Wildlife Research Program, USFWS. Studies of the Puerto Rican Parrot.
1976-77 Research Biologist, Endangered Wildlife Research Program, stationed at Patuxent Wildlife Research Center. Preparation of Puerto Rican Parrot monograph.
1978-80 Research Biologist in charge of Sebring Field Station, Endangered Wildlife Research Program. Studies of the Everglade Kite.
1980-86 Research Biologist in charge of California Field Station, Endangered Wildlife Research Program. Studies of California Condor.
1986-89 Biologist, Arizona Game and Fish Department, reintroduction studies of the Thick-billed Parrot.

WILDLIFE PRESERVATION TRUST INTERNATIONAL EMPLOYMENT

1989-98 Director of Parrot Programs (programs in Mexico, S. Lucia, and Jamaica).

FELLOWSHIPS, GRANTS, AND AWARDS

1962 Honorary Woodrow Wilson Graduate Fellowship
1962-64 Cornell Graduate Fellowship
1964-66 NSF Graduate Fellowship
1964-65 NSF Grant GB2223 for study of alarm reactions of aquatic snails, with Edward C. Raney
1964 Sigma Xi grant for study of alarm reactions of aquatic snails
1968 Research Corporation grant for studies of alarm reactions of aquatic snails
1970 National Geographic Society grant for comparative studies of biology of North American accipiters.
1971 U.S. Department of Interior, USFWS contract for comparative studies of biology of North American accipiters.

1971. National Geographic Society grant for study of pesticide effects on the biology of North American accipiters.
1985 Special Achievemnt Award for Condor studies from USFWS
1989 William Brewster Award of the American Ornithologists' Union for work with Puerto Rican Parrots and California Condors.
1989 Distinguished Achievement Award, Society for Conservation Biology for work with the Puerto Rican Parrot and California Condor.
1992 Conservation Medal of the Zoological Society of San Diego for work with California Condors and Thick-billed Parrots.

PUBLICATIONS

Journal Articles and Chapters in Books:

Kurczewski, F.E., and N.F.R. Snyder. 1964. Observations on the nesting of *Pompilus* (*Ammosphex*) *michiganensis* (Dreisbach)(Hymenoptera; Pompilidae). Proceedings of the Biological Society of Washington 77:215-222.

Kurczewski, F.E., and N.F.R. Snyder. 1968. Evolution of cliff-nesting in digger wasps. Conservationist 23:28-31.

Snyder, N.F.R., and H.A. Snyder. 1969. A comparative study of mollusc predation by Limpkins, Everglade Kites, and Boat-tailed Grackles. Living Bird 8:177-223.

Snyder, N. and H. Snyder. 1970. Alarm response of *Diadema antillarum*. Science 168:276-278.

Snyder, N.F.R., and H.A. Snyder. 1970. Feeding territories in the Everglade Kite. Condor 72:492-493.

Snyder, N.F.R., and H.A. Snyder. 1971. Defenses of the Florida apple snail, *Pomacea paludosa*. Behaviour 40:175-215.

Snyder, N.F.R., and H.A. Snyder. 1971. Pheromone-mediated behavior of *Fasciolaria tulipa*. Animal Behavior 19:257-268.

Snyder, N.F.R., and H.A. Snyder. 1973. Experimental study of feeding rates of nesting Cooper's Hawks. Condor 75:461-463.

Snyder, N.F.R., H.A. Snyder, J.L. Lincer, and R.T. Reynolds. 1973. Organochlorines, heavy metals, and the biology of North American accipiters. BioScience 23:300-305.

Snyder, N.F.R., and H.A. Snyder. 1974. Function of eye coloration in North American accipiters. Condor 76:219-222.

Snyder, N., and H. Snyder. 1974. Can the Cooper's Hawk survive? National Geographic Magazine 145:433-442.

Snyder, H.A., and N.F.R. Snyder. 1974. Increased mortality of Cooper's Hawks accustomed to man. Condor 76:215-216.

Snyder, N.F.R. 1975. Breeding biology of Swallow-tailed Kites in Florida. Living Bird 13:73-97.

Snyder, N.F.R. 1977. Increasing reproductive effort and success by reducing nest-site limitations. Pp 27-33 *in* Endangered Birds, management

techniques for preserving threatened species, S.A. Temple (ed.). U. of Wisconsin Press, Madison.

Snyder, N.F.R. 1977. Puerto Rican Parrots and nest-site scarcity. Pp 47-53 *in* Endangered Birds, management techniques for preserving threatened species, S.A. Temple (ed.). U. of Wisconsin Press, Madison.

Snyder, N.F.R., and J.D. Taapken. 1977. Puerto Rican Parrots and nest predation by Pearly-eyed Thrashers. Pp. 113-120 in Endangered Birds, management techniques for preserving threatened species, S.A. Temple (ed.). U. of Wisconsin Press, Madison.

Snyder, N.F.R., and H.A. Snyder. 1979. Biology of North American accipiters in Arizona and New Mexico. National Geographic Society Research Reports, 1970 projects, pp 487-491.

King, W.B., N.F.R. Snyder, M. Segnestam, and J. Grantham. 1979. Noteworthy ornithological records from Abaco, Bahamas. Am. Birds 33:746-748.

Ogden, J.C., and N.F.R. Snyder. 1981. The view from Ventura (overview of the California Condor Research Program). Point Reyes Bird Observatory Newsletter 53:11-15.

Snyder, N.F.R., W.B. King, and C.B. Kepler. 1982. Biology and conservation of the Bahama Parrot. Living Bird 19:91-114.

Snyder, N.F.R., and H.W. Kale II. 1983. Mollusk predation by Snail Kites in Colombia. Auk 100:93-97.

Snyder, N.F.R. 1983. California Condor reproduction, past and present. Bird Conservation 1:67-86.

Snyder, N.F.R. 1983. Establishment of a viable captive population of California Condors. Pp 333-337 *in* Proceedings, Jean Delacour/IFCB Symposium on Breeding Birds in Captivity, A.C. Risser (ed). Los Angeles.

Snyder, N.F.R., J.C. Ogden, J.D. Bittner, and G.A. Grau. 1984. Larval dermestid beetles feeding on nestling Snail Kites, Wood Storks, and Great Blue Herons. Condor 86:170-174.

Snyder, N.F.R., and J.A. Hamber. 1985. Replacement-clutching and annual nesting of California Condors. Condor 87:374-378.

Snyder, N.F.R., and E.V. Johnson. 1985. Photographic censusing of the 1982-83 California Condor population. Condor 87:1-13.

Snyder, N.F.R., and E.V. Johnson. 1985. Photos key to condor census. Outdoor California 46:21-25.

Snyder, N.F.R., R.R. Ramey, and F.C. Sibley. 1986. Nest-site biology of the California Condor. Condor 88:228-241.

Snyder, N.F.R. 1986. California Condor recovery program. Pp 56-71 *in* Raptor Research Report no. 5, Raptor Conservation in the next 50 years, S.E. Senner, C.M. White, and J.R. Parrish (eds.). Raptor Research Foundation.

Snyder, N.F.R., and M.P. Wallace. 1987. Reintroduction of the Thick-billed Parrot in Arizona. Pp 360-384 *in* Proceedings 1987 Jean Delacour/IFCB Symposium on Breeding Birds in Captivity, Los Angeles.

Beissinger, S.R., and N.F.R. Snyder 1987. Mate desertion in the Snail Kite. Animal Behaviour 35:477-487.

Snyder, N.F.R., E.V. Johnson, and D.A. Clendenen. 1987. Primary molt of

California Condors. Condor 89:468-485.
Snyder, N.F.R. 1988. California Condor. Pp 43-66 in Handbook of North American Birds, vol. 4, R.S. Palmer (ed.). Yale University Press, New Haven.
Snyder, N.F.R., and T. Johnson. 1988. Reintroduction of the Thick-billed Parrot in Arizona. Dodo 25:15-24.
Snyder, N.F.R., and H.A. Snyder. 1989. Biology and Conservation of the California Condor. Current Ornithology 6:175-267.
Snyder, N.F.R., S.R. Beissinger, and R.E. Chandler. 1989. Reproduction and demography of the Florida Everglade (Snail) Kite. Condor 91:300-316.
Snyder, N.F.R., H.A. Snyder, and T.B. Johnson. 1989. Parrots return to the Arizona skies. Birds International 1:41-52.
Snyder, H.A., and N.F.R. Snyder. 1990. The comeback of the California Condor. Birds International 2(2):10-23.
Snyder, N. 1990. Endangered parrots and disease problems. Proceeding of the Annual Conference of the Association of Avian Veterinarians, pp 6-7.
Snyder, N. 1990. Review of A Monograph of Endangered Parrots by Tony Silva. Auk 107:795-797.
Snyder, N.F.R. 1990. Review of Peregrine Falcon Populations, Their Management and Recovery by Tom J. Cade, James H. Enderson, Carl G. Thelander, and Clayton M. White (eds.). Auk 107:803-806.
Derrickson, S.R., and N.F.R. Snyder. 1992. Potentials and limits of captive breeding in parrot conservation. Chapter 6 *in* New World Parrots in Crisis: Solutions from Conservation Biology (S.R. Beissinger and N.F.R. Snyder, eds.). Smithsonian Institution Press, Washington, D.C.
Snyder, N.F.R., F.C. James, and S.R. Beissinger. 1992. Toward a conservation strategy for Neotropical Psittacines. Chapter 11 *in* New World Parrots in Crisis: Solutions from Conservation Biology (S.R. Beissinger and N.F.R. Snyder, eds.). Smithsonian Institution Press, Washington, D.C.
Wiley, J.W., Snyder, N.F.R., and R.S. Gnam. 1992. Reintroduction as a conservation strategy for parrots. Chapter 7 *in* New World Parrots in Crisis: Solutions from Conservation Biology (S.R. Beissinger and N.F.R. Snyder, eds.). Smithsonian Institution, Washington, D.C.
Meretsky, V.J., amd N.F.R. Snyder. 1992. Range use and movements of California Condors. Condor 94:313-335.
Snyder, N.F.R., and H. Snyder. 1992. Biology of North American accipiters in the Chiricahua Mountains. Pp 91-94 *in* Proceedings of the Chiricahua Mountains Research Symposium (A.M. Barton and S.A. Sloane, eds.). Southwest Parks and Monuments Association.
Snyder, N.F.R., and T.B. Johnson. 1992. Reintroduction of the Thick-billed Parrot in the Chiricahua Mountains. Pp 87-90 in Proceedings of the Chiricahua Mountains Research Symposium (A.M. Barton and S.A. Sloane, eds.). Southwest Parks and Monuments Association.
Snyder, N.F.R. 1994. The California Condor program: problems in organization and execution. Pp 183-204 in Endangered Species Recovery: Finding the Lessons, Improving the Process (T.W. Clark, R.P. Reading, and A.L.

Clarke, eds.). Island Press, Washington, D.C.

Wilson, M.H., N.F.R. Snyder, S.R. Derrickson, F.J. Dein, J.W. Wiley, J.M. Wunderle, Jr., C.B. Kepler, A.E. Lugo. D.L. Graham, and W.D. Toone. 1994. Puerto Rican Parrots and potential limitations of the metapopulation approach to species conservation. Conservation Biology 8:114-123.

Miller, B., R.Reading, C. Conway, J.A. Jackson, M. Hutchins, N. Snyder, S. Forrest, J. Frazier, and S. Derrickson. 1994. Improving endangered species recovery programs, avoiding organizational pitfalls, tapping the resources, and adding accountability. Environmental Management 18(5):637-645.

Snyder, N.F.R. 1994. Potentials and limits of captive breeding in endangered species conservation. 1994--Midwest Avian Research Expo: 6:

Snyder, N.F.R. and J.W. Wiley 1994. Puerto Rican Parrot Conservation: a retrospective evaluation by two former leaders of the program (1972-1986). PsittaScene 6(1):6-7.

Snyder, N.F.R., S.E. Koenig, J. Koschmann, H.A. Snyder, and T.B. Johnson. 1994. Thick-billed Parrot releases in Arizona. Condor 96:845-862.

Snyder, N.F.R. 1994. The role of captive breeding in parrot conservation. Pp 173-187 *in* Biologia y conservacion de los psitacidos de Venequela (G. Morales, I. Novo, D. Bigio, A. Luy, and F. Rojas Suarez, eds.). Proceedings of Simposio sobre biologia y conservacion de los psitacidos de Venezuela, Rancho Grande, Octubre 25-28, 1992.

Snyder, N.F.R. 1995. A much improved conservation role for zoos (Commentary on Strategic collection planning: theory and practice by M Hutchins, K. Willis, and R.J. Wiese). Zoo Biology 14:36-38.

Snyder, N.F.R., S. Koenig, and T.B. Johnson. 1995. Ecological relationships of the Thick-billed Parrot with the pine forests of southeastern Arizona. Pp 288-293 *in* Biodiversity and Management of the Madrean Archipelago: the Sky Islands of southwestern United States and northwestern Mexico (L.F. DeBano, P.F. Ffolliott, A. Ortego-Rubio, G.J Gottfried, R.H. Hamre, and C.B. Edminster, tech. coords.). U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station Gen. Tech, Rep. RM-GTR-264.

Snyder, N.F.R., K.A. Hayes, and D.A. Mullon, Jr. 1995. Biodiversity versus mining -- a collision of priorities in Cave Creek Canyon. Pp 438-443 in Biodiversity and management of the Madrean Archipelago: the Sky Islands of southwestern United States and northwestern Mexico (L.F. DeBano, P.F. Ffolliott, A. Ortego-Rubio, G.J. Gottfried, R.H. Hamre, and C.B. Edminster, tech. Coords.). U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station Gen Tech. Rep. RM-GTR-264.

Snyder, N., and E. Enkerlin. 1996. New parrot preserve in Mexico. PsittaScene 8(1):8-9.

Snyder, N. W. Toone, E. Enkerlin, and T. Johnson. 1996. Protecting parrots and pines. Zoonooz 69(7):12-17.

Snyder, N.F.R., S.R. Derrickson, S.R. Beissinger, J.W.Wiley, T.B. Smith, W.D. Toone, and B. Miller. 1996. Limitations of captive breeding in endangered species recovery. Conservation Biology 10:338-348.

Snyder, N.F.R., S.R. Derrickson, S.R. Beissinger, J.W. Wiley, T.B. Smith, W.D. Toone, and B. Miller. 1997. Captive breeding and conservation. Conservation Biology 11:3-5.

Snyder, N.F.R., S.R. Derrickson, S.R. Beissinger, J.W. Wiley, T.B. Smith, W.D. Toone, and B. Miller. 1997. Limitations of captive breeding: reply to Gippoliti and Carpaneto. Conservation Biology 11:808-810.

Snyder, N. 1997. Recovery of the California Condor. Environmental Review 4(5):1-8.

Snyder, N.F.R., and H.A. Snyder. 1998. The Cooper's Hawk. Pp 65-67 *in* Raptors of Arizona (R.L. Glinski, ed.). University of Arizona Press, Tucson.

Snyder, N.F.R., and H.A. Snyder. 1998. The Northern Goshawk. Pp 68-72 *in* Raptors of Arizona (R.L. Glinski, ed.). University of Arizona Press, Tucson.

Snyder, N.F.R., and H.A. Snyder. 1998. The Sharp-shinned Hawk. Pp 58-62 *in* Raptors of Arizona (R.L. Glinski, ed.). University of Arizona Press, Tucson.

Snyder, N.F.R., and A.M. Rea. 1998. The California Condor. Pp 32-36 *in* Raptors of Arizona (R.L. Glinski, ed.). University of Arizona Press, Tucson.

Collins, P.W., N.F.R. Snyder, and S.D. Emslie. 2000. Faunal remains in California Condor nest caves. Condor 102:222-227.

Meretsky, V.J., N.F.R. Snyder, S.R. Beissinger, D.A. Clendenen, and J.W. Wiley. 2000. Demography of the California Condor: implications for restablishment. Conservation Biology 14:957-967.

Meretsky, V.J., N.F.R. Snyder, S.R. Beissinger, D.A. Clendenen, and J.W. Wiley. 2001. Quantity versus quality in California Condor Reintroduction: reply to Beres and Starfield. Conservation Biology 15:1449-1451.

Beissinger, S.R., and N.F.R. Snyder. 2002. Water levels affect nest success of the Snail Kite in Florida: AIC and the omission of relevant candidate models. Condor 104:208-215.

Snyder, N.F.R., and V.J. Meretsky. 2003. California Condors and DDE: a re-evaluation. Ibis 145:136-151.

Snyder, N.F.R. 2007. Limiting factors for wild California Condors, pp 9-33 *in* California Condors in the 21st Century (A. Mee and L. Hall, eds.). The Nuttall Ornithological Club and the American Ornithologists' Union.

Eisner, T., F.C. Schroeder, N. Snyder, J.B. Grant, D.J. Aneshansley, D. Utterback, J. Meinwald, and M. Eisner. 2008. Defensive chemistry of lycid beetles and of mimetic cerambycid beetles that feed on them. Chemoecology 18:109-119.

Garraway, E., H.A. Davis, N. Snyder, and A.J.A. Bailey. 2008. New populations of the Jamaican giant swallowtail, *Papilio* (*Pterourus*) *homerus* (Lepidoptera: Papilionidae). Tropical Lepidoptera Research 18:43-45.

Snyder, N.F.R. 2008. Review of Glimpses of Paradise: the Quest for the Beautiful Parrakeet by Penny Olsen. Auk 125:506-508.

Snyder, N.F.R., G.F. Budney, and E.E. Inigo-Elias. 2010. Evaluation of the Nelson Carolina Parakeet Film. Auk 127:948-951.

Snyder, N.F.R., N. Moore-Craig, A.D. Flesch, R.A. Wagner, and R.A. Rowlett. 2010. Short-tailed Hawks nesting in the Sky Islands of the Southwest. Western Birds 41:202-230.

Snyder, N.F.R., H.A. Raffaele, and J.M. Wunderle, Jr. 2010. Dedication to James Wiley. Journal of Caribbean Ornithology 23:1-3.

Snyder, N.F.R., and J.T. Fry. 2013. Validity of Bartram's Painted Vulture (Aves: Cathartidae). Zootaxa 3613:61-82.

Books and Monographs

Snyder, N.F.R. 1967. An alarm reaction of aquatic gastropods to intraspecific extract. Cornell University Agricultural Experiment Station Memoir #403.

Snyder, N.F.R., and J.W. Wiley. 1976. Sexual size dimorphism in hawks and owls of North America. Ornithological Monographs No. 20.

Snyder, N.F.R., J.W. Wiley, and C.B. Kepler. 1987. The parrots of Luquillo, biology and conservation of the Puerto Rican Parrot. Western Foundation of Vertebrate Zoology, Los Angeles.

Snyder, N.F.R., and H.A. Snyder. 1992. Birds of prey: natural history and conservation of North American raptors. Voyageur Press, Stillwater, MN.

Beissinger, S.R., and N.F.R. Snyder (eds.). 1992. New World parrots in crisis: solutions from conservation biology. Smithsonian Institution Press, Washington, D.C.

Snyder, N.F.R., E.C. Enkerlin-Hoeflich, and M.A. Cruz-Nieto. 1999. Thick-billed Parrot (*Rhynchopsitta pachyrhyncha*). *In* The Birds of North America, No. 406 (A. Poole and F. Gill, eds.). The Birds of North America, Inc., Philadelphia, PA.

Snyder, N., and H. Snyder. 2000. The California Condor: a saga of natural history and conservation. Academic Press, London.

Snyder, N.F.R., and N.J. Schmitt. 2002. California Condor (*Gymnogyps californianus*). *In* The Birds of North America, No. 610 (A. Poole and F. Gill, eds.). The Birds of North America, Inc., Philadelphia, PA.

Snyder, N.F.R., and K. Russell. 2002. Carolina Parakeet (*Conuropsis carolinensis*). *In* The Birds of North America, No. 667 (a. Poole and F. Gill, eds.). The Birds of North America, Inc., Philadelphia, PA.

Snyder, N.F.R. 2004. The Carolina Parakeet. Princeton University Press, Princeton.

Snyder, N.F.R., and H.A. Snyder. 2005. Introduction to the California Condor, California Natural History Guides, No. 81. University of California Press, Berkeley.

Snyder, N. and H. Snyder. 2006. Raptors of North America, natural history and conservation. Voyageur Press, St. Paul, MN.

Snyder, N.F.R. 2007. An alternative hypothesis for the cause of the Ivory-billed

Woodpecker's decline. Monographs of the Western Foundation of Vertebrate Zoology 2.

Snyder, N.F.R., D.E. Brown, and K.B. Clark. 2009. The Travails of Two Woodpeckers. University of New Mexico Press, Albuquerque.

SOCIETY MEMBERSHIPS

Association for Parrot Conservation (former board member)
American Ornithologists' Union (elected member, 1984; fellow, 1989)
Cooper Ornithological Society
Wilson Ornithological Society
Chiricahua Regional Council (board member)

RECOVERY TEAM MEMBERSHIPS

Puerto Rican Parrot (1974-1980)
Puerto Rican Plain Pigeon (1974-1980)
California Condor (1980-1986)

MISCELLANEOUS

1976 Filmed and directed "The Parrots of Luquillo" – 16mm sound film with the U.S. Department of Agriculture.

1979-80 Founding member of I Classici Chamber Museic Concerts (cellist), West Palm Beach, Florida.

1990-93 Led successful effort to protect Cave Creek Canyon, Arizona from mining development, culminating in Cave Creek Protection Act of 1993.

1996-2000 Assisted in creation of two natural reserves in Mexico (El Taray and Tutuaca).

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EDUCATION

B.S. Biology and Chemistry, Murray State University, Murray, KY, May 1988

Ph.D. Environmental Toxicology, Texas Tech University, Lubbock, TX, May 2000

PROFESSIONAL APPOINTMENTS

1/01 – 7/01: Senior Research Associate, Texas Tech University, The Institute of Environmental and Human Health, Department of Environmental Toxicology, Lubbock, TX.

7/01 – 8/02: Research Assistant Professor, Texas Tech University, The Institute of Environmental and Human Health, Department of Environmental Toxicology, Lubbock, TX.

9/02 – 9/08: Assistant Professor, Texas Tech University, Department of Environmental Toxicology, The Institute of Environmental and Human Health, Department of Environmental Toxicology, Lubbock, TX.

11/07 – Present Adjunct Professor, Oklahoma State University, Department of Zoology, Stillwater, OK.

9/08 – Present Associate Professor, Texas Tech University, Department of Environmental Toxicology, The Institute of Environmental and Human Health, Department of Environmental Toxicology, Lubbock, TX.

GRANTS

Current Funded Projects

Title: Wind, Growth Promoters, and Human/Ecological Health
Investigators: **P. Smith (PI)**, G. Cobb, B. Johnson, M. Buser, M. Baker, B. Green
Sponsor: United States Department of Agriculture NFRI
Amount: $597,321
Duration: 4 year

Title: Next-Gen Sequencing Enhancement
Investigators: **P. Smith (PI),** S. Cox, G. Mayer
Sponsor: TTU College of Arts & Sciences
Amount: $150,000
Duration: 2 year

Title: Acquisition of a Field Spectroscopy Environmental Analysis System for Gulf Oil Spill Research
Investigators: D. Liang, B. Nutter, S. Cox, **P. Smith (Co-PI)**
Sponsor: National Science Foundation
Amount: $102,909
Duration: 1 year

Title: Field Assessment of Physiologically Active Compounds and Metals in Manure, Soils, and Effluent in Land Application Areas within the Texas Panhandle
Investigators: **P. Smith (PI),** G. Mayer, D. Klein
Sponsor: Texas Cattle Feeders Association
Amount: $10,000
Duration: 1 year

PUBLICATIONS

Peer-Reviewed Journal Articles

1. Wooten, Kimberly J., **Smith, Philip N.** Simulated canine exposure to phthalates and bisphenol A and potential for estrogenic and androgenic responses. *Chemosphere* (in press).

2. Faust, Derek R., Hooper, Michael J., Cobb, George P., Barnes, Melanie, Shaver, Donna, Ertolacci, Shauna, **Smith, Philip N**., Metals in green sea turtles: Tissue correlations and relationships. (in review).

3. Finch, Bryson E., Blackwell, Brett R., Faust, Derek R., Wooten. Kimberly J., Cox, Stephen B., Maul, Jonathan D., **Smith, Philip N.** 2013. Effects of 17α-trenbolone and melengestrol acetate on *Xenopus laevis* growth, development, and survival. *Environmental Science and Pollution Research*. 20(2)1151-1160.

4. Blackwell, Brett R., Karnjanapiboonwong, Adcharee, Anderson, Todd A., **Smith, Philip N.** 2012. Uptake and biotransformation of 17β-trenbolone by the pinto bean plant (*Phaseolus vulgaris*). *Ecotoxicology and Environmental Safety*. 85:110-114.

5. Faust, Derek R., Knowles, Nathan, McGruder, Elaine, Haukos, David A., Cobb, George P., Maul, Jonathan D., Anderson, Todd A., **Smith, Philip N.** 2012. Inorganic and organic contaminants in sediments from an urban playa and associated toxicity among *Hyalella azteca*. *Toxicological and Environmental Chemistry*. 94(9)1746–1757.

6. Finch, Bryson E., Wooten, Kimberly J., Faust, Derek R., **Smith, Philip N.** 2012. Embryotoxicity of Mixtures of Weathered Crude Oil Collected from the Gulf of Mexico and Corexit 9500 in Mallard Ducks (*Anas platyrhynchos*). *Science of the Total Environment*. 426:155-159.

7. Wooten, Kimberly J., Finch, Bryson E., **Smith, Philip N.** 2012. Embryotoxicity of Corexit 9500 in Mallard Ducks (Anas platyrhincos). *Ecotoxicology*. 21(3):662-666.

8. McMurry, Scott T., Jones, Lindsey E., **Smith, Philip N.**, Cobb, George P., Anderson, Todd A., Lovern, Matt B., Cox, Stephen B., Pan, Xiaoping. 2011. Accumulation and effects of octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX) exposure in the green anole (*Anolis carolinensis*). *Ecotoxicology*. 21(2):304-14.

9. Blackwell, Brett R., Cai, Quingsong, Cobb, George P., **Smith, Philip N.** 2011. Liquid chromatography-tandem mass spectrometry analysis of 17α-trenbolone, 17β-trenbolone and trendione in airborne particulate matter. *Talanta*. 85:1317-1323.

10. Finch, Bryson E., Wooten, Kimberly J., **Smith, Philip N.** 2011. Embryotoxicity of weathered crude oil from the Gulf of Mexico in mallard ducks (*Anas platyrhincos*). *Environmental Toxicology and Chemistry*. 30(8)1885-1891.

11. Chumchall, Mathew M., Rainwater, Thomas R., Osborn, Steven C., Roberts, Aaron P., Abel, Michael T., Cobb, George P., **Smith, Philip N.**, Bailey, Frank C. 2011. Mercury speciation and biomagnification in the food web of Caddo Lake, Texas and Louisiana, USA, a subtropical freshwater ecosystem. *Environmental Toxicology and Chemistry*. 30(5)1153-1162.

12. Finch, Bryson, Wooten, Kimberly, **Smith, Philip N.** 2011. Embryotoxicity of weathered crude oil from the Gulf of Mexico in mallard ducks (*Anas platyrhynchos)*. *Environmental Toxicology and Chemistry.* 30(8):1885–1891.

13. Brausch, Kathryn A., Anderson, Todd A., Smith, **Smith, Philip N.**, Maul, Jonathan D. 2010. Effects of functionalized fullerenes on bifenthrin and tribufos toxicity to Daphnia magna: Survival, reproduction, and growth rate. *Environmental Toxicology and Chemistry*. 29(11)2600-2606.

14. Dinehart, Simon K., Smith, Loren M., McMurry, Scott T., **Smith, Philip N.**, Anderson, Todd A., Haukos, David A. 2010. Acute and chronic toxicity of Roundup Weathermax® and Ignite® 280 Sl to larval *Spea multiplicata* and *S. bombifrons* from the Southern High Plains, USA. *Environmental Pollution*. 158:2610-2617.

15. Brausch, John M., Wages, Mike, Shannahan, Randi, Perry, Gad, Anderson, Todd A., Maul, Jonathan D., Mulhearn, Brian, **Smith, Philip N.** 2010. Surface water mitigates the antimetamorphic effects of perchlorate in New Mexico spadefoot toads (*Spea multiplicata*) and African clawed frogs (*Xenopus laevis*). *Chemosphere*. 78:280-285.

16. Brausch, John M., Blackwell, Brett R., Beall, Blake N., Caudillo, Cynthia, Venkata, Kolli, Godard-Codding, Celine, Cox, Stephen, Cobb, George P., **Smith, Philip N.** 2009. Effects of polycyclic aromatic hydrocarbons in northern bobwhite quail (*Colinus virginianus*). *Journal of Toxicology and Environmental Health*, Part A. 73:540-551.

17. Karnjanapiboonwong, Adcharee, Zhang, Baohong, Freitag, Christy M., Salice, Christopher J., **Smith, Philip N.**, Kendall, Ronald J., Anderson, Todd A. 2009. Reproductive toxicity of nitroaromatics to the cricket, *Acheta domesticus*. *Science of the Total Environment*. 47:5046-5049.

18. Brausch, John M., **Smith, Philip N.** 2009. Development of resistance to cyfluthrin and naphthalene among *Daphnia magna*. *Ecotoxicology*. 18:600-609.

19. Brausch, John M., **Smith, Philip N.** 2009. Mechanism determination for resistance and cross-resistance to agrochemicals in *Thamnocephalus platyurus* (Crustacea: Anostraca). *Aquatic Toxicology*. 92:140-145.

20. Dinehart, Simon, Smith, Loren M., McMurry, Scott T., Anderson, Todd A., **Smith, Philip N.**, Haukos, David A. 2008. Toxicity of a glufosinate- and several glyphosate-based herbicides to juvenile amphibians from the Southern High Plains. *Science of the Total Environment*. 407:1065-1071.

21. Brausch, John M., **Smith, Philip N.** 2008. Pesticide resistance from historical agricultural chemical exposure in *Thamnocephalus platyurus* (Crustacea:Anostraca). *Environmental Pollution*. 157:481-487.

22. Cheng Qiuqiong, Liu, Fujun, **Smith, Philip N.**, Jackson, W. Andrew, McMurry, Scott T., Hooper, Michael J., Smith, Ernest E., Blount, Benjamin C., Valentin-Blasini, Liza, Anderson, Todd A. 2008. Perchlorate distribution, excretion, and depuration in prairie voles and deer mice. *Water, Air, and Soil Pollution.* 192:127–139

23. Liu, Jun, Cox, Stephen B., Beall, Blake, Brunjes, Kristina J., Pan, Xiaoping, Kendall, Ronald J., Anderson, Todd A., McMurry, Scott T., Cobb, George P., **Smith Philip N.** 2008. Effects of HMX exposure upon metabolic rate of Northern bobwhite quail (*Colinus virginianus*) *in ovo*. *Chemosphere*. 71:1945-1949.

24. **Smith, Philip N.**, Cobb, George P., Godard-Codding, Céline, Hoff, Dale, McMurry, Scott T., Rainwater, Thomas R., Reynolds, Kevin D. 2007. Contaminant exposure in terrestrial vertebrates. *Environmental Pollution*. 150:41-64.

25. Brausch, John M., Beall, Blake, **Smith, Philip N.** 2007. Acute and sub-lethal toxicity of three POEA surfactant formulations to *Daphnia magna*. *Bulletin of Environmental Contamination and Toxicology.* 78(6):510-514.

26. Yu, Lu, Coimbatore, Gopal, Cobb, George P., Jackson, William A., McMurry, Scott T., **Smith, Philip N.**, Anderson, Todd A. 2007. Evaluation of passive sampling devices as potential surrogates of metal uptake into soybean. *Journal of Plant Nutrition*. 31:1-17.

27. Yu, Lu, Cobb, George P., Jackson, W. Andrew, McMurry, Scott T., **Smith, Philip N.**, Anderson, Todd A. 2007. Evaluation of passive sampling devices as potential surrogates of perchlorate uptake into soybean. *Water, Air, & Soil Pollution*. 182:107-116.

28. Brausch, John M., Cox, Stephen, **Smith, Philip N.** 2006. Pesticide usage on the Southern High Plains and acute toxicity of four chemicals to the fairy shrimp *Thamnocephalus platyurus*. *Texas Journal of Science*. 58:309-324.

29. Brunjes, Kristina J., Severt, Scott A., Liu, Jun, Pan, Xiaoping, Brausch, John, Cox, Stephen A., Cobb, George P., McMurry, Scott T., Kendall, Ronald J., **Smith, Philip N.** 2006. Effects of octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX) exposure on reproduction and hatchling development in Northern bobwhite quail. *Journal of Toxicology and Environmental Health, Part A*. 70:682-687.

30. Liu, Jun, Severt, Scott A., Pan, Xiaoping, **Smith, Philip N.**, McMurry, Scott T., Cobb George P. 2007. Development of an extraction and cleanup procedure for a liquid chromatographic-mass spectrometric method to analyze octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine in eggs. *Talanta.* 71:627-631.

31. Brausch, John M., **Smith, Philip N.**, 2006. Toxicity of three polyethoxylated tallowamine surfactant formulations to laboratory and field collected fairy shrimp, *Thamnocephalus platyurus*. *Archives of Environmental Contamination and Toxicology.* 52(2): 217-222.

32. Selcer, Kyle W., Nespoli, Lisa M., Rainwater, Thomas R., Finger, Adam G., Ray, David A., Platt, Steven G., **Smith, Philip N.**, Densmore, Llewellyn D., McMurry, Scott T. 2006. Development of an enzyme-linked immunosorbent assay for vitellogenin of Morelet's crocodile (*Crocodylus moreletii*). *Comparative Biochemistry and Physiology, C – Toxicology and Pharmacology.* 143:50-58.

33. Zhang, Boahang, **Smith, Philip N.**, Anderson, Todd A. 2006. Evaluating the bioavailability of explosive metabolites (MNX and TNX) in soils using passive sampling devices. *Journal of Chromatography A*. 1101:38-45.

34. Rainwater, Thomas R., Anderson, Todd A., Platt, Steven G., **Smith, Philip N.** 2006. *Agkistrodon piscivorus leucostoma* (Western Cottonmouth). Diet. *Herpetological Review*. 37(2):228.

35. **Smith, Philip N.**, Severt, Scott A., Jackson, W. Andrew, Anderson, Todd A. 2006. Thyroid function and reproductive success in rodents exposed to perchlorate via food and water. *Environmental Toxicology and Chemistry*. 25(4)1050-1059.

36. Isanhart, John P., McNabb, F.M. Anne, **Smith, Philip N.** 2005. Effects of perchlorate exposure on resting metabolism, peak metabolism, and thyroid function in the prairie vole, *Microtus ochrogaster*. *Environmental Toxicology and Chemistry*. 24(3):678-684.

37. Rainwater, Thomas R., Reynolds, Kevin D., Cañas, Jaclyn C., Cobb, George P., Anderson, Todd A., McMurry, Scott T., **Smith, Philip N.** 2005. Organochlorine pesticides and mercury in cottonmouths (*Agkistrodon piscivorus*) from northeastern Texas. *Environmental Toxicology and Chemistry.* 24(3):665-673.

38. **Smith, Philip N.**, Utley, Sarah J., Cox, Stephen B., Anderson, Todd A., and McMurry, Scott T. 2005. Monitoring perchlorate exposure and thyroid hormone status among raccoons inhabiting a perchlorate-contaminated site. *Environmental Monitoring and Assessment.* 102:337-347.

39. Jackson, W. Andrew, Joseph, Preethi, Laxman, Patil, Tan, Kui, **Smith, Philip N.**, Yu, Lu, Anderson, Todd A. 2004. Perchlorate accumulation in forage and edible vegetation. *Journal of Agricultural and Food Chemistry.* 53:369-373.

40. **Smith, Philip N.**, Yu, Lu, McMurry, Scott T., Anderson, Todd A. 2004. Perchlorate in water, soil, vegetation, and rodents collected from the Las Vegas Wash, Nevada. *Environmental Pollution.* 132:121-127.

41. Cheng, Q., Perlmutter, Lisa, **Smith, Philip N.**, McMurry, Scott T., Jacksonn W. Andrew, Anderson, Todd A. 2004. A study on perchlorate exposure and absorption in beef cattle. *Journal of Agricultural and Food Chemistry*. 52(11):3456-3461.

42. Tan, Kui, Anderson, Todd A., Jones, Matthew W., **Smith, Philip N.**, Jackson, W. Andrew. 2004. Accumulation of perchlorate in aquatic and terrestrial plants at field scale. *Journal of Environmental Quality.* 33:1638-1646.

43. Kendall, Ronald J., **Smith, Philip N.** 2003. Wildlife Toxicology: Reflections on the past and perspectives on the future. *Environmental Science and Technology*. May, 2003 178A-183A.

44. **Smith, Philip N.**, Johnson, Kevin A., Anderson, Todd A., McMurry, Scott T. 2003. Environmental exposure to polychlorinated biphenyls among raccoons (*Procyon lotor*) at the Paducah Gaseous Diffusion Plant, Western Kentucky. *Environmental Toxicology and Chemistry.* 22(2):406-416.

45. **Smith, Philip N.**, Johnson, Kevin A., Bandiera, Stelvio M., McMurry, Scott T. 2003. Measures of mixed-function oxidases in raccoons (*Procyon lotor*) exposed to polychlorinated biphenyls. *Environmental Toxicology and Chemistry.* 22(2):417-423.

46. **Smith, Philip N.**, Lochmiller, Robert L., Qualls, Charles W., and McMurry, Scott T. 2003. Lymphoproliferative responses of splenocytes from wild cotton rats (*Sigmodon hispidus*) following acute exposure to Arochlor 1254. *Bulletin of Environmental Contamination and Toxicology.* 70:97-105.

47. Carr, James A., Urquidi, Linda J., Goleman, Wanda L., Hu, F., **Smith, Philip N.**, and Theodorakis, Chris W. 2003. Ammonium perchlorate disruption of thyroid function in natural amphibian populations: Assessment and potential impact, " *Multiple Stressor Effects in Relation to Declining Amphibian Populations,* ASTM STP 1443, G. Linder, Ed., ASTM International, West Conshohocken, PA.

48. **Smith, Philip N.**, Cobb, George P., Harper, Frances D., Adair, Blakely, and McMurry, Scott T. 2002. Comparison of white-footed mice and rice rats as biomonitors of polychlorinated biphenyl and metal contamination. *Environmental Pollution*. 119(2):261-268.

49. **Smith, Philip N.**, Theodorakis, Christopher W., Anderson, Todd A., and Kendall, Ronald J. 2001. Preliminary assessment of perchlorate in ecological receptors at the Longhorn Army Ammunition Plant (LHAAP), Karnack, Texas. *Ecotoxicology*. 10:305-313.

Books

1. Cobb, George P., **Smith, Philip N**. 2013. Evaluating Veterinary Pharmaceutical Behavior in the Environment. American Chemical Society Symposium Series 1126.

2. Kendall, Ronald J., **Smith, Philip N.**, editors. 2006. *Perchlorate Ecotoxicology.* Pensacola, FL: Society of Environmental Toxicology & Chemistry. 288 p.

3. Kendall, Ronald J., Presley, Steven M., Austin, Galen P., **Smith, Philip N**., editors. 2008. Advances in Biological and Chemical Terrorism Countermeasures. CRC Press, Taylor & Francis Group: Boca Raton, FL. 280 p.

Book Chapters

1. Blackwell, Brett R., Buser, Michael D., Johnson, Bradley J., Baker, Matt, Cobb, George P., **Smith, Philip N.** 2013. Liquid chromatography mass spectrometry: Analysis of veterinary growth promoters in airborne particulate matter. In Cobb, G. and Smith, P. eds. *Evaluating Veterinary Pharmaceutical Behavior in the Environment*, American Chemical Society Symposium Series 1126.

2. **Smith, Philip N.** Perchlorate environmental ecotoxicology. 2006. In: Gu, B., Coates, J. (eds.). *Perchlorate Environmental Occurrence, Interactions and Treatment*. Springer: New York City, NY. pp 153-164.

3. Mayer, Kevin P., Jackson, W. Andrew, Snyder, Shane A., **Smith, Philip N.**, Anderson, Todd A. 2006. State of the Science: Background, History, & Occurrence. In Kendall, Ronald J., Smith, Philip N. eds. *Perchlorate Ecotoxicology.* Pensacola, FL: Society of Environmental Toxicology & Chemistry. pp 1-19.

4. Smith, Ernest E., McNabb, F. M. Anne, Isanhart, John P., **Smith, Philip N.**, Dixon, Kenneth R. 2006. Perchlorate Effects on Mammals. In Kendall, Ronald J., Smith, Philip N. eds. *Perchlorate Ecotoxicology.* Pensacola, FL: Society of Environmental Toxicology & Chemistry. pp 79-95.

5. **Smith, Philip N.**, Wireman Jody, Carr James A., Smith Ernest E., Kendall Ronald J. 2006. Ecological Risk Assessment. In Kendall, Ronald J., Smith, Philip N. eds. *Perchlorate Ecotoxicology.* Pensacola, FL: Society of Environmental Toxicology & Chemistry. pp 229-251.

6. Presley, Steven M., Austin, Galen P., **Smith, Philip N.**, White, John, Kendall Ronald J. 2008. Threats and Vulnerabilities Associated with Biological and Chemical Terrorism. In Kendall, Ronald J., Presley, Steven M., Austin, Galen P., Smith, Philip N. eds., Advances in Biological and Chemical Terrorism Countermeasures. Taylor & Francis: Boca Raton, FL. pp. 13-28.

7. Kendall, Ronald J., Austin, Galen P., Chang, Chia-bo, Cobb, George P., Coimbatore, Gopal, Cox, Stephen B., Fralick, Joe A., Leggoe, Jeremy W., Presley, Steven M., Ramkumar, Seshadri S., **Smith, Philip N.**, Strahlendorf, Jean C., Zartman, Richard E. 2008. Conclusions and Research Needs for the Future. In Kendall, Ronald J., Presley, Steven M., Austin, Galen P., Smith, Philip N. eds., *Advances in Biological and Chemical Terrorism Countermeasures*. Taylor & Francis: Boca Raton, FL. pp. 243-248.

8. **Smith, Philip N.**, Afzal, Mohamad, Al-Hasan, Redha, Bouwman, Castillo, Luisa E., Henk, Depledge, Michael H., Subramanian, Muralidharan, Dhananjayan, Venugopal, Fossi, Cristina, Kitulagodage, Malsha, Kylin, Henrik, Law, Robin, Marsili, Letizia, O'Hara, Spinola, Manuel, Todd, Story, Paul, Godard-Codding, Céline. 2010. Global Perspectives on Wildlife Toxicology: Emerging Issues. In, Kendall, Ronald J., Lacher, Tom, Cobb, George P., Cox, Stephen B. eds., *Wildlife Toxicology: Emerging Contaminant and Biodiversity Issues*. Taylor and Francis: Boca Raton, FL.

9. Kendall, Ronald J., Anderson, Todd A., Cobb, George P., Cox, Stephen B., Hannah, Lee, Lacher Jr., Thomas E., Presley, Steven M., Salice, Christopher J., **Smith, Philip N.**. "Looking Forward: The Global Future of Wildlife Toxicology" in Kendall, Ronald J., Cobb, George P., Cox, Stephen, Lacher, Thomas E., eds. *Wildlife Toxicology: Emerging Contaminant and Biodiversity Issues*, Taylor and Francis, Boca Raton, FL.

PROFESSIONAL TRAINING/CERTIFICATIONS

- HAZWOPER – 40hr Hazardous Waste Operator certified

PROFESSIONAL SOCIETY AND ORGANIZATION MEMBERSHIPS

- Society of Environmental Toxicology and Chemistry (1996 – present)
- The Wildlife Society (1995 – 2005)
    - Wildlife Toxicology Working Group – Director-At-Large, Central Region (2000-2003)
- American Society of Mammalogists (1994 – 1996)

PROFESSIONAL SERVICE

Editorships and Editorial Boards

- Editor, *Environmental Toxicology and Chemistry* (2011 – present)
    - Editorial Board Member (2006 – 2009, 2010 - present)
- Environmental Health section editor, *Earth Systems and Environmental Sciences* (Elsevier; 2013)
- Associate Editor, *Journal of Herpetology* (2010 – 2012)
- Editorial Board Member, *Environmental Pollution* (2004 – present)

Refereed Journal Reviews

- *Canadian Journal of Zoology*
- *Chemosphere*
- *Ecotoxicology*
- *Environmental Health Perspectives*
- *Environmental Pollution*
- *Environmental Research*
- *Environmental Science & Technology*
- *Environmental Toxicology & Chemistry*
- *Journal of Applied Ecology*
- *Journal of Herpetology*
- *Journal of the Institute of Laboratory Animal Research*
- *Journal of Toxicology and Environmental Health Science*
- *Journal of Wildlife Diseases*
- *PLoS ONE*
- *Regulatory Toxicology & Pharmacology*
- *Water, Soil, and Air Pollution*

Invited Reviews

- Reviewer for US EPA Draft Ecological Soil Screening Levels (Eco-SSLs) for Polycyclic Aromatic Hydrocarbons, (2006)
- Reviewer for US Army Center for Health Promotion and Preventative Medicine *Wildlife Toxicity Assessments* (7) through SETAC, (2006)
- External Thesis Reviewer – University of Wollongong, Australia, (2005, 2012)
- EPA STAR Fellowship Scientific Review Panel, (February 2004)
- Reviewer for National Research Foundation of South Africa (2004)
- EPA Technical Report Review (2003)
- EPA STAR Fellowship Scientific Review Panel, February (2002)

UNIVERSITY SERVICE

- Chair, Institutional Animal Care and Use Committee (2011 – present)
    - Vice-Chair (2007 – 2010)
    - Member (2006 – present)
- Faculty Senate Member (2012 – present)
    - Faculty Senate Ethics Advisory Committee Liaison (2012 - present)

College (Arts & Sciences)

- Natural and Physical Sciences Research Council (2010–present)

PUBLIC SERVICE

- Lubbock County Animal Issues Committee (2012-present)
- Region 12 Running Rules Representative, Hunting Retriever Club, Inc. (2012 - present)
- Member City of Wolfforth Comprehensive Plan Steering Committee, (2008 – 2009)
- President (2007-2008), Vice President (2006 - 2007) Frenship Youth Baseball, Inc.
- President, Texas Panhandle Hunting Retriever Club, Inc., (2006 – 2008)
- Judge, Texas State Science Fair, (2004)
- Mentor in the MentorTech Program providing leadership and support to minority undergraduate students, (2004 – 2005)

HONORS

- *Presidential Citation for Exemplary Service* at the 29th Annual Meeting of the Society of Environmental Toxicology and Chemistry in Tampa Florida for outstanding contributions to the Society's peer-review process (2008)
- *Presidential Book Award*, Texas Tech University (2010)

COURSES TAUGHT

- Environmental and Wildlife Toxicology (ENTX 6361), SP13
- Environmental Toxicology Seminar: How to Build a Manuscript and How to Break One Down (ENTX 6115) SP12
- Principles and Techniques of Ecological Risk Assessment (ENTX 6371), FA04 - Present
- Journal Club Seminar "Current Topics in Ecotoxicology (ENTX 6100), SP05
- Journal Club Seminar "Current Topics in Ecotoxicology (TOX 6115), FA03
- Introductory Seminar in Environmental Toxicology (TOX 6105-060), FA00

GRADUATE STUDENT SUPERVISION

Dissertation Committees

Chair (Ph.D.)

Brett Blackwell, Environmental Toxicology, in progress
"Airborne Anabolic Hormones from Confined Animal Feeding Operations"

John Brausch, Environmental Toxicology, completed 2009
"Pesticide Resistance among Invertebrates inhabiting the Playa Lakes of the Southern High Plains"

Member (Ph.D.)

Eric Howell, Biology, in progress
"Effects of Ionizing Radiation and Utility of Science Diplomacy: Exploring Relationships, Applications, and Impacts

Tyson Brown, Animal & Food Sciences, in progress
"Characterization of Steroidal Metabolite Excretion from Implanted Cattle"

Kristyn Urban, Environmental Toxicology, in progress
"Effects of *Flavivirus* on Northern Bobwhite Quail Matrix Metalloproteinase (Mmp)-2, Cardiomyocyte Size, and Potential for a Disruptive Cue to Normal Myocyte Function in Wild Populations"

Simon Dinehart, Natural Resource Management (Oklahoma State University) , completed 2009
"Effects of Herbicides on Amphibians of the Playa Lakes"

John Isanhart, Environmental Toxicology, completed 2008
"Effects of Acid Mine Water on Avian Species"

Jaclyn Cañas, Environmental Toxicology, completed 2005
"The Development and Application of Preconcentration/Preelution Ion Chromatography Methods for the Detection of Trace Perchlorate in Difficult Matrices"

Lu Yu, Environmental Toxicology, completed 2004
"Evaluation of Passive Sampling Devices as Potential Surrogates of Perchlorate or Heavy Metal Uptake in Terrestrial Plants"

Thesis Committees
Chair (M.S.)
Andrew McEachran, Environmental Toxicology, in progress
"Aerial Transport and Fate of Antibiotics and ABR Microbes from Beef Cattle Feedlots"

Kimberly Wooten, Environmental Toxicology, in progress
"Companion Canine Exposure to BPA, Phthalates, and PFOS"

Derek Faust, Environmental Toxicology, M.S. completed 2012
"Environmental Monitoring of the Midland Nature and Educational Center"

Bryson Finch, Environmental Toxicology, M.S. completed 2011
"Effects of Weathered Gulf of Mexico Oil on Avian Embryotoxicity"

Blake Beall, Environmental Toxicology, M.S. completed 2007
"Effects of Polycyclic Aromatic Hydrocarbon Exposure among Northern Bobwhite Quail"

Anna Herboldsheimer, M.S. completed 2003
"Food Safety Assessment of Perchlorate-Contaminated Crops in Prairie Voles (*Microtus ochrogaster*)"

John Isanhart, M.S. completed 2004
"Effects of Perchlorate Exposure on Resting Metabolism, Peak Metabolism, and Thyroid Function in the Prairie Vole (*Microtus ochrogaster*)"

Scott Severt, M.S. completed 2004
"Food Safety Assessment of Perchlorate-Contaminated Crops in Rodents"

Member (M.S.)
Kathryn Brausch, Environmental Toxicology, completed 2010
"Interaction of Chemical Stressors and Predator-Prey Relationships"

Amy Hensley, Environmental Toxicology, completed 2004
"Response of Metamorphosis and Immune Function to Perchlorate Exposure in Spadefoot Toads"

Jun Lu, Environmental Toxicology, completed 2007
"Maternal Transfer and Tissue Distribution of HMX in Quail Eggs"

Lindsey Jones, Environmental Toxicology, completed 2007
"Accumulation and Effects of HMX in the Green Anole (*Anolis carolinensis*)"

Preethi Joseph, Civil Engineering, completed 2004
"The Effect of Nitrogen on the Uptake of Perchlorate in Plants"

Sarah Utley, J.D./M.S., Environmental Law/Environmental Toxicology, completed 2002
"Perchlorate Exposure and Effects in Wildlife"

Scott D. Wright, Ph.D.
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Retired from Federal service December 29, 2011
Currently serving as an Assistant Editor for the *Journal of Wildlife Diseases*

Professional Preparation

St. Petersburg Junior College, USA	Biology	Associates in Arts	1970
University of South Florida,, USA	Zoology	BA	1973
University of Connecticut, USA	Pathobiology (pathology)	PhD	1987
University of Florida, USA	Pathology post doctoral fellow		1987-1989

Appointments

Branch Chief, Disease Investigations Branch, United States Geological Survey, National Wildlife Health Center, Madison Wisconsin 2001-2011

Research Scientist. Florida Marine Research Institute, Marine Mammal Pathobiology Laboratory, Florida Fish and Wildlife Conservation Commission, St. Petersburg, Florida 1989-2001

Director, Marine Mammal Pathobiology Laboratory, Florida Marine Research Institute, St. Petersburg, Florida 1993-2001

Post-Doctoral Fellow, Department of Infectious Diseases, College of Veterinary Medicine, University of Florida. Gainesville, Florida 1987-1989

Graduate Research Assistant, Northeastern Research Center for Wildlife Diseases University of Connecticut, Storrs, Connecticut. 1982-1987

Selected Publications

Bossart, G.D., D.G. Baden, R.Y. Ewing, B. Roberts, and S.D. Wright. 1998. Brevetoxicosis in manatees (*Trichechus manatus latirostris)* from the 1996 epizootic: Gross, histologic, and immunohistochemical features. Toxicologic Pathology 26(2): 276-282.

Pence, D.B. and S.D. Wright. 1998. *Chaelonacarus elongatus* n. gen., n. sp. (Acari; Cloacaridae) from the cloaca of the green turtle (*Chelonia midas*; Cheloniidae). The Journal of Parasitology 84(4): 835-839.

Wright, I.E., S.D. Wright, and J.M. Sweat. 1998. Use of passive integrated transponder (PIT) tags to indentify manatees (*Trichechus manatus latirostris).* Marine Mammal Science 14(3): 641-645.

Ackerman, B.B., S.D. Wright, R. K. Bonde, D.K. Odell, and D.J. Banowetz. 1995. Trends and patterns of mortality of manatees in Florida. 1974-1992. Pages 223-258 *in* T.J. O=Shea, B.B. Ackerman, and H.F. Percival, editors. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1.

Duignan, P.J., C. House, M.T. Walsh, T. Campbell, G.D. Bossart, N. Duffy, P.J. Fernandes, B.K Rima, S. Wright, J.R. Geraci. 1995. Morbillivirus infection in manatees. Marine Mammal Science 11:441-451.

McDowell, L.R., D. J. Forrester, S. B. Linda, S. D. Wright, N. S. Wilkinson. 1995. Selenium status of White-tailed deer in southern Florida. Journal of Wildlife Diseases 31(2):205-211.

Wright, S.D., B.B. Ackerman, R.K. Bonde, C.A. Beck, and D.J. Banowetz. 1995. Analysis of watercraft-related mortality of manatees in Florida, 1979-1991. Pages 259-268 *in* T.J. O'Shea, B.B. Ackerman, and H.F. Percival, editors. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1.

Atkinson, C.T., S.D. Wright, S.R. Telford, Jr., G. S. McLaughlin, D.J. Forrester, M. E. Roelke, and J.W. McCowan. 1993. Morphology, prevalence, and distribution of *Sarcocystis* spp. In white-tailed deer (*Odocoileus virginianus)* from Florida. Journal of Wildlife Diseases 24:73-84.

Bradley, J.L., S. D. Wright, and P.M. McQuire. 1993. The Florida manatee: cytochrome b DNA sequence. Marine Mammal Science 9(2):197-202.

Telford, S.R., Jr., D.J. Forrester, S.D. Wright, M.E. Roelke, S.A. Ferenc, and J.W. McCowan. 1991. The identity and prevalence of trypanosomes in white-tailed deer (Odocoileus virginianus) in southern Florida. Journal of the Helminthological Society of Washington 58:19-23.

Synergistic Activities

Department of the Interior, Security clearance 2010-2015

- President, Wildlife Disease Association, (2005-2007)
- NPS, Yellowstone Wildlife Health Program, program development, (2007)
- Avian Influenza National Response Plan, writer on strategic planning panel (2006)
- USAHA, Steering Committee member, Brucellosis in the GYA (2005)
- National Plan for the Response to Chronic Wasting Disease, co-leader for Diagnostics (2003)
- Implementation Plan for Chronic Wasting Disease, co-author (2004)
- Dept. of the Interior, Technical Lead, Chronic Wasting Disease (2002-2004)
- Dept. of the Interior, briefed Secretary Norton on Chronic Wasting Disease, (2002)
- West Nile Virus, member of Interagency Steering Committee, (2002-2004)
- Dept. of the Interior, Technical Lead on West Nile Virus, (2002-2004)
- Dept. of the Interior, briefed Secretary Norton on Foot and Mouth Disease (2001)
- Reviewer for Journal of Wildlife Diseases (1988-present)
- Assistant Editor for Journal of Wildlife Diseases (2010-present))

Graduate and Postdoctoral Advisors:

Masters Advisor: Mark Sweat, University of South Florida, USA
Masters Advisor: Leslie Ward, University of South Florida, USA

Thesis Advisor and Postgraduate-Scholar Sponsor:

Veterinary Externship Advisor, approximately 12 students (1996-2001), Marine Mammal Pathobiology Laboratory. St. Petersburg, Florida

Research Advisor, Special projects for senior veterinary students, (1987-1989), University of Florida, College of Veterinary Medicine.

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APPENDIX B: INDIVIDUAL REVIEWER COMMENTS

REVIEWER 1

USFWS Review of Lead Exposure and Effects to Scavenging Birds
An External Scientific Peer Review
February 20, 2014

General Comments: The scope of this white paper is considerably narrower than the issue of lead toxicosis as a result of exposure to spent lead ammunition. There are decades of data documenting the negative effects of lead in many species of birds in addition to scavenging birds, as well as reptiles, amphibians, and several species of mammals including humans. However, this more focused review of lead in scavenging birds does describe the issue in detail. In my view, the authors do clearly establish the "cause and effect" of lead exposure in scavenging birds and that the source of lead for these birds is spent ammunition, including fragmented bullets found in carcasses and/or offal left in the environment. Despite the fact that this white paper does not address human health, a little further reading and reasonable logical thinking suggest that lead in the form described in this paper is a threat to human health as well.

The Question of "Scientific Uncertainty"
With regard to the question of lead being harmful and spent ammunition being a source of lead to wild scavenging birds, there is little scientific uncertainty. If this was a recently discovered issue, (such as White-nose Syndrome in bats), where there is a paucity of data and little time to accumulate data because the disease is spreading so quickly, there would be a higher level of scientific uncertainty. Nevertheless, multiple federal and state agencies have taken action to attempt to retard or prevent the spread of White-nose Syndrome throughout North America before scientific certainty has been established.

In 1991, lead shot was banned for hunting waterfowl before there was as much information available about lead toxicosis as there is now. At the time of the implementation of the ban, there was not as much known about the physiological effects of lead, better diagnostic tools for detection of lead were not developed, and the overall population effects of the ban were not known. Yet, as shown by numerous studies subsequent to the ban, it successfully reduced mortality rates of waterfowl. Lead, in the form of spent shot in freshwater sediments, is still available and waterfowl are still exposed but the overall negative affect to waterfowl at the population level has been reduced.

It is reasonable, despite some scientific uncertainty, to suggest that alternatives to the use of lead ammunition be explored in order to reduce further contamination to the environment and wildlife (here specifically wild scavenging birds). The authors of this white paper suggest some areas of uncertainty although these are not specifically indicated as such. Instead, I have extracted them from the Executive Summary; they include:

- lead exposure elicits measureable physiological results that are variable between species of birds and therefore these responses are not the same in all species nor are they known for all species of birds.
- the progression of lead poisoning leads to death; however, the effects of sub-lethal exposure could weaken the individual making it more vulnerable to other threats.

- while general thresholds of tissue concentrations of lead have been established, varying individual and species sensitivities to intoxication from lead suggests employing a combination of tissue concentrations and clinical signs in order to best establish a diagnosis. It is relevant to point out that the majority of birds submitted to diagnostic laboratories for necropsy and a determination of a cause of death were not observed prior to death and so there are no clinical signs (my statement).
- carcasses and offal piles are a food source for birds and other wildlife. However, the actual number of carcasses and offal piles left in the environment each year is unknown although it is estimated to be in the millions.
- Lead pellets or lead fragments from bullets are found in the stomach and intestines of scavenger and predatory birds. However, the absence of the physical presence of lead could result from, lack of ingestion, regurgitation of particles or complete absorption (especially of smaller fragments). Therefore, the uncertainty is that the absence of particles does not always and automatically mean the lack of exposure (my statement).
- a related issue is that spherical shot likely takes longer to degrade in the stomach and intestines of birds than fragmented particles of bullets. This is hypothesized based upon the greater surface area of the bullet fragments compared to pellets.
- although there is ample documentation of lead toxicosis in several species of predatory and scavenging birds throughout the nation, these cases likely under-represent the number of actual deaths each year. This is due, in part, because the larger birds (eagles, condors, vultures, etc.) are easier to see and more likely found and submitted for necropsy than smaller birds.

There are some additional areas of scientific uncertainty that the authors may wish to consider:

- wildlife may sustain long term sub-lethal effects on coordination and cognitive behavior. It is likely that repetitive long term sub-lethal exposure could result in permanent neurological/behavioral damage.
- the mortality rates of birds as a result of lead toxicosis are not clearly known because determination of the mortality rate is dependent upon discovery of carcasses.
- sampling methods of free-ranging wild birds and experiments with captured or wild caught wild birds have inherent biases. Whatever information gathered in these species is considered indicative, in a general sense, of the response in related but not tested species. For example, lead studies performed on captive Kestrels are considered an example of how predatory birds are affected. Likewise, studies of farm-raised mallards are indicative of a waterfowl response.
- long-term sub-lethal exposure to wild populations of birds could have limiting effects on reproduction. Most experimental studies are not long term, focusing instead on acute toxicity. Reduced reproductive vigor, while a reasonable outcome from prolonged exposure to lead, would be difficult to assess in a wild population.

Further Research to Address Scientific Uncertainty

As described above there are questions of uncertainty regarding some effects of lead poisoning in scavenging and other birds. However, these questions do not stand in the way of the pursuit of alternatives to the use of lead ammunition. This is because the answers to the questions of uncertainty do not change the role of lead as a poison to birds and other animals. The answers

would not change the importance of spent lead ammunition including fragmented lead bullets as a source of lead to scavenging birds. Therefore, because of the vast amount of information already available, there is no reasonable justification for further study.

Currently all experimental use of animals is conducted under the regulation and authority of the Animal Welfare Act (AWA). The use of animals applies to all animals including wildlife and including field studies of wild animals. All research institutions are subject to the AWA. Only federal agencies are exempt, however nearly all comply with the Act. Each research institution must appoint an Institutional Animal Care and Use Committee (IACUC) and there are procedures and requirements for the development of a committee and its structure. Each committee is tasked with approving research proposals that involve using animals. Paramount to the IACUC review of a research proposal is to question whether the proposed research is duplicative and to review and reflect on the justification provided by the researcher for the use of animals. Given the existing vast amount of information available on many aspects of the issue of lead toxicosis in many species of animals it is difficult to justify further use of animals to conduct yet another study on lead.

Peer reviewers will review this approach and assess the sufficiency of the report's conclusions regarding outstanding questions of scientific uncertainty and the contribution of ammunition to lead exposure in the species under consideration.

In my view, the authors of this report have established via review of peer-reviewed literature the contribution of ammunition to lead exposure to scavenging and birds of prey. This "approach" works in this case because of the preponderance of information available on the subject. Enough specific information is presented in the report to address attempts to postpone or stop calls for the development of alternatives to lead ammunition. At the same time, there is sufficient scientific rigor presented in this paper such that demands for further research before any action can be taken to prevent further use of lead ammunition is neither warranted nor justified.

I would not support this approach unless there was sufficient information on a subject to establish a threat of harm and scientific uncertainty. Scientific uncertainty is an indication of what is known and not known. It is not correct to interpret scientific uncertainty as nothing is known. Once a threat of harm is established, which it clearly is in this report, then steps must be taken to avoid harm before scientific certainty is established.

"The reviewers must ensure that any scientific uncertainties are clearly identified and characterized, and the potential implications of the uncertainties for technical conclusions drawn are clear".

The "scientific uncertainties" that I listed above were extracted by me based upon my interpretation of the text written in the "Executive Summary" of this report. The additional scientific uncertainties I suggested for consideration are ideas I developed from reading other sources. This report does not "clearly identify and characterize scientific uncertainties" nor does it discuss "the potential implications of these uncertainties." As best I can tell this aspect of the report was not done or it is certainly not clearly indicated in the report. The "Discussion Points" boxes at the end of each section in the report are simply summaries of the previous text. For the

most part the points in the boxes are correct (as they relate to the text). I did not find a discussion of the implication of scientific uncertainties within this report.

For this section of my review, I will answer the questions posed in The Charge to the Panel contained in Section A Scope of Work. I will not re-state each question; rather indicate it numerically. For the sake of brevity in my answers, I will refer to "USFWS Review of Lead Exposure and Effects to Scavenging Birds" as the Report.

Question 1. I believe that the objectives of the Report are clearly stated although not completely met. As I state above in my general comments, I do not see in this document a clear stand-alone statement of scientific uncertainties or a statement addressing the implications of these uncertainties within the Report. I believe the content of the Report is within stated parameters except when the parameters are not addressed (as in scientific uncertainties). I am not sure that the case reports (condors and eagles) provide that much additional support to the objectives of the Report. While they do provide greater detail about the two species, they do not introduce different concepts related to lead toxicosis. Stated differently, the core concepts of lead poisoning in scavenging birds are not exclusive to condors and eagles. I believe it is useful for the authors to consider shortening the Report by rethinking the value of the case reports as they relate to the issue of lead toxicosis.

Question 2. For the most part, I believe that the authors do summarize the text in the Discussion Points boxes. However, there is no discussion of the points. Rather, the statements in the boxes are re-statements, perhaps paraphrased, of material presented in the text.

Box on page 19: the lead statement is that "lead toxicity to birds is extremely well understood……". Yet, there are issues where information is not known (please see my list of scientific uncertainties above). Perhaps a better phrasing would be, "well documented" instead of "extremely well understood". The last statement in this box refers to the need of combining tissue concentrations of lead with clinical signs. There is no mention however, that the majority of birds presented for necropsy have no clinical signs. We are lucky to find the bird carcasses. This is an area of scientific uncertainty that needs discussion.

Box on page 24: the fourth statement relates to the lead shot ban and the conservation of ducks and the birds that feed on ducks. The last paragraph of the section, first sentence ends: "…. and birds that prey on waterfowl". Yet there are no data presented or referenced in the preceding text of this section on birds preying on waterfowl. All the information in the section is on waterfowl only.

Box on page 26: last statement, "carcasses and offal with lead ammunition fragments are available and attractive to scavenging birds". This implies that carcasses and offal that do not contain lead fragments are not attractive to scavenging birds?

Box on page 30: last statement, "…group feeding behavior can enhance their vulnerability to lead exposure……." This statement would benefit by an additional clarifying statement that multiple birds feeding on the same contaminated carcass become exposed and so more individuals within the same population are affected at the same time.

Box on page 43: here in the last statement the authors state that clinical signs diagnostic of lead toxicosis may not always be exhibited despite known exposure. Here again we have a statement rather than a discussion. There are now two references to the importance of clinical signs but there is also the fact that not all poisoned live birds present with clinical signs and most

carcasses presented for necropsy to determine a cause of death do not have observed clinical signs prior to death. This issue needs to be discussed.

Box on page 50: third statement: please avoid using the term "high" with regard to incidence of lead exposure in eagles as you have no reference to compare exposure rates. It is better to use "increased exposure" during hunting season.

Box on page 53: last statement: I understand what the authors are trying to get to but I think this needs more thought. I think the value of tissue concentration is understated in this Report. Please remember that lead is not supposed to be in anything alive. In general, birds submitted for necropsy have tissue levels of lead well above threshold levels. The presence of lead in the gastrointestinal tract is only indicative of exposure, not toxicosis. Tissue concentrations in the absence of clinical signs or presence of lead material are still diagnostic.

Box on page 70-71: be careful using the term "significant" as there is no associated mathematical/statistical rigor applied to the statement. Here it is being used as a relative descriptor that is largely a value statement of the authors.

Question 3. I believe that the authors have accurately summarized the existing peer-reviewed scientific literature on the effects of lead on scavenging birds. They have also established, by providing information from the peer reviewed scientific literature, that spent lead ammunition including fragments of lead bullets are an important source of lead to these birds. I do not believe that there are instances in this Report where different but equally reasonable conclusions might be drawn that differ from the conclusions of this Report and is supported by the literature.

Question 4. I believe that the authors have accurately summarized the data, analyses, and conclusions drawn by the authors of the scientific papers reviewed in the Report. The authors of the Report have drawn reasonable conclusions based upon the information they have complied for this report. The information the authors have compiled for the Report was written by qualified scientists who have published their results in reputable scholarly scientific journals.

Question 5. I believe the authors of this Report would benefit by reading the following paper: Cababree, EJ and Baldwin LA. 2002. Defining hormesis. Human and Experimental Toxicology 21:91-97.

Question 6. I do not believe that there are other potential sources of lead exposure that should been considered by the authors of the Report.

Question 7. I do not understand this question. The "scientific foundation" of this Report is in the experimental design, data collected and analyzed, and conclusions drawn by the authors of the papers cited in the Report. The authors of the Report do not present any original data or analyses. They have drawn their conclusions based upon the research of others. The writing of the Report could be improved, terminology tightened, information better presented graphically, but these are editorial changes that have nothing to do with the scientific foundation of the Report.

Editorial comments

While I recognize that it is not within the charge to the reviewers to provide comments on editorial aspects of this Report, I believe that there are some errors that need to be addressed and suggestions that will improve the quality of the Report. I offer the following comments:

This Report would benefit greatly from a complete editorial review. However, the following comments only relate to errors, consistency, and presentation.

Page 7 first full paragraph, last sentence: Please rewrite this sentence as I suspect carcasses and offal piles are attractive to scavenging birds whether they contain lead or not.
Page 7 second paragraph, last sentence: please consider providing more information as it is not intuitive that group feeding behavior enhances vulnerability to lead exposure.
Page 10 first full paragraph, first sentence: this is an incomplete sentence; "…from these metals is…"
Page 18 first full sentence on the page, 38 ppm dry weight (10 ppm dry weight)…
Page 18 Table 1: Kidney ppm should be wet weight.
Page 19 hormetic effect; please see Calabree and Baldwin, 2002. Defining hormesis. Human and Experimental Toxicology 21:91-97.
Page 20 first paragraph last sentence: please use laboratory instead of lab; slang.
Page 42 bottom of the page: did you mean "myocardial necrosis" instead of myocardial nephrosis?
Page 43 top of the page, …"were present in epithelium in turkey vultures…." which epithelium?
Page 45 Table 5, this table is very busy and difficult to read. It is an important message that would be better presented graphically.
Page 47 Turkey vultures, Kentucky; last sentence…..please replace "written up for publication" with "published".
Page 48 second paragraph, last sentence: threshold value of blood (1.0ppm) does not agree with Table 1 page 18
Page 48 last paragraph, first sentence: what are "lead exposure events"?
Page 51 "Two of these cases were positively confirmed by chemical analysis…….please more clearly state that the metallic particles were in fact lead.
Page 52 top of the page last sentence, "……over 50% of bald eagles admitted to rehabilitation facilities have ingested ammunition." Based upon what? Radiographs, chemical tests, dissection?
Page 52 Figure 8 this is a radiograph of a Bald eagle with a radio-opaque object in its abdominal cavity. Please provide more information as to how this object was confirmed to be not only metallic but a fragment of ammunition.
Page 53 text on isotopic studies; this information is important and powerful but could be better presented. As written, it is difficult to compare isotope levels. Please consider a graphic presentation of these data.

Please note that there are many more editorial corrections that I have not included as they will be addressed by the editorial review.

REVIEWER 2
Peer Review of:
USFWS Review of Lead Exposure and Effects to Scavenging Birds

GENERAL COMMENTS:

The document entitled *USFWS Review of Lead Exposure and Effects to Scavenging Birds* (hereafter referred to as the "document") serves as a useful review of literature and current science. It contains relevant information and data drawn from peer-reviewed published literature, government reports, and, to a limited extent when appropriate, persons and non-governmental organizations with appropriate expertise. In general, the document is a current and comprehensive treatise on the topic current up to the time at which it was written. There appear to be relatively few incidents of missing information, some of which are identified later in this review. All-in-all, the scientific foundation for this document appears to be sound.

However, there are some issues which hinder comprehension of material presented, and thus limit the utility of the document. First, and perhaps most importantly, the objective of the document is quite broad and vague. From the very outset, the intended purpose of the document is not specified. Further, it is unclear what species are to be addressed, which geographical region(s) are to be covered, and how alternates to lead ammunition fit into the discussion (clearly not alluded to in the title or objectives). As a result, the document does not appear to follow a predetermined outline, nor does it proceed towards a specifically stated goal. It seems to wander somewhat randomly from topic to topic like a compilation of literature reviews derived from different authors. An example of the lack of direction in presentation of information, or perhaps as a manifestation of the lack of focus, is the repetitive nature of the document. Numerous instances of repetition occur throughout. Moreover, there are numerous passages that are unclearly written, incomplete logic progressions, or simply grammatically challenged. Some of the "*Discussion Points*" are not well supported by information presented in preceding sections. These are all identified in a marked-up copy of the document which accompanies this review (See accompanying file in Appendix B).

Overall, the document contains the vast majority of pertinent data and information on this topic. Yet the organization of the document and its presentation limit the effectiveness of valuable information contained within. This reviewer encourages considerable revision of the document with particular attention given to clearly defining the objectives, focusing on the objectives and removing tangential information, avoiding repetition, and a general improvement in technical writing. Specific responses to the reviewer's charge follow:

1. ***Are the objectives of the USFWS Review of Lead Exposure and Effects to Scavenging Birds clearly stated and logical? Is the content of the report within the parameters of these objectives? If not, please identify the specific objectives that are unclear or illogical, or where content has strayed from the stated objectives.***

The primary objectives for the document are ostensibly laid out on page 11 (indicated as such by the table of contents). Here it states that the objectives of the "*paper*" are "*to gather and review*

current science on exposure and effects to wildlife from lead ammunition, and to address questions regarding scientific uncertainty." First, it is unclear what is meant by "paper." Is this document intended for publication? If so, in what format, and what is the intended outlet/readership? This issue should be addressed in the document in order to determine if it is suitable for its intended purpose.

The document gathers and reviews current science on exposure to, and effects from lead among birds in particular, but not for "wildlife" in general as stated here. Very little exposure data, and virtually no effect data are described for any species other than birds. As such, these objectives are either incorrect, or the document fails to address its stated purpose. The last portion of the objective refers vaguely to scientific uncertainty. One is left asking the question, "Uncertainty with regards to what?" It is difficult to determine if the document completes this objective, because it is, in and of itself, uncertain. Further, subsequent sections do not clearly identify what is uncertain. Clearly identifying areas of uncertainty, and then following up with data and information addressing areas of uncertainty would tremendously improve the outline, format, and flow of the document, and perhaps provide stronger support for *Discussion Points* included at the end of most sections.

Failure to provide a strong and specific objective statement at the outset of this document may have ultimately contributed to its seeming random and repetitive presentation of information. Clearly, there are significant amounts of information that fall within the parameters of the objective, but there are also significant amounts of material that seem to stray from the objectives (or are tangential at best). For example, there is an inordinate amount of information included on the ban of lead shot in waterfowl hunting. Without question, there are lessons to be learned from this historical event, but those lessons are not clearly spelled out, identified, or applied to the current crisis. Further, repetitive references and descriptions of bald eagles in the Great lakes has clear significance that informs about lead exposure and effects, but again, the take home message (specifically applied to all scavenging birds) is not clearly established. Another example which seems to veer from the topic is the passage dealing with alternative ammunition. Is this one of the "*questions regarding scientific uncertainty*" alluded to in the objective statement? If so, this and other scientific uncertainties should be clearly identified prior to each relevant section to assist the reader in understanding why issues are addressed in this document. Sub-objectives pertaining to each section could not be identified (and would be helpful), thus there did not appear to be an overall plan, or predetermined document outline.

2. Do the authors of the USFWS Review of Lead Exposure and Effects to Scavenging Birds draw the correct conclusions for each section of the report (see Discussion Points boxes), and are they supported by the material presented within that section? If not, please identify those that are not and the specifics of each situation.

In most instances, the authors draw correct conclusions from information presented in each section. However, there are instances where the authors appear to have overreached, or arrived at conclusions not entirely supported by information presented in preceding sections of the document. This reviewer will provide, in addition to comments written here, an accompanying PDF document identifying questionable *Discussion Points* and explaining why they do not

appear to be correct or sufficiently supported by information provided in the text. (See accompanying file in Appendix B).

3. Do the authors of the USFWS Review of Lead Exposure and Effects to Scavenging Birds draw reasonable and scientifically sound conclusions from the scientific information presented in the report? Are there instances in the report where a different but equally reasonable and sound scientific conclusion might be drawn that differs from the conclusion drawn by the Service and is supported by data in the literature? If any instances are found where that is the case, please provide the specifics of that situation.

The authors draw reasonable and scientifically sound conclusions from information presented, in most instances. However, there are instances where the authors do not appear to have drawn reasonable and scientifically sound conclusions, or they are not entirely supported by information presented. This reviewer will provide, in addition to comments written here, an accompanying PDF document identifying passages/assertions that do not appear to be reasonably sound conclusions or are not sufficiently supported by information provided in the text. (See accompanying file in Appendix B).

4. Does the USFWS Review of Lead Exposure and Effects to Scavenging Birds base its interpretations, analyses and conclusions upon the best available science? If any instances are found where the best available science was not used, please provide the specifics of each situation.

The majority of interpretations, analyses, and conclusions presented in this document are indeed based on the best available science. There is one significant exception, and one rather minor exception. The section on Toxicological Impacts of Lead in Birds, and more specifically, the Organism health response and effects section do not appear to contain sufficient information to fully understand some of the primary toxicological manifestations of lead exposure. Despite the fact that throughout the entire document neurological and neuromuscular deficits are described as symptoms of lead intoxication, nowhere does the document describe the mechanistic foundation(s) associated with these adverse effects, nor the apparent heightened sensitivity of developing organisms to the neurotoxic effects of lead.

Of somewhat less importance is the reliance of the authors on the Vyas, 1999 reference. The authors seem to equate difficulties associated with finding passerine species following pesticide application with the troubles associated with locating dead or moribund condors. It is likely that most dead, injured, or sick wildlife are obscured from observation, but extrapolating from a dead robin to a condor is a bit extreme.

Other than these instances, there appear to be only a few additional peer-reviewed published papers that perhaps should have been considered for inclusion/integration in the document to ensure that it was based on the most current science. They are:

Burnett et al., 2013, Eggshell thinning and depressed hatching success of California condors reintroduced to Central California, The Auk 115(3)477-91.

Cogan et al., 2012, Analysis of California condor (*Gymnogyps californianus*) activity using satellite telemetry data, The Open Ornithology Journal 5:82-93.

Helander et al., 2009, Ingestion of lead from ammunition and lead concentrations in white-tailed sea eagles (*Haliaeetus albicilla*), Science of the Total Environment 407(21)5555-63. Addresses impacts of a partial lead ban (no changes in incidence of lead poisoning).

Hunt, WG., 2012, Implications of sublethal lead exposure in avian scavengers, Journal of Raptor Research 46(4)389-393.

5. Are there any seminal peer-reviewed scientific papers that the USFWS Review of Lead Exposure and Effects to Scavenging Birds omits from consideration that would enhance the scientific quality of the document, or contribute to alternate conclusions that are scientifically sound? Please identify any such papers.

This reviewer could not identify any ***seminal*** peer-reviewed papers that would enhance the quality of the document or contribute to alternate conclusions that are scientifically sound. However, there are a few papers (listed above) that might be considered for inclusion should revision occur.

6. Are there other potential sources of lead contamination that are not addressed in the USFWS Review of Lead Exposure and Effects to Scavenging Birds that should be considered as exposure routes for scavenging birds, and specifically in the assessment of condor recovery?

There do not appear to be any other significant known or potential sources of lead contamination that are not appropriately considered by the authors. That does not discount the fact that there may be isolated incidents wherein unusual circumstances lead to exposure among scavenging birds; a contingency that is appropriately acknowledged in the document. In fact, the authors tended to be overly conservative when acknowledging that sources other than lead ammunition may contribute to scavenger exposure. It seems quite reasonable to more forcefully discount potential for lead exposure among scavengers (particularly with reference to condors) from any source other than lead ammunition and lead paint as detailed within the document. Foraging and nesting behaviors would appear to limit scavenger exposure to only ammunition and microtrash (including paint chips). A more definitive statement to this effect would seem warranted based on information provided. There again, with uncertainty with regards to which types of scavenging birds (condors, vultures, eagles, other raptors, etc.) are the intended foci of this document, there may indeed be other unimagined sources. As far as condors, most likely not.

7. Is the scientific foundation of the USFWS Review of Lead Exposure and Effects to Scavenging Birds reasonable and how can it be strengthened? Please identify any options to strengthen the scientific foundations.

The scientific foundation of this document is solid, sound, and reasonable. It appears to have been based on the latest and most relevant scientific data and studies. But, as stated above, the document does not appear to contain sufficient information to fully understand many of the neurotoxic effects associated with lead exposure. Specific mechanistic information is needed on neurological and neuromuscular deficits associated with lead intoxication, and it may be useful to include information on lead-induced neurodeficits among developing organisms.

There are numerous other instances where text presented within the document could be improved or clarified. These are detailed in an accompanying PDF document which identifies those sections and passages that are unclear, confusing, have alternate explanations/interpretations, or are not supported by information provided in the text or in the referenced literature (See accompanying file in Appendix B). Addressing those comments and/or providing clarification would likely serve to strengthen the scientific foundation of this document and clarify presentation thereof.

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REVIEWER 3

Peer Review of USFWS Review of Lead Exposure and Effects to Scavenging Birds

My overall assessment of the review is quite positive, although there are a number of sections that would benefit from some additional discussion and references, particularly with respect to the California Condor. I find no major gaps or errors in the presentation and believe it does a good job of providing a critical assessment of the subject.

Question 1. Are the objectives of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* clearly stated and logical? Is the content of the report within the parameters of these objectives? If not, please identify the specific objectives that are unclear or illogical, or where content has strayed from the stated objectives.

The content of the report appears to lie within the bounds of the objectives specified.

Question 2. Do the authors of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* draw the correct conclusions for each section of the report (see Discussion Points boxes), and are they supported by the material presented within that section? If not, please identify those that are not and the specifics of each situation.

I find no cases where conclusions appear not to be supported by materials presented.

Question 3. Do the authors of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* draw reasonable and scientifically sound conclusions from the scientific information presented in the report? Are there instances in the report where a different but equally reasonable and sound scientific conclusion might be drawn that differs from the conclusion drawn by the Service and is supported by data in the literature? If any instances are found where that is the case, please provide the specifics of that situation.

I agree with the authors in their interpretations and conclusions.

Question 4. Does the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* base its interpretations, analyses and conclusions upon the best available science? If any instances are found where the best available science was not used, please provide the specifics of each.

See Question 7 for instances where the report would benefit from amplifying discussions and references

Question 5. Are there any seminal peer-reviewed scientific papers that the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* omits from consideration that would enhance the scientific quality of the document, or contribute to alternate conclusions that are scientifically sound? Please identify any such papers.

See Question 7 for a list of references that would enhance the scientific quality of the review

Question 6. Are there any other potential sources of lead contamination that are not addressed in the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* that should be considered as exposure routes for scavenging birds, and specifically in the assessment of condor recovery?

I am not aware of additional credible potential sources of lead contamination that should be addressed. The lead in batteries might be suggested, but it is difficult to envision circumstances where scavenging birds would be exposed to this source and I am not aware of any examples here. Perhaps some brief comments could be added on this potential source.

Question 7. Is the scientific foundation of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* reasonable and how can it be strengthened? Please identify any options to strengthen the scientific foundations.

I find the scientific foundation of the review to be reasonable, but believe it can be strengthened is a number of respects, as follows:

Page 27, paragraph 2, line 6. Authors may want to add some discussion here of the following issue: Some large vultures are known to collect bone as diet for nestlings, presumably to meet calcium needs for growth, and it is possible that such species may preferentially ingest small hard objects that they encounter in the flesh of carcasses, including lead ammunition fragments. Although I am not aware of any rigorous documentation of such a tendency, it appears to be consistent with a known tendency for California Condors to often pass by large carcasses (which have little bone material small enough to be ingestible) to feed on smaller carcasses (with much more ingestible bone). Granted there may be other reasons for condors to prefer small carcasses, such as ease of penetration of hides, but it is noteworthy that studies of bones found in condor nests (e.g., Collins et al. 2000) indicate a heavy emphasis on bones of relatively small mammalian species, such as squirrels and rabbits, and relatively few bones of adult large mammals such as cattle and deer. If condors have a preferential tendency to ingest small hard materials encountered in the flesh of carcasses, such as ammunition fragments, this may increase their exposure to lead (see discussion in Snyder and Snyder 2005).

Also, somewhere in the review, and I am not sure exactly where, there should be some discussion of the low frequency with which condors regurgitate pellets of indigestible material such as fur, which is a potential route for reducing exposure to ingested lead objects. Compared to most large raptors, pellet casting is quite rare in condors, as discussed in Snyder and Snyder (2005), probably because condors ingest little fur in feeding and concentrate on soft tissues.

Page 30, last paragraph: With respect to scavengers ingesting whole bullets, it is relevant to mention a case documented by Snyder et al. (1986) where sifting of the substrate of a formerly active condor nest yielded a nearly whole lead bullet that had evidently been fired into the nest site, judging from distinctive rock grains embedded in the head of the bullet, but which had evidently been later ingested by a condor (most likely a nestling), judging from mammalian fur packed into the hollow core of the bullet. Whether the bullet had caused any mortality of condors, either by direct impact or by later ingestion, was unknown, but once present in a condor nest cave such a bullet could potentially poison successive generations of condors, as condor

chicks readily ingest hard objects from their nest substrates, and such a bullet could potentially pass through the guts of multiple chicks over the years, converting the nest site into a reproductive "black hole."

Page 43, bottom paragraph, line 5. This paragraph needs a better explanation for why data were limited to examples after 1991. Waterfowl surely did not suddenly become clean of lead fragments on that date and still continue to show some contamination today. Perhaps the 1991 date was chosen to reveal whether or not exposure of various species to lead continued despite the 1991 ban on lead shot for waterfowl? Needs clarification.

Page 48, bottom paragraph. The correlation of lead contamination with hunting seasons appears to be one of the most powerful arguments implicating exposure to lead ammunitions, as opposed to other sources of lead. Nevertheless, there is no reason to expect poaching activities to be limited to legal hunting seasons, and where year-round poaching becomes a major source of contaminated carcasses, the correlation with hunting seasons may be weakened *without* indicating that the contamination might be coming from sources other than ammunitions. Some discussion of this issue would be beneficial. At least several decades ago, deer poaching was very common in the range of California Condors in California (more than half the deer shot, by some estimates), so correlations of contamination events with hunting seasons should not be expected to always be very strong, even in locations where *all* contamination might be produced by ammunitions.

Another sort of lead-ammunition contamination within California Condor range that was not strictly confined to hunting seasons was the dispatch of coyotes caught in leghold traps by the Animal Damage Control (ADC) branch of the USFWS up until 1984 (see discussion in Snyder and Snyder 2000). Thus, part of the problem of lead contamination of condors may have been due to operations of the Service itself.

Page 71, paragraph 1 under Case Study 2, lines 4-6. The sentence ending in "(Grantham)" is OK for early historical times, but it does not accurately portray the state of knowledge regarding causes of the condor's decline in the 1980s. Suggest insert several sentences here to give a more thorough indication of how the lead issue came to the forefront with condors, as follows:
Causes of the condor decline in the 1980s were under intensive study by the USFWS and primarily implicated excessive mortality, at least in part due to lead poisoning, while breeding effort and success appeared to lie within normal expected limits (Meretsky et al. 2000, Snyder 2007). No persuasive evidence for significant stress from shooting, DDE contamination, or declining food supplies was found (Snyder and Snyder 2000, Snyder and Meretsky 2003). That lead poisoning may have been the main source of excessive mortality was suggested by (1) three documented condor deaths to lead contamination, (2) frequent lead contamination of living blood-sampled condors, (3) most mortalities occurring during the hunting season, and (3) almost identical mortality rates in juvenile condors as in adults. The lack of age dependency in lead contamination found in the study of Golden Eagles by Pattee et al. (1990) seems consistent with this latter finding. The growing evidence for lead contamination problems in the species, together with a loss of 40% of the wild population over the winter of 1984-85, led to a decision by the California Fish and Game Commission and the USFWS in 1985 to bring all wild

California Condors into captivity, as there was no politically feasible way to end the lead contamination threat in the wild in a timely manner.

Page 73, line 20. DDE normally operates through reproductive stress, not mortality. The evidence of DDT/DDE stress in the current coastal population is not persuasive. The paper of Burnett et al. (2013) found no significant correlation of DDE with eggshell thinning, and although this paper claimed a relationship of DDE levels with shell abnormalities, it provided no credible quantitative evidence linking these variables. Snyder and Meretsky (2003) found no relationship between shell abnormalities and DDE levels for the southern California population of the 1980s and likewise they were unable to document a persuasive correlation between DDE and shell thinning in this population. The paper of Kiff et al. (1979) has not proved replicable in its results and conclusions.

Page 74, paragraph 2, third line before end. Should add a reference to Meretsky and Snyder (1992) after "supplies"

References cited in the above paragraphs

Burnett, L.J., K.J. Sorensen, J. Brandt, E. Sanhaus, D. Ciani, M. Clark, C. David, J. Theule, S. Kasielki, and R.W. Risebrough. 2013. Eggshell thinning and depressed hatching success of California Condors reintroduced to central California. Condor 115:477-491.

Collins, P.W., N.F.R. Snyder, and S.D. Emslie. 2000. Faunal remains in California Condor nest caves. Condor 102:222-227.

Kiff, L.F., D.B. Peakall, and S.R. Wilbur. 1979. Recent changes in California Condor eggshells. Condor 81:166-172.

Meretsky, V.J. and N.F.R. Snyder. 1992. Range use and movements of California Condors. Condor 94:313-335.

Meretsky, V.J., N.F.R. Snyder, S.R. Beissinger, D.A. Clendenen, and J.W. Wiley, 2000. Demography of the California Condor: implications for reestablishment. Conservation Biology 14:947-967.

Pattee, O.H., P.H. Bloom, J.M. Scott, and M.R. Smith 1990. Lead hazards within the range of the California Condor. Condor 92:931-937.

Snyder, N.F.R. 2007. Limiting factors for wild California Condors, pp 9-33 *in* California Condors in the 21st Century (A. Mee and L. Hall, eds.). The Nuttall Ornithological Club and the American Ornithologists' Union.

Snyder, N.F.R., and V.J. Meretsky. 2003. California Condors and DDE: a re-evaluation. Ibis 145:136-151.

Snyder, N.F.R., R.R. Ramey, and F.C. Sibley. 1986. Nest-site biology of the California Condor. Condor 88:229-241

Snyder, N.F.R., and H.A. Snyder. 2000. The California Condor, a saga of natural history and conservation. Academic Press, London.

Snyder, N.F.R., and H.A. Snyder. 2005. Introduction to the California Condor. University of California Press, Berkeley.

Some Typos and other Minor Confusions deserving correction

Page 9, bottom paragraph, second sentence. This sentence is confusing, please clarify

Page 10, paragraph 2, first senence. Add "nontoxic" to end of sentence

Page 55, paragraph 1, line 7. "nesting" should be changed to "roosting." I'm not aware of any nesting of condors at the towers

Page 67, line 7 from bottom. "Dunstand" should be changed to "Dunstan"

Page 67, line 6 from bottom. "On" should be changed to "In" at end of line

Page 72, line 7. "17" should be changed to "7"

Page 80. First sentence on this page is gibberish. Please clarify.

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REVIEWER 4

Peer Review Comments: **Lead Exposure to Scavenging Birds: A Review for the U.S. Fish and Wildlife Service**

General Comments:

This report reviews much of the available scientific literature concerning exposure of scavenging birds to lead, and the toxicological implications. Although corvids are considered briefly, the focus is on raptorial birds, with in-depth evaluation of two case studies, the Bald Eagle and the California Condor. Although not stated as such (see more below), the tacit objective of the report is to make a case for restrictions on the use of lead projectiles for hunting. The report makes a strong case that California Condor populations continue to be impacted by lead from hunting projectiles, and that without further restrictions, populations will not thrive and expand, despite supplementation from captive breeding and rehabilitative interventions. The report also makes a case that the population(s) of Bald Eagles breeding and wintering in the Great Lakes is significantly impacted by exposure to the residues from lead projectile use. The report could make a stronger case that the problem is much broader, in fact continental, in scope. A comprehensive tabular summary of the literature on lead exposure and poisoning of wildlife across the U.S or preferably all of North America, possibly with a map showing the spatial scope, would help make this case. In the process it would highlight regions for which data is lacking. There are also a number of specific elements of the report and the scientific basis that could be strengthened.

Question 1: Are the objectives of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* clearly stated and logical? Is the content of the report within the parameters of these objectives? If not, please identify the specific objectives that are unclear or illogical, or where content has strayed from the stated objectives.

The stated objectives are as follows: "… to gather and review current science on exposure and effects to wildlife from lead ammunition, and to address questions regarding scientific uncertainty. While general toxicity of lead will be reviewed, the specific focus will be scientific literature regarding effects to scavenging birds from secondary poisoning."

The objectives are restated in the shaded box on P.12.

The objectives are clear enough, to review science relevant to lead poisoning of scavenging birds, as distinct from, for example, from waterfowl or upland game birds.
However, the report goes further in considering alternative materials than lead, and in making a case for banning or restricting use of lead ammunition. This should be included in the stated objectives or removed from the report.

For example, in the section on P. 30 "Bioavailability of ammunition to birds: Fragmentation", the authors compare fragmentation and availability of alternatives to lead, particularly, copper. While valid in the broader context, this section is not within the scope of the stated objectives.
The section beginning on P. 81, "Toxicity of alternative metals used in ammunition" goes beyond the stated objectives.

In the objective statement, the authors propose to 'address questions regarding scientific uncertainty'. Some further detail or clarity would be useful here. Is it uncertainty around variation in exposure, related to diet, foraging range, ability to detect and regurgitate pellets, in toxicity, for example, variation in species sensitivity to lead, chronic toxicity and survival in the wild under multiple stressors, etc.?

2. Do the authors of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* draw the correct conclusions for each section of the report (see Discussion Points boxes), and are they supported by the material presented within that section? If not, please identify those that are not and the specifics of each situation.

Generally the authors do a reasonable job of drawing the correct conclusions for each section of the report.
Some exceptions are noted below:
Box 2, P 19-20. Bullet 1 in this box is an overstatement of the situation. I would suggest rewording as follows: "Lead toxicity in birds has been studied in a variety of species and is relatively well understood".

Whereas, the progression of acute and chronic poisoning of birds dosed with or exposed to lead is well understood, there are still many outstanding questions of relevance to impacts of lead on wild bird populations. For example, little is known, although much can be surmised from the mammalian literature, about the impact of early developmental exposure of lead on the brain and, therefore, learning and subsequent survival and reproductive success in birds. We have no idea of threshold values in nestling blood, for example, that could be associated with chronic and significant effects in later life. The few available studies are summarized on P 18-19.

Bullet 4, while likely true and stated cautiously ("…may render…"), is still an overstatement based on the available literature.

Discussion Points box, P 48 – Note double wording problem in Bullet 1.

Discussion Points box, P 70 – Note wording problem in Bullet 4.

Discussion Points box, P 81 – Bullet 8 - discussion and conclusion on use of blood lead levels. Although the statement about the relatively rapid clearance of lead from blood is likely to be generally correct, this conclusion is based and supported by reference to Finkelstein et al 2012 study of California Condors. However, there is neither data on kinetics of lead in the Finkelstein paper, nor a primary reference. The supporting reference used by Finkelstein et al is unpublished report by Fry and Mauer 2003. The kinetics of lead uptake and clearance from blood, particularly in the case of ingestion of lead particles, is likely to be complex. Whereas, it is true that annual blood sampling as an approach for monitoring lead exposure in a wild population, such as the condor, probably would underestimate the degree of exposure, as stated in the report, the authors need to put these arguments on a sound footing. See for a start:

Barbosa Jr, F., Tanus-Santos, J. E., Gerlach, R. F., & Parsons, P. J. (2005). A critical review of biomarkers used for monitoring human exposure to lead: advantages, limitations, and future needs. *Environmental health perspectives*, 1669-1674.

3. Do the authors of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* draw reasonable and scientifically sound conclusions from the scientific information presented in the report? Are there instances in the report where a different but equally reasonable and sound scientific conclusion might be drawn that differs from the conclusion drawn by the Service and is supported by data in the literature? If any instances are found where that is the case, please provide the specifics of that situation.

In response to this question, the report's conclusions, as described on Pages 86 and 87, are examined in turn:

- "Scientific evidence points to lead ammunition as the most frequent cause of mortality in lead exposure cases"

A strong case has been made to support that conclusion.

- "A suite of evidence exists to support this conclusion, including the behavioral ecology and physiology of scavenging birds…"

A reasonably strong case has been made that aspects of the foraging behavior, including group foraging and flocking at carcasses is important as a factor to avian scavenger exposure to lead ammunition from game carcasses. Similarly, the particular gut physiology is also important in the digestion of ingested lead particles. I think that a good case has been made on this topic.

- "… their sensitivity as exhibited in controlled dosing studies…"

Presumably this means 'relative' sensitivity in a toxicological sense. In this case, I do not believe that are good comparative data from controlled dosing studies under the same experimental conditions with different species of birds to show that scavenging birds, particularly raptors and vultures, are relatively more sensitive to lead.

- "… the bioavailability of lead ammunition due to fragmentation, recovery of ingested lead fragments or pellets…observations of birds feeding…"
-

These part of the conclusions statement is particularly well examined and supported in the report.

- "…isotopic analyses relating tissue concentrations to ammunition…"

The data on this topic is somewhat complex, but has been given reasonable treatment.

- ...patterns of mortality coincident with hunting seasons…"

This available data are consistent with the overall conclusion.

- "…diagnosis of lead poisoning by well-established tissue thresholds and clinical signs…"

There is a reasonable body of published literature that supports the conclusions of the report.

"...lack of abundant evidence for other sources of lead…"

Lead from mining sources would benefit from further evaluation. See below.

However, paragraph 2 of the concluding statement is not consistent with stated objectives of the report to review the literature. It deals with measures that could be taken to reduce the degree of exposure and poisoning.

Further evaluation of the conclusions in paragraph 2: The only conclusion that is really feasible and should be listed first is: "Lead can be replaced in ammunition by alternative metals…" But the report does not address many aspects of this possible action, so this conclusion should be removed or the report should address this topic more comprehensively from objectives through to assessment of relevant information such as effectiveness of alternatives, cost factors, and essentially socio-economic and legal factors such likelihood of broad acceptance by hunting communities, degree of compliance, whether a broad ban on lead ammunition is readily enforceable, politics, such as the response of other jurisdictions with legal authority over hunting, which in the U.S. presumably means state governments, etc.

4. Does the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* base its interpretations, analyses and conclusions upon the best available science? If any instances are found where the best available science was not used, please provide the specifics of each situation.

The review draws heavily on two case studies, the Great Lakes Bald Eagle and the California Condor. Given that the geographical focus of the work is the U.S., those are logical choices. They are also well documented case studies with large sample sizes in one case, and an extensive body of supporting research in the other.

Some other relevant U.S. work that is not cited:

Pagel, J. E., Sharpe, P. B., Garcelon, D. K., Little, A. E., Taylor, S. K., Faulkner, K. R., & Gorbics, C. S. (2012). Exposure of bald eagles to lead on the northern Channel Islands, California. *Journal of Raptor Research*, *46*(2), 168-176.

There is also a broader literature on lead exposure and poisoning of eagles in western Canada, mainly by Mark Wayland and colleagues, that is relevant and would be supportive of the report's conclusions, e.g. (and works listed on p. A-25):

Langelier, K. M., Andress, C. E., Grey, T. K., Wooldridge, C., Lewis, R. J., & Marchetti, R. (1991). Lead poisoning in bald eagles in British Columbia. *The Canadian Veterinary Journal*, *32*(2), 108.

Elliott, J.E., K.M. Langelier, A.M. Scheuhammer, P.H. Sinclair, P.E. Whitehead. 1992. Incidence of lead poisoning in Bald Eagles and lead shot in waterfowl gizzards from British Columbia, 1988-91. *Canadian Wildlife Service, Progress Notes*, Ottawa, ON, No. 200, 7pp.

NOTE: the above paper is incorrectly cited in the Reference list, and also is incorrectly listed in Table 5 as an example of lead exposure after "the 1991 lead shot ban". The data in the report cover the period up until 1991. Also, note, that the title of Table 5 is inaccurate. The 1991 ban was not North American wide. British Columbia banned lead shot for water fowl hunting in 1996, but the national ban in Canada was not implemented until 1999. The history of regulatory decisions is detailed in the following article:

Scheuhammer, A. M., & Thomas, V. G. (2011). Eliminating Lead from Recreational Shooting and Angling: Relating Wildlife Science to Environmental Policy and Regulation in North America. In *Wildlife Ecotoxicology* (pp. 359-382). Springer New York.

More Western Canadian lead and raptor studies:

Miller, M. J., Restani, M., Harmata, A. R., Bortolotti, G. R., & Wayland, M. E. (1998). A comparison of blood lead levels in bald eagles from two regions on the great plains of North America. *Journal of Wildlife Diseases*, *34*(4), 704-714.

Wayland, M., Neugebauer, E., & Bollinger, T. (1999). Concentrations of lead in liver, kidney, and bone of bald and golden eagles. *Archives of environmental contamination and toxicology*, *37*(2), 267-272.

Miller, M. J. R., Wayland, M. E., & Bortolotti, G. R. (2001). Exposure of migrant bald eagles to lead in prairie Canada. *Environmental Pollution*, *112*(2), 153-162.

Miller, M. J., Wayland, M. E., & Bortolotti, G. R. (2001). Hemograms for and nutritional condition of migrant bald eagles tested for exposure to lead. *Journal of wildlife diseases*, *37*(3), 481-488.

Miller, M. J. R., Wayland, M. E., Dzus, E. H., & Bortolotti, G. R. (2000). Availability and ingestion of lead shotshell pellets by migrant Bald Eagles in Saskatchewan. *Journal of Raptor Research*, *34*(3), 167-174.

Wayland, M., Wilson, L. K., Elliott, J. E., Miller, M. J. R., Bollinger, T., McAdie, M., ... & Froese, J. M. W. (2003). Mortality, morbidity, and lead poisoning of eagles in western Canada, 1986-98. *Journal of Raptor Research*, *37*(1), 8-18.

There is a paper (Peterson, C.A., S.L. Lee and J.E. Elliott. 2001. Scavenging of waterfowl carcasses by avian predators in the Fraser River delta of British Columbia. *Journal of Field Ornithology* 72: 150-159) which examines and describes avian scavenging on waterfowl carcasses. Although concerned originally with pesticide poisoning, the paper shows how various scavengers are attracted to carcasses and a single carcass can result in a large congregation of eagles. It is pertinent to the statements at L 1 and the end of para 2, P 27, P 28, P 29, P 44. That

study also found that a variety of raptor species, in addition to the normally recognized avian scavengers, were drawn to and attempt to feed on duck carcasses.

Related to that work, studies of pesticide poisoning of raptors is potentially relevant to lead exposure and poisoning. Elliott et al (Elliott, J.E., A. Birmingham, L.K. Wilson, M. McAdie, and P. Mineau. 2008. Fonofos poisons raptors and waterfowl several months after labeled application. *Environmental Toxicology & Chemistry*. 27:452-460) related an increase incidence of pesticide poisoned eagles to population recovery in south western British Columbia. There are parallels to lead exposure as the eagles were exposed to pesticides from feeding on carcasses of ducks which had ingested pesticides granules while feeding during winter in farmed fields, treated the previous spring.

There is some excellent work on a the congeneric White-tailed Sea Eagle *Haliaeetus albicilla* from Germany that perhaps would provide another case study, or should at least be cited and considered more than it is. The overall landscapes of the two countries differ, in that the U.S. still has much larger expanses of less developed and intensively managed land. However, there are also many similarities. Deer species in particular are heavily hunted in both countries, as are upland small game and wild pigs. The discarded carcasses and offal piles provide a continuing source of lead. These two *Haliaeetus* species are almost indistinguishable in terms of ecology and behavior. The report refers to some of the work done in Germany, such as the one cited paper by Krone; however, there are other related papers, and likely also relevant grey literature that possibly could be obtained by contacting Dr. Oliver Krone. See for example:

Nadjafzadeh, M., Hofer, H., & Krone, O. (2013). The link between feeding ecology and lead poisoning in white-tailed eagles. *The Journal of Wildlife Management*, *77*(1), 48-57.

The socio-economic situation in Germany is also comparable with regard to attempts to mitigate lead poisoning of raptors in that both countries have large, powerful and conservative hunting lobbies. See for example:

Trinogga, A., Fritsch, G., Hofer, H., & Krone, O. (2013). Are lead-free hunting rifle bullets as effective at killing wildlife as conventional lead bullets? A comparison based on wound size and morphology. *Science of the Total Environment*, *443*, 226-232.

Other highly relevant European work on lead exposure and poisoning in Haliaeetus specifically comes from Sweden. It is work that could help support the scientific conclusions of this report:

Helander, B., Axelsson, J., Borg, H., Holm, K., & Bignert, A. (2009). Ingestion of lead from ammunition and lead concentrations in white-tailed sea eagles (< i> Haliaeetus albicilla</i>) in Sweden. *Science of the total environment*, *407*(21), 5555-5563.

From elsewhere in Europe:

Fernandez, J. R. R., Höfle, U., Mateo, R., de Francisco, O. N., Abbott, R., Acevedo, P., & Blanco, J. M. (2011). Assessment of lead exposure in Spanish imperial eagle (Aquila adalberti) from spent ammunition in central Spain. *Ecotoxicology*, *20*(4), 670-681.

Hernández, M., & Margalida, A. (2009). Assessing the risk of lead exposure for the conservation of the endangered Pyrenean bearded vulture (< i> Gypaetus barbatus</i>) population. *Environmental Research, 109*(7), 837-842.

Kenntner, N., Crettenand, Y., Fünfstück, H. J., Janovsky, M., & Tataruch, F. (2007). Lead poisoning and heavy metal exposure of golden eagles (Aquila chrysaetos) from the European Alps. *Journal of Ornithology, 148*(2), 173-177.

Knott, J., Gilbert, J., Hoccom, D. G., & Green, R. E. (2010). Implications for wildlife and humans of dietary exposure to lead from fragments of lead rifle bullets in deer shot in the UK. *Science of the total environment, 409*(1), 95-99.

Komosa, A., & Kitowski, I. (2008). Elevated lead concentration in skeletons of diurnal birds of prey Falconioformes and owls Strigiformes from eastern Poland—ecological approach and review. Ecol. *Chem. Eng. S, 15*, 349-358.

Mateo, R., Cadenas, R., Manez, M., & Guitart, R. (2001). Lead shot ingestion in two raptor species from Doñana, Spain. *Ecotoxicology and Environmental Safety*, *48*(1), 6-10.

From other localities:

Nam, D. H., & Lee, D. P. (2009). Abnormal lead exposure in globally threatened Cinereous vultures (Aegypius monachus) wintering in South Korea. *Ecotoxicology*, *18*(2), 225-229.

5. Are there any <u>seminal</u> peer-reviewed scientific papers that the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* omits from consideration that would enhance the scientific quality of the document, or contribute to alternate conclusions that are scientifically sound? Please identify any such papers.

Already list above, but would suggested these are seminal papers that have been omitted:

Trinogga, A., Fritsch, G., Hofer, H., & Krone, O. (2013). Are lead-free hunting rifle bullets as effective at killing wildlife as conventional lead bullets? A comparison based on wound size and morphology. *Science of the Total Environment, 443*, 226-232.

Other recent and important papers that may prove seminal:

Johnson, C. K., Kelly, T. R., & Rideout, B. A. (2014). Lead in Ammunition: A Persistent Threat to Health and Conservation. *EcoHealth*, 1-10.

Thomas, V. G. (2013). Lead-free hunting rifle ammunition: product availability, price, effectiveness, and role in global wildlife conservation. *Ambio*, *42*(6), 737-745.

6. Are there other potential sources of lead contamination that are not addressed in the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* that should be considered as exposure routes for scavenging birds, and specifically in the assessment of condor recovery?

Consideration of the available information on alternate sources of lead has received reasonable coverage in the report. The one area that probably should receive further examination is mining and smelting.

The swan data from the Coeur d'Alene area is cited, but there is also data on a raptor, the osprey.

Henny, C. J., Blus, L. J., Hoffman, D. J., Grove, R. A., & Hatfield, J. S. (1991). Lead accumulation and osprey production near a mining site on the Coeur d'Alene River, Idaho. *Archives of Environmental Contamination and Toxicology*, *21*(3), 415-424.

One of the papers by Berglund is cited in the report. Additionally, there are a number of other studies of exposure and effects on birds of lead from mining sources in Europe. Most of this work focuses on passerine species, which are more likely to provide a vector of lead to bird-eating raptors or other predators, rather than avian scavengers per se. They do, however, demonstrate movement of lead from mining and smelting sources into wildlife food chains. Some examples:

Berglund, Å. M., Klaminder, J., & Nyholm, N. E. I. (2008). Effects of reduced lead deposition on pied flycatcher (Ficedula hypoleuca) nestlings: tracing exposure routes using stable lead isotopes. *Environmental science & technology*, *43*(1), 208-213.
Berglund, Å., Sturve, J., Förlin, L., & Nyholm, N. E. I. (2007). Oxidative stress in pied flycatcher (Ficedula hypoleuca) nestlings from metal contaminated environments in northern Sweden. *Environmental research*, *105*(3), 330-339.

Berglund, Å., & Nyholm, N. E. I. (2011). Slow improvements of metal exposure, health-and breeding conditions of pied flycatchers (Ficedula hypoleuca) after decreased industrial heavy metal emissions. *Science of the Total Environment*, *409*(20), 4326-4334.

De Francisco, N., Ruiz Troya, J. D., & Agüera, E. I. (2003). Lead and lead toxicity in domestic and free living birds. *Avian Pathology*, *32*(1), 3-13.

Eeva, T., Belskii, E., Gilyazov, A. S., & Kozlov, M. V. (2012). Pollution impacts on bird population density and species diversity at four non-ferrous smelter sites. *Biological Conservation*, *150*(1), 33-41.

Eeva, T., Lehikoinen, E., & Pohjalainen, T. (1997). Pollution-related variation in food supply and breeding success in two hole-nesting passerines. *Ecology*, *78*(4), 1120-1131.

Eens, M., Pinxten, R., Verheyen, R. F., Blust, R., & Bervoets, L. (1999). Great and blue tits as indicators of heavy metal contamination in terrestrial ecosystems. *Ecotoxicology and Environmental Safety*, *44*(1), 81-85.

Scheifler, R., Coeurdassier, M., Morilhat, C., Bernard, N., Faivre, B., Flicoteaux, P., ... & Badot, P. M. (2006). Lead concentrations in feathers and blood of common blackbirds (Turdus merula) and in earthworms inhabiting unpolluted and moderately polluted urban areas. *Science of the total environment*, *371*(1), 197-205.

7. Is the scientific foundation of the *USFWS Review of Lead Exposure and Effects to Scavenging Birds* reasonable and how can it be strengthened? Please identify any options to strengthen the scientific foundations.

The overall scientific foundation could be strengthened by broadening the spatial scope of the considered literature and evidence, as outlined above. Big game and 'varmint' hunting occurs across North America as do scavenging birds, and it is likely, therefore, that exposure and poisoning also occurs across the continent, including those jurisdictions where it has not been well documented to date.

One option may be to map known concentrations of raptors during fall and winter along with areas of dense hunting activity and look for overlaps, essentially expanding on Figure 9. This could be along the lines of overlays of waterfowl hunting and lead exposure of ducks (Scheuhammer, A.M., Norris, S.L., 1995. A review of the environmental impacts of lead shotshell ammunition and lead fishing weights in Canada. Canadian Wildlife Service Occasional Paper No. 88,Ottawa, Canada.).

Also the parallels with the situation in Germany and elsewhere of eagle and vulture species would enhance the argument that ammunition lead is a significant and wide spread factor reducing survival, and likely reproductive success of birds which scavenge readily on game carcasses.

Specific comments:

P 77 – 78 – there seems to be some confounding of blood versus liver lead threshold values. See for example, section 2, L 4-5 – liver versus blood lead concentrations. Similarly, P 78, para 2, L 5 – liver lead?

P 80 – the narrative / paraphrasing of the information from Parish et al 2009 is not well written and should be revised. Why are the last sentences in this paragraph written in the conditional form ("would move", "would stay")? Narrate in past tense.

There are other wording / grammatical issues throughout the text, which needs to be carefully edited.

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REVIEWER 5

REVIEW OF: "Lead Exposure to Scavenging Birds: A Review for the U.S. Fish and Wildlife Service by the Environmental Contaminants Program, July 2, 2013"

DATE OF REVIEW: February 14, 2014

General Comments:

The authors have made a worthy effort in laying out the published facts relating to lead exposure in avian scavengers, and they have done so in a pleasingly organized way. They have cited the key references, although perhaps not always in sufficient depth. An explanation, for example, of what Green et al. 2008 suggested about the implications of multiple exposures comes to mind (more later). I cannot tell to what extent the authors focused on "...an evaluation of the scientific uncertainty," as promised in the Objectives section, and called for six times in the peer group's Scope of Services. The paper contains little of that, presumably because the authors found almost nothing to quibble about. Perhaps it is asking too much of them to detect flaws and contradictions within so large a volume of material, and it might be more to the point were they to devote a section at the end to listing and explaining the gaps in needed knowledge they may have detected during their review. Again, and as a prime example of uncertainty, the incidence of death from single-event exposures versus multiple exposures over time has been largely ignored in the published literature, and yet the issue is of considerable importance in understanding the epidemiology of lead exposure and death in condor (and eagle) populations and what levels of management are required to sustain them.

All in all, a proper explanation of the pathways and implications of lead exposure to avian scavengers requires knowledge of toxicology, avian life history, behavior, population ecology, and an understanding of firearms and hunting practices. The paper deals best with toxicology, but sometimes a bit more superficially with the other topics. In consideration of the diverse readership of the document that emerges from this exercise and the need for clear communication, I recommend consultation with an avian population ecologist and perhaps an experienced hunter to assist the authors in a revision. This suggestion in no way ignores the depth of the current stage of examination and its clarity of presentation. As good examples of these virtues, I refer to the entire section on "Toxicological Impacts of Lead in Birds" (pages 12-20) and to the first eight lines under the heading "Use of stable isotopes..." on page 53.

Question 1. *Are the objectives of the USFWS Review of Lead Exposure and Effects to Scavenging Birds clearly stated and logical? Is the content of the report within the parameters of these objectives? If not, please identify the specific objectives that are unclear or illogical, or where content has strayed from the stated objectives.*

The Objectives paragraph appears to contain the essential elements one might expect from the title and the purpose of such a document. My job is be picky, however, and alas, I do find the paragraph somewhat indirect and would rearrange it as follows: "The objective of this paper is to gather and review current science on the effects of ingested lead upon scavenging birds and their populations. The document accordingly focuses upon the general toxicity of lead, its bioavailability, physiology of uptake, methods of measuring and evaluating its presence and

pathology, exposure pathways, population-level effects, case studies of lead-affected wildlife, other lead sources, alternatives to lead ammunition, and questions regarding scientific uncertainty."

Question 2. *Do the authors of the USFWS Review of Lead Exposure and Effects to Scavenging Birds draw the correct conclusions for each section of the report (see Discussion Points boxes), and are they supported by the material presented within that section? If not, please identify those that are not and the specifics of each situation.*

The first discussion point on page 19 and earlier in the text asserting that lead toxicity in birds is "extremely well understood" is an overstatement. It is quite enough to say "...well understood," and even then, presumptuous. Consider, for example, the ever widening discoveries of lead's sublethal effects upon humans, and from concentrations deemed insignificant only a short while ago. Avian toxicologists have, in comparison, made little progress in exploring the sublethal impacts of lead upon birds and the extent to which those effects influence demography. In mentioning this, I note that the authors quite adequately deal later on with what literature there is on sublethal effects. My point is simply that most of the subject awaits an understanding.

The last discussion point in the box on page 19 refers to "...specific physiological responses that are measurable, but may vary among individuals and species," and paraphrases an earlier mention of this on page 6 of the Executive Summary. The statement is true but fails to inform the reader of lead's more general nature. The authors delay until page 38 the mention that lead mimics calcium at binding sites, a fact that makes lead so universally pernicious, especially with respect to nerve action. The authors might enlarge a bit upon the wide diversity of lead's human health manifestations, even in very small concentrations, and perhaps make (or refute) the point that avian lead physiology cannot be expected to differ categorically from that of humans. The authors might consider discussing such issues earlier in the document. For Discussion Points, they might consider saying something like "Lead exposure yields a number of specific and measurable responses, but is also largely universal in its effects;" and perhaps "Science is repeatedly discovering new manifestations of its pathology in humans and other species."

Question 3. *Do the authors of the USFWS Review of Lead Exposure and Effects to Scavenging Birds draw reasonable and scientifically sound conclusions from the scientific information presented in the report? Are there instances in the report where a different but equally reasonable and sound scientific conclusion might be drawn that differs from the conclusion drawn by the Service and is supported by data in the literature? If any instances are found where that is the case, please provide the specifics of that situation.*

The authors (on page 75) appear to support the argument that "...anything less than complete removal of lead ammunition from the condor's habitat could result in a high frequency of exposure in the population." That idea was conceived by Green et al. (2004) with respect to Old World vultures threatened by diclofenac, a veterinary compound in livestock carcasses that was shown to be lethal in a single meal. Whereas it is true that single doses of lead can be lethal in condors, the incidence of such outcomes is unknown. Moreover, we now know from blood monitoring that virtually all free-ranging condors are fairly often exposed to lead, and that they usually survive it without intervention. Green et al. (2009) modeled data from the condor

population in Arizona and suggested that the condition of multiple exposures over several months was the more likely factor accounting for lethality, and most probable through "....a cumulative effect of protracted high blood lead levels on organ function." I think the authors of the document I am reviewing may have overlooked this possibility when they examined Finkelstein et al. (2012). That paper argued (wrongly, in my opinion) *"...that if only 0.5% of carcasses are contaminated with lead, the probability that, over 10 y, a condor will feed on a contaminated carcass is still 85–98%. Thus, very low carcass contamination rates are required to avoid high probabilities of lead poisoning* [my emphasis] *within the condor population."* Obviously, the complete elimination of lead-based ammunition would greatly enhance the likelihood of condor recovery and is thus highly desirable, but one must not enlarge the case beyond its factual underpinnings. The authors are to be commended for having not done so (they mention "exposure" rather than "poisoning"), but in the interest of discussing the faults and virtues of the published literature, they might consider making some of these points and at least modifying the one implying the necessity of complete removal quoted in the first sentence of this paragraph. Remember that, diclofenac permeates a carcass whereas ammunition lead is particulate in its distribution, such that a meal does not equate to an exposure.

Question 4. *Does the USFWS Review of Lead Exposure and Effects to Scavenging Birds base its interpretations, analyses and conclusions upon the best available science? If any instances are found where the best available science was not used, please provide the specifics of each situation.*

Yes, all in all, I believe the authors consulted the best available science, as evidenced within their list of cited references. My concerns mainly focus upon the need for deeper, clearer explanation.

Question 5. *Are there any seminal peer-reviewed scientific papers that the USFWS Review of Lead Exposure and Effects to Scavenging Birds omits from consideration that would enhance the scientific quality of the document, or contribute to alternate conclusions that are scientifically sound? Please identify any such papers.*

Seminal? I can think of none.

Question 6. *Are there other potential sources of lead contamination that are not addressed in the USFWS Review of Lead Exposure and Effects to Scavenging Birds that should be considered as exposure routes for scavenging birds, and specifically in the assessment of condor recovery?*

The authors mention the issue of "varmint shooting," but I suggest they expand upon it, as it is common practice throughout the southwest. Lead-based bullets manufactured and advertized for coyotes and other "varmints" are specifically designed to explode upon impact. Coyotes shot with those bullets, for example, often show no exit-wound, and the entire array of hundreds of bullet fragments may therefore remain within the animals. The authors might consider describing these types of bullets and the implications of their use. There are a number of brands, and only slightly modified versions are advertised for big game hunting, with many hunters inappropriately using them for deer and elk. Some manufacturers mislead users in describing these bullets as exhibiting "controlled expansion," as on the following web page: http://www.nosler.com/ballistic-tip/ The video on that page falsely depicts the bullet as

remaining in one piece upon impact. Now take a look at the second row in Table 2 in Grund et al. (2010).

I recommend the authors delete the inference on page 33 that one type of "controlled expansion" bullet was relatively safe according to Grund et al. (2010). The term is far too loosely applied by advertisers.

On page 7, the authors mention that "non-expanding bullets" may nevertheless fragment. The reason why they are generally not used in hunting, however, is because, by not "mushrooming," they are inefficient at transferring energy and are therefore less quickly lethal (and therefore less humane). Later on in that paragraph, it should be mentioned that monolithic copper bullets in use today "mushroom" very effectively and are as humane or more so than lead-based bullets in effecting quick kills.

Again, to the extent that varmints are left in the field, varmint shooting has the potential of presenting a great deal of consumable lead to scavengers and the matter should be discussed more thoroughly. There are some illuminating radiographs available that could be placed in the report.

I have not seen clear evidence that ground squirrel and jackrabbit shooting is an important factor in condor lead exposure, but the activity is commonplace in some parts of the condor range, as is coyote shooting. After mentioning a list of hunted taxa on page 74, the authors follow (top of page 75) with a statement about the volunteer lead abatement program in Arizona as mitigating, but they should also mention that the program applies only to ungulates.

Question 7. *Is the scientific foundation of the USFWS Review of Lead Exposure and Effects to Scavenging Birds reasonable and how can it be strengthened? Please identify any options to strengthen the scientific foundations.*

Yes, the overall scientific foundation is reasonable.

The authors might explain (and early on) the several levels of impact that lead-caused mortality and morbidity can inflict upon scavenger populations. The first is population inviability, a condition that would characterize lead's effect on the California condor in the absence of periodic releases, blood-lead monitoring, and treatment. The second is instability in which the additive effects of each morality agent and reproductive impairment render populations of territorial species (e.g., eagles) less secure by reducing non-breeder reserves (floaters). The third is sublethal impairment of individuals, a topic which authors capably reviewed.

Along these lines, the report should also explain that condor and eagle populations are far more sensitive to adult deaths than to deaths of younger birds, and that while some mortality agents disproportionately affect the younger age categories, others are indiscriminate in killing older birds. As it is, the authors (and others) lump the deaths of young and old condors in their reporting, an obscuration that understates the significance of lead poisoning. See, for example, pages 76 and 77.

On the top of page 35, I observe that "0.01 - 17.21 mg" is not a concentration.

The authors wait until page 80 to mention depuration and the short half-life of lead in condor blood, a factor that makes blood lead level measurements usually unreliable in assessing the degree of exposure. The reader needs such understanding to evaluate Table 1 and information on blood lead levels and threshold values throughout. I recommend a much earlier explanation.

Another point that should be made is that hunters normally target the thorax of large animals, and that is why offal piles usually contain many fragments. Hunt et al. (2006) found that 50% of 20 deer offal piles in their sample contained over 100 fragments.

Near the top of page 26, the authors seem (mildly) to suggest that varmint shooting is relatively uncommon. Varmint shooting can, in fact, be widespread and intense in regions inhabited by condors and golden eagles and likely represents a significant source of lead to these species, especially considering the highly fragmenting bullets often used for such purposes.

On pages 77 and 78, the authors allude to chelation therapy as a cause of visceral gout and death, but they do not explain that the injurious therapy was of a particular type, an oral succimer treatment which is no longer in use, or has been revised. Conflating standard chelation injections with oral succimer is therefore misleading. The assertion on page 78 that condor death in Arizona resulted from chelation therapy is false; the succimer treatment was never applied in Arizona. Moreover, the statement on line 5 of the first paragraph on page 78 to the effect that five condors had blood levels below the threshold level for toxicosis at the point of death is also misleading in consideration that chelation reduces blood lead levels. Lastly is the issue of the blood lead levels themselves. On page 78, 1st paragraph, there is the parenthetical "...all under 5.2 ppm wet weight," which equates to a whopping 520 micrograms per deciliter (also note the 3.2 ppm in the blood on the previous page). And wet weight for a blood lead level? The sentence (and paragraph, and indeed, almost the entire condor section except for the last paragraph) is fraught with ambiguity.

Explaining the implications of additive mortality to the population dynamics of raptors and other scavengers would fit nicely with the objectives of this document.

I may have missed this, but the authors might mention the idea that scavengers of ungulates and other large mammals may not encounter enough calcium to adequately compete with lead for binding sites. Would not bone calcium be generally less available to scavengers of large mammals than of small ones? Also scavengers of large mammals may ingest less hair than scavengers of small mammals, making it less likely that lead would be bound and regurgitated in a pellet.

To hunters, the term "upland game" normally applies to hunted birds other than waterfowl. The authors tend throughout to confuse it with big game hunting, small game hunting (e.g., rabbits), and varmint shooting.

In the second-to-last sentence of the first paragraph on page 57, the phrase beginning with "...rises in isotopic ratios" could be struck. Environmental ambience would not produce a spike of 50-90 ppm, although I might have misunderstood.

The authors should mention that, among condors that have simply disappeared and have not been resighted, it is likely that some proportion have died of lead poisoning, meaning that current knowledge of the incidence of lead-related death is an underestimate.

The authors might mention the increase of surface-area (available to stomach acids) with increased fragmentation in the section on "Physiology of raptors and scavenging birds."

Bullet fragments radiate rather than migrate from wound channels (see pages 7, 30, 32, 33, 37).

At the bottom of page 69 and elsewhere, the authors point out that not all lead-poisoned bald eagles are found and collected. They later mention that bald eagles are conspicuous (often associated with water bodies), and that increases the chance that fatalities or moribund birds will be found. They might also mention somewhere the obvious fact that stricken golden eagles are unlikely to be detected within the vast landscapes they inhabit, and that their rates of mortality and morbidity from lead ingestion may also be high, considering their affinity for carrion. The point is that mortality rates for bald eagles may not be "...increased" or in any way unique, as suggested in Discussion Point 1 at the bottom of page 70.

On the bottom of page 73 in the second-to-last paragraph, we see "Microtrash ingestion, predation, depredation, and exposure to DDT/DDE in the coastal population provide additional mortality events." How is "depredation" a "mortality event?" Indeed, a review of the four times in the ms (pages 10, 73, 75, and 81) that the word "depredation" appears suggests that the authors may be uncertain as to its meaning. Also, DDT/DDE is best classed as a reproductive impairment rather than a mortality factor. I suppose an embryo is a life stage subject to mortality, but it does not figure as such in standard demographic analysis. Sperm, for example, is also a mortal life stage. The authors might better end with "...impact vital rates."

Figure 3 (page 31) does not appear to be an offal pile, although it might be a small portion of one (wrapped in paper?). Note the distinct margins and the size of the fragments relative to the entire image. An informed reader would be skeptical, so perhaps more information is needed as to what the photo actually represents.

The section that begins on the bottom of page 73 and continues into page 75 presents a series of non-sequiturs that do not fulfill the expectation of the condor's "unique" susceptibility to lead ammunition. I suggest a tightening of that section.

Unless I missed it, the authors might also somewhere explain that the most important contributor to condor population vulnerability is the condor's protracted maturation, its very slow rate of reproduction, and its consequent high sensitivity to mortality among the older age classes. This is not to say that other characteristics aren't also predisposing, but the very low natural demographic potential of condor populations is the most powerful issue in their vulnerability to lead, indiscriminate, as it is, with respect to condor age.

Regarding the ^{207}Pb:^{206}Pb ratios (page 54 and 55) in ammunition lead on which Church et al. (2006) based their conclusions, subsequent analysis of a larger sample of bullets (showing far wider ratios) undermine their conclusions. The authors cite Finkelstein et al. 2012 when mentioning the wider ratios ("0.78-0.87"), but I cannot find that information in the PNAS paper or in its supplementary information. The authors do not directly speak of the implications of that uncertainty, although in the Discussions Points (page 58) they quite appropriately avoid placing any weight upon the idea that Pb ratios provide an ammunition signature. No complaints here, only that I cannot find the referenced data in the citation.

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APPENDIX C: REVIEWER 2 EDITED VERSION OF DRAFT REPORT

Lead Exposure to Scavenging Birds: A Review for of the U.S. Fish and Wildlife Service by the Environmental Contaminants Program

July 2, 2013

Submitted by:

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Executive Summary

Birds are exposed to lead in the environment from multiple sources. Specifically, exposure to lead ammunition has resulted in harmful effects to migratory birds and endangered species for over a century. The U.S. Fish and Wildlife Service recognized the problem of avian exposure to lead shot used for waterfowl hunting and enacted restrictions in 1991. However, the use of lead ammunition continues for other forms of hunting, presenting an ongoing risk to upland or terrestrial migratory birds that ingest spent shot directly from the ground or as a result of predating or scavenging carcasses that have been killed with lead ammunition and left in the field. This white paper addresses the effects of lead ammunition on scavenging birds by reviewing the current scientific literature including areas of potential scientific uncertainty.

Lead has been recognized as a contaminant for centuries and its toxicity to birds is extremely well understood from study in a variety of species. Ingested lead, including metallic lead in the form of ammunition, is soluble in the digestive system and absorbed into tissues. This exposure results in specific physiological responses that are measurable, but may vary among individuals and species. While the progression of lead poisoning ultimately results in mortality, sublethal conditions may render a bird more susceptible to death from other causes. General thresholds in tissues of birds have been established to help determine when lead exposure has occurred and when it has led to poisoning. However, due to differing sensitivity of individuals and species, the diagnosis of lead poisoning is best achieved through a combination of tissue thresholds and clinical signs.

With an annual mortality rate of at least 2%, exposure to lead shot from hunting resulted in effects to waterfowl at the population level. Secondary poisoning to scavengers, including the bald eagle (*Haliaeetus leucocephalus*), resulted from consumption of waterfowl containing embedded or ingested lead shot. The lead shot ban enacted in the United States in 1991 helped conserve North American waterfowl populations and reduce exposure to birds feeding on waterfowl carcasses contaminated with lead. The nontoxic shot approval process developed in

conjunction with this ban has resulted in the approval of suitable alternatives to lead that present limited environmental threats.

Lead ammunition continues to be in use for upland game hunting, a popular and widespread activity with millions of participants per year. Lead pellets or fragments can remain on the landscape from these activities in prey that is wounded or killed by hunters but not retrieved, or from offal piles remaining from kills. In the United States, these sources may number in the millions per year from deer hunting alone. Carcasses and offal with lead ammunition fragments are an attractive and available food source to scavenging birds.

An individual's vulnerability to lead ammunition is based on its exposure potential and sensitivity. Some scavenging birds feed on carcasses exclusively, and others as part of a broader diet. Obligate scavengers may have an increased risk of poisoning from lead ammunition due to a potentially higher frequency of exposure. The tendency of scavengers to exhibit group feeding behavior can also enhance their vulnerability to lead exposure.

Bioavailability is the extent to which a contaminant is free for uptake into an organism. While ammunition pellets are small enough to be ingested by most birds, an intact bullet is generally too large. For this reason, the bioavailability of lead from carcasses and offal left by hunters is greatly enhanced by the tendency of lead bullets to fragment into small pieces, migrate from wound channels, avoid detection or regurgitation by scavengers, and be abundant enough to expose several scavengers feeding on a single carcass. Regardless of the type of game or bullet, all lead bullets that have been studied fragment, sometimes substantially (i.e., hundreds of fragments per carcass), when fired into an animal. Lead was frequently confirmed as the fragmenting metal (as opposed to another metal from the base or jacket of the bullet) by recovery and dissection of fragments or lead analysis of surrounding tissue. The use of non-expanding bullets may reduce fragmentation, but do not perform as such consistently in controlled studies. In contrast, monolithic copper bullets consistently hold together and produce a low number of fragments, if any, within carcasses. Non-fragmentation of bullets significantly reduces their bioavailability to predators and scavengers.

Carnivorous birds contain physiological adaptations that facilitate the absorption of metallic lead into tissues. The solubility of ingested metallic lead in scavenging birds has been demonstrated in lab studies by increased blood concentrations and reduced enzyme activity following treatment. Scavenging birds may retain shot in the gastrointestinal tract, though the absence of shot or fragments may indicate either a lack of exposure, or regurgitation or complete absorption following ingestion. Like other avian species, tissue residues of lead-exposed and lead-poisoned scavenging birds can vary and should be interpreted in conjunction with other diagnostic signs. Clinical signs diagnostic of lead poisoning may not always be exhibited by exposed species or individuals, despite known toxicosis.

Lead exposure has been documented in numerous species of predatory and scavenging birds, and in many regions of North America. These known cases likely represent a much larger number of affected individuals. Some documented cases of elevated lead exposure include evidence suggestive of exposure to lead ammunition. Some show an increase of exposure and mortality rates during hunting seasons. Other cases include recovery of lead fragments or pellets from birds, their presence on radiographs, or observation of exposed birds feeding on a contaminated carcass. While these types of evidence are not always available or sought by those investigating cases of lead poisoning, numerous examples exist in the literature that help to confirm this exposure pathway. Likewise, the absence of these observations does not negate the possibility of ammunition as the source of exposure.

When used with other lines of evidence such as observations, behavioral ecology, or recovery of ingested items, isotopic ratios can provide another indication of the source of lead for an exposed individual. For example, isotopic ratios in blood of exposed birds and a known source of exposure (e.g. bullet fragment or paint chip removed from bird) will be highly similar and can confirm that lead was absorbed from that source into the bird's tissue, and where applicable, resulted in poisoning. Changes in isotopic ratios in avian tissues can reveal when a novel source of lead has been introduced into the diet. A noteworthy example is the decline in ^{207}Pb:^{206}Pb ratios associated with increased lead exposure as California condors (*Gymnogyps californianus*)

transition from captivity to the wild. Serially sampling of feathers can provide a more refined view of lead exposure in an individual over time. When interpreted with caution, adult or fully grown feathers can be useful tools for analyzing changes in lead exposure via the coupling of lead concentrations and isotopic ratios.

Numerous sources of lead contribute to its availability on the landscape, including lead-based paints, mining operations, and shooting ranges. These routes cannot be ruled out as potential sources of lead exposure. However, the foraging and behavioral traits of scavenging birds make these sources less likely and there are few documented cases of lead poisoning from alternate exposure routes as compared to lead ammunition.

Two populations of birds have been shown to be particularly affected by exposure to lead ammunition: bald eagles in the Great Lakes and California condors throughout their range. For bald eagles, a significant portion of the continental population as well as birds that have migrated from Canada are competing for food in the Great Lakes in the winter. Scavenging on deer offal is common for wintering bald eagles, and data from wildlife rehabilitation facilities and wildlife diagnostic laboratories confirm that lead exposure makes up a significant portion of moribund and dead eagles annually. Lead poisoning alone may account for over 25% of the annual maximum sustainable yield for the Great Lakes population.

California condors feed exclusively on carcasses including those which have been killed by hunters. Lead exposure to other sources cannot be ruled out, however, the diet and foraging behavior of this species makes it particularly vulnerable to exposure to ammunition as a source of lead. Numerous mortalities have been attributed to lead poisoning, and rates may be even higher without current levels of intervention (monitoring and chelation therapy). In California, over the period between 1997 and 2010, 48% of the free flying birds had blood lead concentrations over the lead poisoning threshold, a number of which were treated and released. Measures to reduce the amount of lead ammunition used have likely contributed to a decline in lead availability, but lead ammunition may still be present in the range of the condor from upland

game hunting, nuisance animal depredation, and dispatching domestic livestock, as well as poaching, and non-compliance of lead ban regulations.

One way to break the exposure pathway of scavenging birds to lead ammunition is via the use of alternative metals, provided that ammunition derived from these metals is. A comprehensive testing method exists in the United States to ensure that alternative metals used to hunt waterfowl do not cause sickness and death when ingested by migratory birds. These data can be extrapolated to ammunition used for other forms of hunting. The nontoxic shot approval process has resulted in the approval of suitable alternatives to lead that present limited environmental threats. The types of nontoxic metals available and approved for use offer the public many options. After the 1991 lead ban, steel became the number one alternative to lead and is still widely used. Copper, the primary alternative currently used in bullets, exhibits low toxicity to birds in its metallic form.

Introduction

Lead is a naturally occurring and highly toxic element that has no known biological function. Poisoning has been documented in humans for at least 2500 years and in waterfowl from spent lead shot for over 100 years (Eisler 2000). In that time, the ecotoxicological properties of lead have been extensively reviewed (Eisler 2000). Today lead is primarily used in the manufacture of storage batteries, alloys, pigments and chemicals, and in ammunition. Present-day lead concentrations in air, soil, and water near inhabited areas are estimated to be from 2 to 10,000 times naturally occurring levels (Pain 1995). Atmospheric lead can also be derived from fossil fuel combustion, lead mining and smelting, and manufacturing. These sources of lead are especially significant as a local threat.

Wildlife can be exposed to lead from numerous sources, including mining and smelter emissions, lead-based paint, fishing sinkers, and ammunition. Effects to birds reached the population level when poisoning resulting from hunting was estimated to result in the mortality of at least 2% of all waterfowl (~2 million ducks and geese) annually exposed by ingestion of spent shot from sediments (Bellrose 1951). Later, effects to bald eagles (*Haliaeetus leucocephalus*) preying upon

exposed waterfowl were documented (Griffin et al. 1980, Pattee and Hennes 1983). To alleviate this problem, the U.S. Fish and Wildlife Service (USFWS) instituted a nationwide restriction on the use of lead shot for hunting waterfowl and American coots (*Fulica americana)* in 1991 (USFWS 1986, 1995). While the ban of lead for this use resulted in a reduction of exposure in waterfowl (Anderson et al. 2000), lead shotgun pellets and rifle bullets are still widely used for hunting upland and large game animals and lead poisoning from the ingestion of lead ammunition and fragments thereof persists in some groups of avian species.

The likelihood of lead exposure to ammunition in birds varies according to feeding habits and the use of grit. At greatest risk are waterbirds that feed on items in wetland sediments where lead shot has accumulated, species that ingest grit to aid in the grinding of food in the ventriculus, birds that feed on seeds in heavily hunted upland areas, and avian scavengers that consume carcasses or offal (gut piles) of animals shot with lead ammunition and left in the field. In addition to many species of waterfowl, lead exposure and poisoning has been reported in a variety raptors, terrestrial species, and aquatic non-waterfowl species in the United States including those protected under the Migratory Bird treaty Act, the Endangered Species Act, and the Bald and Golden Eagle Protection Act (Bellrose 1959, Pain et al. 2009).

Objectives

The objectives of this paper are to gather and review current science on exposure and effects to wildlife from lead ammunition, and to address questions regarding scientific uncertainty. While general toxicity of lead will be reviewed, the specific focus will be scientific literature regarding effects to scavenging birds from secondary poisoning.

Discussion Points: Introduction and Objectives

- Birds may be exposed to lead in the environment from multiple sources.
- Exposure to lead ammunition has resulted in adverse effects to migratory birds and endangered species for over a century.
- The U.S. Fish and Wildlife Service recognized the problem of avian exposure to lead shot

used for waterfowl hunting and enacted restrictions in 1991.

- The use of lead ammunition continues for other forms of hunting.
- Birds can ingest spent shot directly from the ground or as a result of predating or scavenging carcasses that have been killed with lead ammunition and left in the field.
- This white paper addresses the effects of lead ammunition on scavenging birds by reviewing the current scientific literature including areas of potential scientific uncertainty.

Toxicological Impacts of Lead in Birds

In North America, mortality of birds due to lead poisoning from the ingestion of lead shotgun pellets was first reported in waterfowl in 1894 in Texas and North Carolina, and by the 1950s an estimated 2-3% (1.6-2.4 million) waterfowl across all North American flyways were dying annually of lead shot poisoning (Grinnell 1894, Hough 1894, Bellrose 1959). The early accounts by Grinnell (1894) and Hough (1894) include the first descriptions of gross toxicological effects of lead poisoning in wild birds in the United States. Wetmore (1919) reviewed clinical signs and lesions of lead poisoning in waterfowl ~~to date~~ and reported the results of an experimental study of lead shot poisoning in ducks. The study showed that mortality varied in mallards (*Anas platyrhynchos*) dosed with one to three #6 lead shot, but six #6 shot pellets were always fatal. Similar findings were noted in northern pintails (*Anas acuta*) and redheads (*Aythya americana*) (Wetmore 1919). Shillinger and Cottam (1937) reported that the frequency of lead shot ingestion in several thousand gizzards from various species of ducks ranged from 1-39% and suggested that lead poisoning may be an important factor in the decline of waterfowl populations. In an early study of a variety of waterbird species, lead poisoning was the third largest cause of mortality noted in 3,000 carcasses and the authors commented on the frequency of poikilocytosis (abnormal shape), anisocytosis (unequal size), and reduced hemoglobin content of red blood cells (Quortrup and Shillinger 1941).

Organism health response and effects

Lead has no known beneficial role in biological systems and is a systemic toxin that affects all body systems. The first measureable physiological effect is the inhibition of delta-aminolevulinic acid dehydratase (ALAD), an enzyme necessary for hemoglobin synthesis and a very sensitive indicator of lead exposure in birds (Finley et al. 1976). Birds can tolerate considerable reductions in ALAD activity without adverse hematological effects, but ALAD depression by high levels of lead exposure results in anemia characterized by lowered hemoglobin and hematocrit (Franson et al. 1983, Pain and Rattner 1988). Lead also inhibits ferrochelatase (heme synthetase), an enzyme responsible for combining ferrous iron and protoporphyrin IX (PPIX) to form heme. Inhibition of ferrochelatase results in the accumulation of PPIX in the erythrocytes and its quantification in blood samples has been used as an indicator of lead exposure in birds (Roscoe et al. 1979, Franson et al. 1996). Additional clinical signs of lead poisoning vary somewhat among species groups, with waterfowl exhibiting perhaps the most, including submandibular edema, lethargy, wing droop, ataxia, anorexia, green bile staining of the vent, leg paralysis, and convulsions (Figure 1) (Locke and Thomas 1996, Rattner et al. 2008, Franson and Pain 2011). In a study of lead acetate poisoning in six captive avian species, the most consistent clinical signs across all taxa were weight loss, anemia, and increased concentrations of PPIX (Beyer et al. 1988). Similarly, bald eagles dosed with lead shot lost weight and had reduced hematocrit, hemoglobin, and ALAD activity, as well as changes in serum biochemistries (Hoffman et al. 1981, Pattee et al. 1981).

Figure 1. Bald eagle (*Haliaeetus leucocephalus*) at rehabilitation center displaying clinical signs of lead poisoning.

Photo courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

Birds that die within a few days from an acute exposure to a large concentration or dose of lead may be in good flesh. However, lead poisoning is typically a chronic disease resulting in anorexia, loss of fat reserves, muscle wasting, and debilitation (Locke and Thomas 1996). Time to death in experimental studies varies by species and dosage regimen, with waterfowl generally succumbing within 2-4 weeks, although some raptors survived for more than 15 weeks (Barrett and Karstad 1971, Pattee et al. 1981, Franson et al. 1986, Beyer et al. 1998, Pattee et al. 2006). Other gross lesions include impaction of the esophagus, proventriculus, and ventriculus with food (particularly in waterfowl), bile staining of the ventriculus and intestinal contents, distension of the gall bladder with dark green viscous bile, necrosis evidenced by light streaks on

the surface of the heart or the cut surface of the gizzard muscle, pale and atrophied internal organs, and flabby heart (Locke and Thomas 1996, Rattner et al. 2008, Franson and Pain 2011). In a study of 421 lead poisoned waterfowl of various species, the most reliable gross indications of lead poisoning were reported to be impactions of the alimentary tract, submandibular edema, necrosis of heart muscle, and bile staining of the liver (Beyer et al. 1998).

Locke et al. (1966) were the first to report inclusion bodies in histologic sections of kidney tissue of lead poisoned birds, in that case mallards. These structures occur within the nuclei of cells in the proximal convoluted tubules of the kidney, and when stained with the Ziehl-Neelson acid-fast technique appear scarlet in color. Other gross and microscopic lesions of lead poisoning are nonspecific and may be observed in association with other conditions, but only lead exposure is known to produce acid-fast intranuclear inclusion bodies in the kidneys of birds. However, although the inclusion bodies are indicative of lead poisoning, they are not present in all cases. Thus, renal inclusions occurred in 64% of lead poisoned red-winged blackbirds (*Agelaius phoeniceus*), 69% of brown-headed cowbirds (*Molothrus ater*), 75% of mallards, 86% of northern bobwhites (*Colinus virginianus*), and 100% of common grackles (*Quiscalus quiscula*) and eastern screech owls (*Otus asio*) that died from lead acetate poisoning (Beyer et al. 1988). Intranuclear inclusion bodies were not found in a study of lead poisoned bald eagles and have been infrequently reported in poisoned Canada geese (*Branta Canadensis)* (Bagley et al. 1967, Locke et al. 1967, Barrett and Karstad 1971, Pattee et al. 1981, Sileo et al. 2001). Renal inclusions have been reported in several other species of birds poisoned by lead, including mourning doves (*Zenaida macroura*), rock doves (*Columba livia*), mute swans (*Cygnus olor*), whooper swans (*Cygnus cygnus*), Andean condors (*Vultur gryphus*), turkey vultures (*Cathartes aura*), and white-tailed eagle (*Haliaeetus albicilla*) (Locke and Bagley 1967, Simpson et al. 1979, DeMent et al. 1987, Ochiai et al. 1992, Kenntner et al. 2001, Carpenter et al. 2003, Pattee et al. 2006). Additional histopathologic lesions noted in lead poisoned birds include hepatic hemosiderosis, renal tubular cell degeneration, myocardial and gizzard muscle necrosis, fibinoid necrosis of arterioles, erythroid hyperplasia, encephalopathy, and peripheral neuropathy (Locke and Thomas 1996, Wobeser 1997, Franson and Pain 2011).

Solubility and distribution

When metallic lead is ingested by birds, the stomach's acid and grinding action in species with a muscular gizzard begin to dissolve it, resulting in the formation of toxic lead salts. As these salts are absorbed in the intestinal tract, lead enters the bloodstream and measurable increases in blood lead concentrations occur within hours (Roscoe et al. 1979). Lead is distributed throughout the body, including growing feathers, via the circulatory system and a dynamic equilibrium controls deposition and removal in various tissues. Franson and Pain (2011) reviewed the distribution of lead in avian tissues and factors influencing the concentrations of lead in tissues. In general, the highest concentrations are found in bone, liver, and kidney, with intermediate concentrations in brain and blood, and low concentrations in muscle. In birds that survive lead exposure, lead concentrations in soft tissues will decline over time. Lead shot dosing studies with mallards and canvasbacks (*Aythya valisineria*) have shown that blood lead and PPIX concentrations may remain elevated, and ALAD activity may remain depressed, for several weeks to as long as three months (Finley and Dieter 1978, Roscoe et al. 1979, Franson et al. 1986). Because lead is released from bone far more slowly than from soft tissues, bone functions as a long term repository. Bone lead concentrations may also differ between males and females and among females, depending on season. Finley and Dieter (1978) reported that lead concentrations in femurs of laying mallards were four times higher in laying than nonlaying females. As calcium is mobilized from bone for eggshell formation, intestinal absorption of calcium, and with it lead, increases (Krementz and Ankney 1995, Scheuhammer 1996).

Diagnosis of lead poisoning and effects thresholds

In addition to history information, clinical signs, and gross and microscopic lesions, concentrations of lead in tissues have been used to evaluate lead exposure and poisoning in birds. Concentrations of lead in blood of live birds and in liver and kidney of dead birds are the tissues commonly used to assess lead exposure. Concentrations in bone are not a good reflection of recent lead exposure and are somewhat difficult to interpret because of continual accumulation and slow release. The use of feathers for evaluating lead exposure, in live or dead birds, is based on the premise that metals are deposited during the period of feather growth, reflecting circulating

blood lead concentrations at the time of feather formation. A variety of factors, including feather type, timing of molt and exposure, and variations among feather parts can affect the interpretation of metals in feathers, and consideration should be given to the possibility that lead deposition from the atmosphere may result in surface contamination (Burger 1993, Pain et al. 2005). However, an experimental study with black-crowned night heron (*Nycticorax nycticorax*) nestlings showed that lead concentrations in feathers were a good indicator of exposure and were correlated with lead concentrations in other tissues (Golden et al. 2003). The authors discussed similar findings in juvenile birds of several other species and commented that adult feathers appear to be a less reliable indicator of lead exposure. Eggs are not known to be particularly useful for monitoring lead exposure in birds. A laboratory experiment with Japanese quail (*Coturnix coturnix*) showed that, although some lead is transferred to eggs, concentrations were much lower than in the diet (Leonzio and Massi 1989). Field studies with common eiders (*Somateria mollissima*) failed to detect correlations between lead concentrations in eggs and concentrations in feathers or blood of laying females (Grand et al. 2002, Burger et al. 2008).

Lead concentrations in birds with no history of lead exposure are typically <0.2 ppm in blood, <2 ppm wet weight in liver and kidney, and <10 ppm dry weight in bone (Table 1; Franson and Pain 2011). Some orders appear to be more resistant to lead poisoning that others, as indicated by higher lead concentrations in tissues reported in association with lead poisoning. However, suggested guidelines are that lead concentrations >0.5 ppm in blood or >6 ppm wet weight in liver and kidney are evidence of clinical poisoning, and would likely be accompanied by signs such as anemia, weight loss, and muscular incoordination (Franson and Pain 2011). As with all wildlife, birds suspected to have died of lead poisoning should be subjected to a complete necropsy examination. Although interpretive guidelines are available, lead poisoning as a cause of death should be distinguished from simple lead exposure based solely on tissue residues. Ideally, a determination of lead poisoning as cause of mortality should be based on an evaluation of field circumstances, any observed clinical signs, gross lesions and pathological findings, tissue residues in excess of 6 ppm wet weight in liver or kidney, and when possible other laboratory testing to rule out other contaminants, and infectious or parasitic diseases. If observations of clinical signs and necropsy findings are not available, a more conservative approach is

warranted. In a study of lead poisoned waterfowl, Beyer et al. (1998) reported that 95% of birds had liver lead concentrations of at least 38 ppm dry weight (10 ppm dry weight), but fewer than 1% of birds that died of other causes had a concentration that high. The authors concluded that 38 ppm dry weight lead in liver is a defensible criterion for identifying lead poisoning in waterfowl in the absence of pathologic observations.

Table 1. Thresholds for Lead Toxicosis. Note that all tissue concentrations herein have been converted from their original unit to parts per million, ppm, for ease of comparison across studies and against diagnostic thresholds. For measurements taken for blood, the margin of error can be up to 4% as a result of converting a volumetric measurement to standard SI units. Thresholds from Franson and Pain (2011).

Tissue	**Background**	**Subclinical**	**Clinical**
Blood (ppm, wet weight)	<0.2	0.2-0.5	> 0.5
Liver (ppm, wet weight)	<2.0	2.0-6.0	> 6.0
Kidney (ppm, dry weight)	<2.0	2.0-6.0	> 6.0

Sublethal effects and mortality

When evaluating the adverse effects of lead exposure in individuals and populations, one should consider potential sublethal effects that could impair birds, in addition to the alterations in enzyme activities and hematology mentioned earlier. Some sublethal effects impact health directly and some may render birds more susceptible to predation, hunting, collisions with objects, and disease. In particular, lead exposure may affect growth, body and organ mass, behavior, neurological and physiological parameters, feeding activity, and reproduction. For example, Burger et al. (1994) discussed a series of studies of lead exposure in young common terns (*Sterna hirundo*) and herring gulls (*Larus argentatus*) that showed a variety of abnormalities, including decreased growth, fledging size, locomotion and balance ability, and decreased feeding activity. Reduced brain weight has been associated with lead exposure in young mallards, American kestrels (*Falco sparverius*), and European starlings (*Sturnus vulgaris*)

(Hoffman et al. 1985, Grue et al. 1986, Douglas-Stroebel et al. 2004). Mixed results have been reported in studies of lead impacts on immune response. Antibody-mediated immunity was suppressed in Japanese quail and mallards exposed to lead at levels resulting in other clinical signs (Trust et al. 1990, Rocke and Samuel 1991, Grasman and Scanlon 1995). However, lead ingestion at levels that impaired growth and hematology in Japanese quail did not affect humoral immune reponse (Morgan et al. 1975). No evidence of immunotoxicity was reported with low-level exposure to lead in Japanese quail and red-tailed hawks (*Buteo jamaicensis*) and, in fact, a hormetic effect of increased disease resistance was found in the quail study (Redig et al. 1991, Nain and Smits 2011). Effects of lead exposure on reproduction include lower fertilization rate in ring-necked pheasants (*Phasianus colchicus*), lower egg production in Japanese quail, reduced hatchability in ring-necked pheasants and mourning doves, and smaller clutches and increased nestling mortality in pied flycatcher (*Ficedula hypoleuca*) (Edens et al. 1976, Buerger et al. 1986, Berglund et al. 2010, Gasparik et al. 2012). Testicular changes have been noted in chickens (*Gallus gallus domesticus*), Japanese quail, and ringed turtle-doves (*Streptopelia risoria*) (Morgan et al. 1975, Veit et al. 1983, Mazliah et al. 1989). Gangoso et al. (2009) reported decreased mineralization in bones of Egyptian vultures (*Neophron percnopterus*) with increasing lead concentration. Bellrose (1959) reported that mallards dosed with lead shot and released were 1.5 times more vulnerable to hunting than controls. Mute swans that collided with power lines, cables, or other objects had elevated lead concentrations in tissues (O'Halloran et al. 1989, Kelly and Kelly 2005).

Discussion Points: Toxicological Impacts of Lead on Birds

- Lead toxicity to birds is extremely well understood and has been studied in a variety of species.
- Ingested lead ammunition is soluble and absorbed in tissues.
- The progression of lead poisoning ultimately results in mortality.
- Sublethal conditions may render a bird more susceptible to other causes of mortality.
- Lead exposure results in specific physiological responses that are measurable, but may vary among individuals and species.

- General thresholds have been established in avian species to help diagnosis lead exposure and poisoning. However, diagnosis of lead poisoning is best achieved through a combination of tissue thresholds and clinical signs due to the differing sensitivity of individuals and species.

Review of the Lead Ban for Waterfowl

The 1991 United States lead shot regulations

Studies conducted in the 1960's were among the first to confirm toxicity of lead to waterfowl and to determine clinical signs such as impairments to normal biological functions, severe weight loss, and mortality (Bellrose 1965, Irby et al. 1967, Locke et al. 1967). Irby et al. (1967) and Locke et al. (1967) examined the effects of lead to mallards dosed with three types of commonly used shot: lead, plastic-coated lead, and lead-magnesium. The study found mortality to 96% of the mallards dosed with lead, 93% dosed with plastic-coated shot, and 58% dosed with lead-magnesium (Irby et al. 1967). The lead dosed mallards developed anemia, atrophy of adipose and liver tissue, and enlarged gall bladders distended with bile, as well as cellular effects such as hemosiderosis and destruction of the kidney tubule cells, acid-fast intranuclear inclusion bodies, and enlarged nuclei in the cells of the proximal convoluted tubules (Locke et al. 1967). These early studies provided the initial data on the susceptibility of waterfowl to lead poisoning that sparked a progression for more studies. As lab toxicity data became more available on the adverse effects of lead to waterfowl, studies investigating population loss were initiated.

In the early 1980's linkages between bald eagle deaths and lead shot used in waterfowl hunting were on the rise. At that time, the bald eagle was listed as endangered under the Endangered Species Act. A number of cases determined that bald eagles were exposed to lead by feeding on crippled or non-retrieved hunter shot waterfowl containing ingested or embedded shot (Griffin et al. 1980, Pattee and Hennes 1983). Between 1967 and 1982, an estimated 7% of the bald eagle population in the U.S. was lead poisoned (Pattee and Hennes 1983). In 1986, the USFWS required lead shot in hunting waterfowl and American coots to be phased-out over a 5-year

timeframe initiated during the 1987-1988 hunting season (USFWS 1986, 1995). The regulation became a nationwide restriction in 1991 and the USFWS started developing an approval process for nontoxic shot. Canada soon followed with a similar federal regulation in 1999 (Rattner et al. 2008). Table 2 provides a historical timeline of the events that led up to the 1991 lead shot ban for waterfowl hunting in the U.S.

The USFWS defined that word "nontoxic" as shot that does not cause sickness and death when ingested by migratory birds and developed testing guidelines to develop and approve new types of shot as nontoxic that currently remain in place (USFWS 2013a, b). More information regarding these testing protocols and the outcome of their use is provided in the "Toxicity of alternative metals used in ammunition" section below.

Table 2. Milestones in the transition to nontoxic shot use for waterfowl in the United States, Friend et al. (2009).

Year	Discovery	Concern	Regression	First Actions
1874	Anecdotal mortality reported			
1894	First documented mortality			
1915	Numerous shot found in swan gizzards			
1916	Numerous shot found in sediments near duck blinds			
1919	First lead toxicity study in wild ducks			
1930		Scientists report widespread lead poisoning		

1936		Nontoxic shot development pursued		
1937	First broad-scale evaluation of shot ingestion by waterfowl			
Early 1940s		Lead poisoning reported to be great concern for ducks		
1948		Olin Corporation initiates quest for nontoxic shot		
Early 1950s		Expanded concerns and investigations		
Mid 1950s			Habitat conditions restore duck populations; interest in lead poisoning wanes; nontoxic shot stops development	
1959	Bellrose report on lead as a waterfowl mortality factor			
Early 1960s		Major waterfowl populations decline; interest in lead poisoning heightened		
1965				First field test of nontoxic shot. Flyway Council urges development of nontoxic shot
1972				First nontoxic shot use requirements
1974-1976				USFWS proposes use of nontoxic shot nationwide

1976		First lawsuit opposing nontoxic shot regulations		
1978		USFWS prevented from initiating or enforcing nontoxic shot requirements without State approval		
Early 1980s	Documentation of numerous lead poisoning cases in bald eagles	Lawsuit filed against state and federal nontoxic shot regulations	Government prevails	
Late 1980s		Lawsuit filed against USFWS to prevent nontoxic shot in California		
1991			Nontoxic shot required nationwide for waterfowl hunting	

Effectiveness of lead shot ban

The effectiveness of the 1991 regulations that banned lead for waterfowl hunting in the U.S. in lieu of this new nontoxic shot has been evaluated by studying lead shot ingestion rates in areas where the ban was in effect. Five years after the ban, a study of 16,651 mallards along the Mississippi Flyway estimated that mortality from lead shot exposure declined 64%, with 1.4 million of 90 million ducks in the 1997 fall flight spared from poisoning (Anderson et al. 2000). Although the study indicated a similar overall rate of pellet ingestion for these birds (8.9% post-ban vs 8% pre-ban; Bellrose 1959, Sanderson and Bellrose 1986), 2/3 of the pellets were nontoxic. Samuel et al. (1992, 2000) investigated the effectiveness by studying blood lead concentrations in American black ducks (*Anas rubripes*) in Tennessee before (1986-1988) and after (1997-1999) the ban. Using blood lead concentrations of ≥0.2 ppm as an indication of lead exposure, Samuel and Bowers (2000) reported pre- and post-ban exposure rates of 11.7% and 6.5%, respectively, in adult black ducks. However, no difference in the exposure rate was detected before and after the implementation of nontoxic shot regulations in juveniles, possibly

resulting from differences in food habits or habitat use. In mottled ducks (*Anas fulvigula*) collected in Texas during the 1987-2002 hunting seasons, lead shot ingestion rates were 7% along the upper Texas coast and 18% in those collected from the central Texas coast (Merendino et al. 2013). This compares with an ingestion rate of about 32% in mottled ducks collected along the gulf coast of Texas from 1973-1975, before nontoxic regulations were implemented in the early 1980s (Moulton et al. 1988). A study in Canada reported that bone lead concentrations in ducks declined by about 50% after the 1997 ban on lead shot for waterfowl hunting took place (Stevenson et al. 2005).

As revealed in these investigations, the lead shot ban was effective at reducing but not eliminating lead exposure in waterfowl and birds that prey upon waterfowl. Lead shot discharged prior to the ban remains on the landscape and can continue to be ingested by waterfowl unless removed from the environment. Wayland and Bollinger (1999) sampled eagles from 1990-1997 from the Canadian prairie provinces after the U.S. and Canada lead shot bans were in place. The study found greater lead exposure in deceased bald eagles collected from areas of high waterfowl hunting intensity compared to eagles in areas of low waterfowl intensity. Another study investigated lead exposure to trumpeter swans (*Cygnus buccinator*) in the upper Midwest and found 25% of swan mortality from 1991-2007 was attributed to lead toxicity (Strom et al. 2009).

Discussion Points: Review of The Lead Ban for Waterfowl

- Exposure to lead shot affected waterfowl populations
- Secondary poisoning to scavengers, including the bald eagle, resulted from consumption of waterfowl containing embedded or ingested lead shot.
- The switch to nontoxic shot resulted in lower rates of lead shot ingestion in waterfowl.
- The lead shot ban helped conserve North American waterfowl populations and reduce exposure to birds feeding on waterfowl carcasses contaminated with lead.
- Even after the lead shot ban, pellets remaining in the environment still have the potential to expose waterfowl and predatory birds.

Sources of Lead Ammunition Remaining After the Ban

Despite the effectiveness of the ban on the use of lead shot in waterfowl hunting, this restriction addressed only a subset of all hunting and shooting activities. The U.S. has a long history of using firearms to hunt game (Dunlap 1988). This history dates back to European settlement and subsistence hunting to modern sport hunting. Sportsman and hunting ethics have contributed to the social construct of natural resource conservation in the U.S. and funds generated by the sales of ammunition under the Wildlife and Sport Fish Restoration Program help fund conservation projects (Peterson 2004, USFWS 2013c).

Modern firearms used for hunting in the U.S. discharge a variety of ammunition that includes projectiles of various sizes such as clusters of pellets, rifle bullets, and shotgun slugs (Thomas 2013). The ammunition is designed to transfer energy from the projectile on the target to maximize the power to mortally wound the game. This is often accomplished by the lead projectile fragmenting into smaller pieces and scattering along the trajectory pathway into the carcass (Grund et al. 2010). Historically, these projectiles were composed mostly of lead, but more recently copper and other non-lead alternative projectiles have been introduced into the ammunition market (Thomas 2013). Despite studies that indicate that the ballistic performance of projectiles such as copper may be better than or equal to lead (Knot et al. 2009; Batha and Lehman 2011), the use of non-lead projectiles is very new, more expensive, and not widely accepted by hunters (Thomas 2013).

Deer hunting is the most popular type of hunting in the U.S., with 10.1 million participants nationwide in 2006 (USFWS 2006). At least 3.8 million deer were harvested by resident hunters and 0.7 million deer harvested by non-resident hunters in 2006 (USFWS 2006). Each harvested deer results in an offal pile that may be accessed by scavenging birds. In addition, studies have revealed that a significant number of deer are shot but not retrieved by hunters. Though exact wounding rates are difficult to enumerate and are likely to vary across regions, attempts at estimations have produced similar results: 21 - 24% in Illinois, 24% in Montana, and 17-32% in Indiana (Stormer et al. 1979, Dusek et al. 1989, Nixon et al. 2001).

There is also substantial participation in other types of hunting. Small game such as rabbit, squirrel, pheasant, quail, and dove comprised the next largest group after deer with about 7.5 million hunters (USFWS 2006). Turkey hunting is the third most popular category at about 2.6 million participants. Waterfowl hunting accounts for 1.8 million hunters, though no longer contributes to lead in the environment since its use was banned for this purpose (USFWS 2013d). Hunting for varmint (coyote, raccoon, fox) and other game (e.g., upland birds and mammals) also takes place but is less popular (USFWS 2006).

Discussion Points: Sources of Lead Ammunition Remaining After the Ban

- Game hunting is a popular and widespread activity with millions of participants per year in which lead ammunition is still used.
- Lead pellets or fragments remain on the landscape in prey that is wounded or killed by hunters but not retrieved, or from offal piles remaining from kills. These sources may number in the millions per year from deer hunting alone.
- Carcasses and offal with lead ammunition fragments are available and attractive to scavenging birds.

Avian Scavengers at Risk

The vulnerability of an individual of a species to a toxicant is based on its exposure potential and sensitivity (Golden and Rattner 2003).

Exposure potential

The potential for a bird to be exposed to lead ammunition is a function of multiple factors, including its diet, foraging strategy, and the frequency that lead will be encountered in the environment. Birds that scavenge on carcasses or offal piles are especially susceptible to exposure to ammunition in the tissues of hunter-killed game. Scavengers can feed on carcasses, thus removing them from the environment, with efficiency. Removal rates have been documented as high as 98% after 24 hours in controlled studies, but can vary widely, influenced

by factors such as season, location, and species (Prosser et al. 2008). Other forms of direct exposure that are unlikely to pose significant risk to scavenging birds include direct ingestion of spent shot from the ground, or predation of game species containing embedded ammunition.

Influence of diet on exposure

Birds can either be obligate scavengers, meaning they feed exclusively (or almost exclusively) on carrion, or combine scavenging with predation on live prey or other food items. Obligate North American avian scavengers include the California condor (*Gymnogyps californianus*), black vulture (*Coragyps atratus*), and turkey vulture. Specific dietary items are dependent on geographic location, but condors and black vultures rely heavily on large mammals, while turkey vultures are more adaptable and forage on smaller and more varied food items (Kirk and Mossman 1998, Buckley 1999, USFWS 2013e). Other avian species such as bald eagles, golden eagles (*Aquila chrysaetos*), caracaras (*Caracara cheriway*), crows (*Corvus brachyrhynchos*), and ravens (*Corvus corax*) consume carrion as part of a broader diet (Boarman and Heinrich 1999, Buehler 2000, Kochert et al. 2002, Verbeek and Caffrey 2002, Morrison and Dwyer 2012). These species tend to be opportunistic, and like obligate scavengers, diets vary with geography, habitat, and availability. For some species, such as bald eagles, adults may be more apt than immatures to capture live prey due to better developed foraging ability (Buehler 2000). Both species of eagles consume a greater percentage of carrion in their diets during winter months (Buehler 2000, Kochert et al 2002). Crows and ravens consume the widest variety of food, including plant material, insects, garbage, eggs, small animals, and carcasses (Boarman and Heinrich 1999, Verbeek and Caffrey 2002). Some species, such as the ferruginous hawk (*Buteo regalis*), generally prey on live animals, but may scavenge when resources are available (Stephens et al. 2008).

Foraging strategy

While most scavenging species will do so either individually or in groups, several factors related to foraging strategies can result in group feeding behavior at carcasses (Figure 2). Turkey vultures have well-developed olfactory organs and are often the first to locate carrion (Kirk and Mossman 1998). Species such as black vultures, condors, and bald eagles are more reliant on

visual clues, such as other scavengers feeding on a carcass, and thus tend to fly high, keeping other scavengers in view to follow them to carcasses (Buckley 1999, Buehler 2000, USFWS 2013e). Vultures and smaller birds cannot open thicker skin of some carcasses, so must wait for larger scavengers such as eagles or condors to open intact carcasses, which may displace them in the process (Kirk and Mossman 1998, Buckley 1999). However, specialization by location, type of carcass, and even within the carcass can result in no one species dominating a food source. For example, turkey vultures tend to feed on smaller items, less often near humans, and favor muscle and connective tissue, as opposed to viscera (Kirk and Mossman 1998). Species such as black vultures and bald eagles may visit the same carcasses for several days, thus providing a pathway for others in the roost to discover the carcass and increasing the number of individuals exposed to a single carcass (Buckley 1999, Buehler 2000). Group size has been studied in black vultures, and the number of vultures feeding at a carcass increased significantly with carcass size, with groups as large as 98 individuals recorded at a single feral hog (*Sus scrofa)* carcass (Buckley 1999).

Figure 2. Bald eagles feeding on the offal pile from a white-tailed deer killed in Monroe County, Iowa in January 2013.

Photo courtesy of Peter Eyerhalde. Iowa State University.

The tendency of scavengers to exhibit group feeding behavior may enhance their vulnerability to lead exposure in a number of ways. For one, the probability of vultures finding food has been found to be related to the density of vultures in the habitat (Jackson et al. 2008). As the number of foragers increases, so does the probability of finding a carcass. Likewise, if vulture populations experience decline and fall below a critical level, the feeding efficiency of each individual falls dramatically. In addition, the propensity for many individuals to feed on a single food source can expose them to a common source of contamination. Demographic modeling of vultures in the Indian subcontinent affected by poisoning from use of the non-steroidal anti-inflammatory drug diclofenac revealed that observed population declines (22-50% annually) could be attributed to a very small proportion of carcasses (between 1:130 and 1:760) containing lethal concentrations of the toxicant (Green et al. 2004).

Discussion Points: Avian Scavengers at Risk - Fragmentation

- Birds that feed on carcasses are at risk of ingesting lead ammunition from hunter-killed game. Birds that feed exclusively on carcasses may have increased risk.
- The tendency of scavengers to exhibit group feeding behavior can enhance their vulnerability to lead exposure at both an individual and population level.

Bioavailability of ammunition to birds: Fragmentation

Bioavailability is the extent to which a contaminant is free for uptake into an organism. While ammunition pellets are small enough to be ingested by most birds, an intact bullet is generally too large. For this reason, the bioavailability of lead from carcasses and offal left by hunters is greatly enhanced by the tendency of bullets to fragment into small pieces and disperse within a carcass (Figure 3). Several studies have documented that lead-containing bullets fragment and migrate a considerable distance in prey animals upon impact. This property makes bullet fragments easily ingested, difficult to avoid when ingesting contaminated tissue, and potentially available to multiple scavengers. Copper bullets, in contrast, exhibit much less frangibility and tend to remain intact, and thus are much less bioavailable to consumers of carcasses (Figure 4).

Figure 3. Radiograph shows lead fragments as white specks in the offal pile from a white-tailed deer shot with a .50 caliber muzzleloader in 2012 on the Upper Mississippi River National Wildlife and Fish Refuge. The bullet's intact copper jacket (shown in the upper left corner) mushroomed, exposing the lead core which fragmented into 107 pieces that were spread throughout the offal pile.

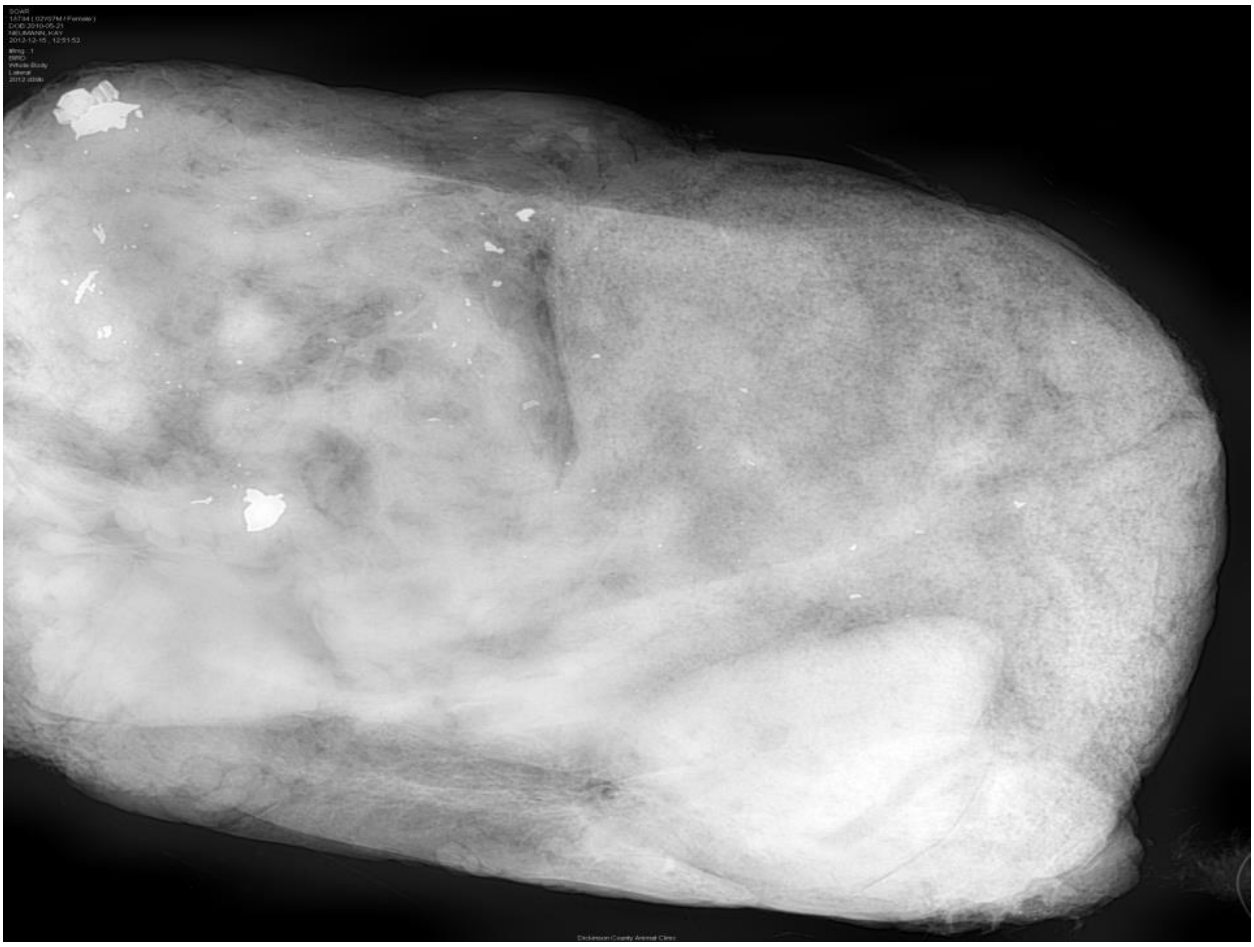


Photo courtesy of USFWS Upper Mississippi River National Wildlife and Fish Refuge, 7071 Riverview Rd. Thomson IL 61285.

Figure 4. Fragmentation of spent lead ammunition, copper jacket with lead core (left) and pure copper (right).

Photo courtesy of Institute for Wildlife Studies, P.O. Box 1137 Tres Pinos, California 95075

Fragmentation in large game

Experimental fragmentation studies in large game and examination of hunter-killed carcasses showed similar patterns in bullet fragment numbers, size, and migration from the wound site.

Hunt et al. (2006) collected whole or partial remains of deer killed by hunters with centerfire, breach-loading rifles in California and Wyoming between 2002 and 2004. Thirty-four were killed using copper-jacketed bullets with lead cores, and 4 with monolithic copper expanding bullets. Local veterinarians radiographed areas of bullet transition, and fragments were counted manually; the presence of metal was verified by dissection in one sample. No attempt was made to distinguish between lead and copper fragments. All whole or eviscerated deer killed with lead-containing bullets (N=24) contained fragments (38-738), with over 100 fragments counted in 74% of samples. Fragments ranged in size about 0.5mm to >5mm, and clusters radiated as far as 15 cm from the wound channel. Ninety percent of offal piles (N=20) contained fragments with

counts ranging from 2 to 521. For deer shot with copper bullets, only 6 fragments total were found in four whole carcasses, and one fragment in four offal piles.

Grund et al. (2010) shot euthanized sheep broadside from a range of 50 m as surrogates for deer. Six types of lead-containing bullets were studied (two rapid expansion, two controlled expansion designed to reduce fragmentation, one slug, and two muzzle-loaders) and one copper bullet. Fragments were detected in all carcasses shot with lead-containing bullets, but varied according to bullet type. While all expansion bullets had similar mass and velocity, one of two controlled expansion bullets produced fewer fragments and less migration from the wound site (2 - 28 fragments per carcass within 25 cm) than other expansion bullets (21 - 498 fragments per carcass within 45 cm). Similar results were obtained from 8 deer shot with rapid expansion bullets as part of a disease management program (Figure 5). Slugs and muzzleloaders had greater mass and lower velocity than expansion bullets, though still fragmented, albeit to a lesser extent (slugs produced up to 3 - 127 fragments, muzzleloaders up to 1- 105 fragments). Lead fragments from these bullet types tended to travel less. Rinsing of carcasses caused lead to be detected further from the exit wound for all bullet types. Copper bullets produced between 1 - 4 fragments per carcass. The authors concluded that while all lead-containing bullets produced fragments, hunters could minimize fragmentation via bullet selection, though could not rely on the advertised claims for selection. The authors also concluded that based on fragment migration within the carcass that all meat from a deer harvested with a lead bullet has the potential to contain at least some lead (e.g., Figure 5 - migration of lead shot throughout the body cavity).

Figure 5. Radiograph of sheep shot with lead ammunition (rapid expansion bullet fired from a 0.308 Winchester) behind the scapula. Bullet fragments are within the red circles and are throughout the thoracic cavity and into the pelvic cavity.

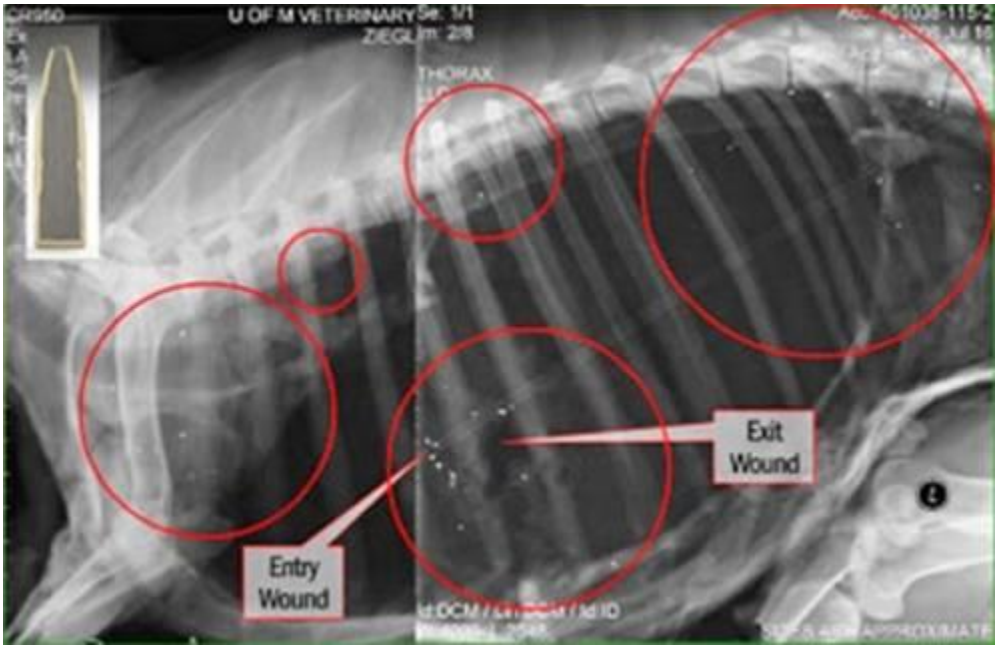


Photo courtesy of Minnesota Department of Natural Resources, Farmland Wildlife Populations and Research Group, 35365 800th Ave, Madelia, MN 56062.

Dobrowolska and Melosik (2008) collected muscle tissue from wild boar (*Sus scrofa*) and red deer (*Cervus elaphus*) killed by hunters in Poland. Bullet types were selected by hunters and varied, though no attempt was made to establish a relationship between bullet type and lead contamination in their analysis. Elevated lead was detected 15 cm from the bullet pathway in all boar carcasses (N=10) and 30 cm from the bullet pathway in 3 of 10 carcasses. Similarly, elevated lead was detected 15 cm from the bullet pathway in all deer carcasses (N=10) and 30 cm from the bullet pathway in 3 of 10 carcasses.

Fragmentation in small game

To study the potential hazard of predators and scavengers consuming small game, fifteen Richardson's ground squirrels (*Spermophilus richardsonii*) were shot using a .22 caliber rifle and hollow-point rimfire ammunition (Knopper et al. 2006). Bullet fragments were visible on 14 of

15 carcasses as minute debris rather than larger pieces of bullet. Carcass lead concentrations ranged from 0.01 – 17.21 mg (median = 3.23 mg). The authors theorized that larger fragments have enough momentum to leave a small carcass, but dust-like lead debris left behind may be an appreciable source of lead for consumers. Pauli and Buskirk (2007) examined fragmentation in black-tailed prairie dogs (*Cynomys ludovicianus*) shot with non-expanding lead bullets enclosed by a copper jacket or expanding soft-point lead bullets. All prairie dogs were shot at 20 – 200 meters to mimic typical shooting events. Radiographs revealed fragments in 26 of 30 prairie dogs shot with expanding bullets and 2 of 29 shot with non-expanding bullets. To identify metal type visible in radiographs, all fragments were extracted from 10 carcasses shot with expanding bullets, and the 2 carcasses shot with non-expanding bullets that contained visible fragments. Fragments averaged 317.8 mg (235.7 mg from the lead core, 23.2 mg from the copper jacket) for expanding bullets, and 43.0 mg (19.8 mg lead core, 23.2 mg copper jacket) for the non-expanding bullets. Most fragments were of a small enough size (73% less than 25 mg each) that the authors hypothesized would be too small to be either avoided during ingestion or egested through regurgitation by a predator or scavenger. Stephens et al. (2008) detected bullet fragments in 4 of 10 black-tailed prairie dog carcasses from shooters at Thunder Basin National Grassland, Wyoming. Lead was the primary metal in fragments of 1 carcass and copper the primary metal in the other 3 carcasses. Lead content averaged 57.3 mg in fragments from 3 carcasses with higher than trace amounts of lead. 

Fragments in meat from hunter-killed game

Lead fragments have also been detected in meat processed from hunter-killed carcasses. Fragments were commonly observed via radiograph in white-tailed deer (*Odocoileus virginianus*) killed by hunters using copper-jacketed lead bullets during the Wyoming hunting season and then commercially processed (Hunt et al. 2009). All 30 carcasses contained bullet fragments (15 – 409 fragments per carcass), with fragment separation up to 45 cm. In ground meat, fragments were visible in packages from 24 of 30 deer (80%) and in 74 of 234 packages (32%). Analysis of fragments excised from ground meat from 13 deer identified lead in 25 of 27 samples (93%). Nine samples contained copper at greater than background levels. In Wisconsin, lead was detected in 30 of 199 (15%) commercially processed packages of venison, and 8 of 98

(8%) samples collected from hunters (Thiboldeaux 2008). Lead concentrations averaged 15.9 ppm in commercially processed samples positive for lead and 21.8 ppm in hunter submitted samples positive for lead. Pharmacokinetic modeling predicted a risk of elevated blood lead concentrations from eating as little as one venison meal per month. In North Dakota, 100 samples of ground venison packages were selected from 15,250 packages donated to food pantries (Cornatzer et al. 2009). High definition computer tomography revealed metal fragments in 59 of 100 packages. Of 15 random samples from all 100 packages, one tested positive for 120 ppm lead. Lead concentrations in five samples known to contain metal fragments ranged from 4200 – 55,000 ppm.

In a follow-up investigation to the North Dakota study by the Centers for Disease Control (CDC), blood samples were collected from 736 volunteers in six cities in North Dakota (Iqbal 2009). Participants that consumed wild game had higher blood lead levels than those who did not, with those consuming the greatest serving sizes having the highest concentrations. The average blood lead concentration was 0.0117 ppm, with 1.1% of samples $\geq$0.05 ppm. No samples exceeded the CDC recommended case management threshold of 0.001 ppm, though the authors note that there is no clinical threshold of lead in the human body that is considered safe, and that that increased risk of factors such as myocardial and stroke mortality have been observed at $\geq$0.02 ppm. Since the publication of this study, the CDC has lowered its threshold to identify children at risk to 0.05 ppm, and removed the phrase "level of concern" from its publications, noting that no safe blood lead level in children has been identified (CDC 2012).

Discussion Points: Avian Scavengers at Risk - Fragmentation

- Regardless of the type of game or bullet, all studies showed that lead bullets fragment, sometimes substantially, when fired into an animal.
- Confirmation that metal fragments contained lead (as opposed to another metal from the base or jacket of the bullet) was performed either by dissection of fragments or lead analysis of surrounding tissue.
- Fragment size and number varied, and distance from the wound channel was generally up

to 15 - 45 cm in large game. Some carcasses contained hundreds of fragments.

- Use of non-expanding bullets may reduce fragmentation, but did not perform as such consistently.
- The creation of small lead fragments increases availability to scavengers due to their tendency to migrate from wound channels, avoid detection or regurgitation by scavengers, and be abundant enough to expose several scavengers feeding on a single carcass.
- In contrast to lead-contained bullets, monolithic copper bullets consistently held together and produced a low number of fragments, if any, within carcasses. Non-fragmentation of bullets significantly reduces their bioavailability to predators and scavengers.

Sensitivity

Avian sensitivity to lead is influenced by diet and physiology. Species and individuals may also possess inherent sensitivity driven by genetic makeup: three polymorphic genes have been identified that influence the bioaccumulation and toxicokinetics of lead in mammals though this has not yet been investigated in birds (Onalaja and Claudio 2000).

Physiology of raptors and scavenging birds

Birds have a unique physiology that can enhance their vulnerability to lead toxicosis by facilitating breakdown and absorption into tissue. Species such as waterfowl that feed on coarse objects like grain or plant material have larger muscular gizzards for grinding (Farner 1960). This grinding facilitates the erosion of ingested metallic lead, making it more bioavailable for absorption in gastrointestinal tract and subsequent transport to other organs (Jordan and Bellrose 1951). While carnivorous birds may have highly reduced gizzards (and omnivorous birds intermediate to the two groups), other digestive characteristics facilitate the absorption of items from the gastrointestinal tract. While stomach fluids are highly acidic in most species, raptors have been found to have an especially low pH in their stomachs, even compared to other carnivorous birds (Duke 1995). The average pH of 1.6 measured during gastric digestion in falcons translates to about six times more hydrogen ion per ml in their basal gastric secretion than was measured in owls (average pH 2.35) (Duke 1995). Other species studied included

turkeys (*Meleagris gallopavo*; pH 3.0) and domestic ducks (*Anas platyrhynchos x Cairina moscata* hybrid; pH 2.1). Pellets of raptors exhibit more thorough corrosion of bone as compared to those of owls, likely due to this difference in gastric pH (Duke et al. 1975). In addition, birds possess a unique trait in gastrointestinal mobility that increases residence time of ingested materials in the digestive system, including the highly acidic stomach. Periodic reverse peristalsis moves the contents of the upper ileum and duodenum back into the stomach, an adaptation hypothesized to allow for greater digestion of nutrients without lengthening the gastrointestinal tract, which would be disadvantageous to flying due to added weight (Duke 1997). Raptors feeding on mice had 1-2 refluxes of this nature per hour to aid in digestion (Duke 1997).

Influence of diet on sensitivity

Diet can have an effect not only the exposure of birds to lead, but on its toxicity and storage. Jordan and Bellrose (1951) reported that diet was a more important influence on the toxicity of lead in waterfowl than was the dosage of lead, within a range of one to four #6 pellets. Dietary factors such as physical form, nutritional properties, and rate of consumption impacted the toxicological effect of lead. Toxicity can be enhanced by nutritional deficiencies in the diet (e.g., calcium, zinc, iron, protein, fat) and decreased by excesses of others (e.g., zinc, protein, fat) (Eisler 2000). In particular, lead possesses physical properties similar to those of calcium, allowing it to mimic or inhibit the action of this substance, and compete for absorption in the gut (Quarterman 1986). Studies in waterfowl have found that individuals ingesting foods high in protein and calcium were less susceptible to toxic effects of lead (Sanderson and Bellrose 1986). Dietary influence on lead toxicity has since been studied in other species, and findings indicate that toxic effects are less severe when birds are on nutritionally balanced diets high in protein and calcium. The mitigating effects of such diets may be the result of high calcium and protein levels reducing lead absorption in the gastrointestinal tract and lowering the body burden of lead (Koranda et al. 1979, Sanderson 1992, Scheuhammer 1996). Differences in lead concentrations found in species with similar ingestion rates have been attributed to this variation in diet, with species feeding on increasing amounts of animal matter showing a lower accumulation of lead in tissues (Stendell et al. 1979). Reproductively active female birds have been found to accumulate

lead at a greater rate than males, a characteristic presumably linked to increased intestinal calcium absorption during eggshell formation (Scheuhammer 1987). The effects of dietary preferences upon lead toxicity have not been specifically studied in scavengers, but their high intake of animal matter may influence the effects of ingested lead, and could vary among species that specialize on different parts of the carcass.

Results of toxicity testing of scavengers

Laboratory toxicity studies of lead ammunition have been performed on three species of scavenging birds, the Andean condor (*Vultur gryphus*) (Pattee et al. 2006), turkey vulture (Carpenter et al. 2003), and bald eagle (Hoffman et al. 1981, Pattee et al. 1981). For all three species, dosed birds exhibited individual variability in sensitivity, possibly related to factors such as length of shot retention, number of shot retained, amount of lead eroded, and individual susceptibility. Though protocols differed across studies (Table 3), the Andean condor appeared to be more sensitive than other raptor species tested, with all 4 birds dosed with either 2 or 6 lead pellets (size 00) showing signs of lead poisoning within 50 days, and two of those succumbing. This contrasts with a time to mortality of 10 – 155 days in 5 bald eagles dosed with 10 – 156 lead pellets (size 4), and a minimum time to death of 143 days in 6 turkey vultures dosed with up to 3 or 6 lead pellets (size BB). Two turkey vultures were euthanized at 211 days showing no overt signs of lead poisoning. The authors concluded that the vultures showed considerable tolerance to lead shot when compared to other experimentally treated raptors.

Shot retention varied among individuals of each species. Redosing of turkey vultures was described as "constant" due to defecation or regurgitation of shot (Carpenter et al 2003). Bald eagles retained shot as little as 12 hours and as long as 48 days (Pattee et al 1981). Dosed shot was recovered from all 4 condors at necropsy 39-49 days after dosing (with one bird having been redosed at day 7 following regurgitation) (Pattee et al. 2006). Shot retention has been reported in eagles that have succumbed to lead poisoning in the wild (e.g., 77 shot recovered in one eagle, Jacobson et al. 1977; Figure 6 - lead shot pellets in a bald eagle as seen on an x-ray). The variability in shot retention seen here is consistent with that seen in other species that ingest lead

pellets, which may erode or pass pellets through the gut before death occurs (Roscoe et al. 1979, Schulz et al. 2006).

Table 3: Comparison of dosing protocols in three scavenging species

Species	*No. pellets dosed*	*Shot number*	*Shot diameter (mm)*	*Approx. no. pellets in 1 oz*	*Total lead eroded (mg)*
Andean condor[1]	2 or 6	00	9.14	6.2	126-603
Turkey vulture[2]	3 or 10	BB	4.57	50	111-247
Bald eagle[3]	10-156	4	3.28	135	19-185

[1]Pattee et al. 2006, [2]Carpenter et al. 2003, [3]Pattee et al. 1981

Figure 6. X-ray showing two #5 lead shot in the digestive tract of a bald eagle admitted to a wildlife rehabilitation center in Iowa. Blood levels measured were .65 ppm. Eagle died at facility

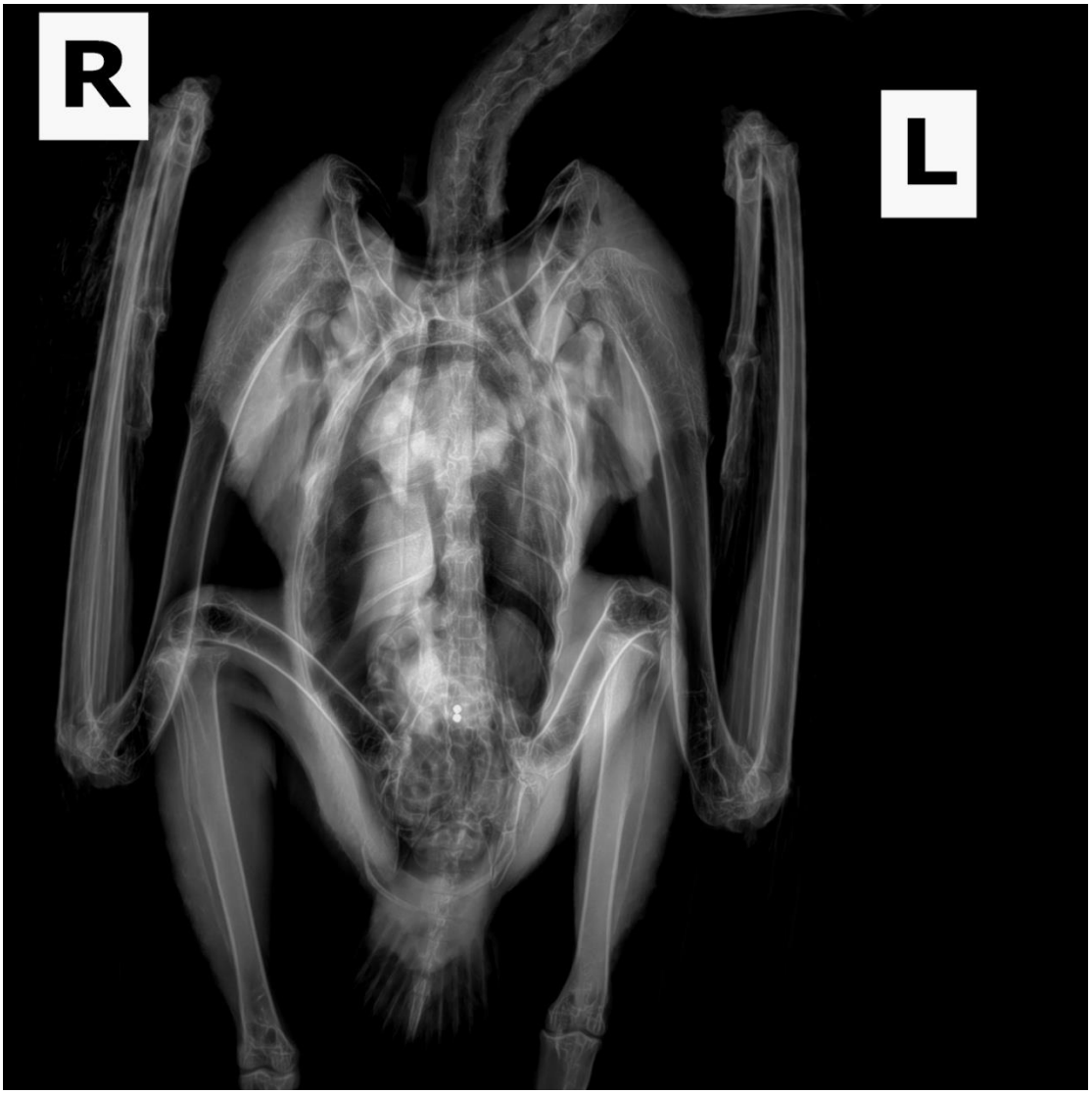


Photo courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

Studies on all three scavenging birds confirmed that dosed metallic lead pellets were soluble in the digestive tract either by measuring erosion of lead pellets post-dosing or by detection of lead in blood and other internal organs shortly after dosing. Total lead eroded after dosing varied among individuals and ranged from 126-603 mg (condors), 111-247 mg (turkey vultures), and 19-185 mg (bald eagles) (Table 3). For all species, an increase in blood lead concentrations and a decrease in ALAD followed treatment, with blood lead continuing to rise after dosing. These

changes were detected in bald eagles within 24 hours of treatment (Hoffman et al. 1981), and in condors and vultures at the first blood collection seven days after dosing.

General guidelines for the use of tissue concentrations in defining exposure and effects in birds are <2 ppm wet weight in liver for birds with no history of lead exposure and >6 ppm wet weight in liver in for birds with evidence of clinical poisoning (Franson and Pain 2011). Mortality can be diagnosed by clinical findings and residues in excess of 6 ppm wet weight in liver. While liver concentrations for these three species were in general agreement with these diagnostic thresholds (Table 4), the individual variability exhibited stresses the need to incorporate diagnostic criteria such as field observations, clinical signs, gross lesions and pathological findings when making a finding of lead poisoning.

Table 4: Comparison of hepatic lead concentrations in three experimentally treated scavenging species

	Lead in liver at necropsy (ppm wet weight)			
	Control	No signs of intoxication	Intoxication, euthanized	Mortality
Andean condor[1]	Not analyzed	-	49.88, 109.09	45.46, 58.52
Turkey vulture[2]	0.05, 0.10	1.48, 2.22	6.79, 18.71	20.73, 33.78
Bald eagle[3]	0.4	-	3.4	11.5 – 27.0

[1]Pattee et al. 2006, [2]Carpenter et al. 2003, [3]Pattee et al. 1981

Signs of toxicosis in condors, vultures and eagles showing clinical poisoning included lack of coordination, loss of appetite, lethargy, weakness, reduced activity, postural change, drooped wing, and frequent opening of the mouth, each occurring in one or more species (Pattee et al. 1981, Carpenter et al. 2003, Pattee et al. 2006). All three species exhibited emaciation, with turkey vultures showing a loss of pectoral muscle, subcutaneous fat, and coelomic fat, and condors showing a loss of subcutaneous, abdominal, and coronary fat. Histological findings included spongiosis in brain tissue of vultures and condors, and nephrosis in some individuals of all species. All four eagles exhibited myocardial nephrosis. It is notable that acid-fast inclusions

were present in epithelium in turkey vultures and condors, but not in bald eagles. As noted above (see: Toxicological Impacts of Lead to Birds), inclusion bodies are indicative of lead exposure, but are not consistently present in all species or all individuals within a species. It is notable that despite overall evidence of clinical poisoning, individual gross and histological signs varied among individuals and were not consistently displayed.

Discussion Points: Avian Scavengers at Risk - Sensitivity

- Despite the reduced gizzard, carnivorous birds contain physiological adaptations (low pH, reverse peristalsis) that facilitate the absorption of metallic lead into tissues.
- Scavenging birds can retain shot in the gastrointestinal tract, though the length of time may vary. The absence of shot or fragments may indicate either a lack of exposure, or regurgitation or complete absorption following ingestion.
- The solubility of ingested metallic lead has been demonstrated in lab studies by increased blood concentrations and reduced enzyme activity following treatment.
- Tissue residues of lead-exposed and lead-poisoned birds can vary and should be interpreted in conjunction with other diagnostic signs.
- Clinical signs diagnostic of lead poisoning may not always be exhibited by exposed species or individuals, despite known toxicosis.

Mortality and Morbidity of Avian Scavengers

The exposure potential and sensitivity of scavenging birds to lead ammunition described above has resulted in numerous documented cases of their mortality and morbidity. One study found that of 130 species documented to be affected by lead ammunition, 24% were raptors and scavengers (Tranel and Kimmel 2009). Herein, we discuss examples of lead poisoning or exposure in North American predatory and scavenging birds. However, to avoid conflation with lead poisoning that may have resulted from predation on waterfowl, we included only examples after the 1991 ban. Table 5 lists such examples of lead exposure in 14 species of raptors. Some cases include evidence suggestive of exposure to lead ammunition, such as seasonality

contemporary with hunting activities or radiography of gastrointestinal contents. Below we discuss selected cases in further detail.

In cases of mortality, incident reports are likely representative of only a subset of the actual exposure or mortality attributable to lead. In order to document a contaminant-related mortality, a carcass must be observed, reported, collected, and chemically analyzed while still relatively fresh (Vyas 1999). Carcass-detection studies have found that even when searches are performed on carcasses known to exist (e.g., placed by a researcher for study), a significant percentage will never be found due to scavenging, location in remote, inaccessible areas, or size or coloration that renders the carcass inconspicuous (Vyas 1999). Therefore, detection and diagnosis of any contaminant-related mortality is generally believed to represent a much larger number of incidents.

Table 5. Examples of lead exposure after the 1991 lead shot ban in predatory and scavenging birds in North America

Species	Reference
California condor (*Gymnogyps californianus*)	[acd]Fry and Maurer (2003); [a]Church et al. (2006); [abcd]Cade (2007); [a]Finkelstein et al. (2012); [acd]Rideout et al. (2012); [abcd]USFWS (2012, 2013e)
Turkey vulture (*Cathartes aura*)	[a]Clark and Scheuhammer (2003); [a]Martin et al. (2008); [b]Kelly et al. (2011); [b]Kelly and Johnson 2011)
Northern harrier (*Circus cyaneus*)	[a]Martin and Barrett (2000); [a]Martin et al. (2008)
Sharped-shinned hawk (*Accipiter striatus*)	[a]Martin and Barrett (2000)
Cooper's hawk (*Accipiter cooperii*)	[a]Martin and Barrett (2000); [a]McBride et al. (2004)
Northern goshawk (*Accipiter. gentilis*)	[a]Martin and Barrett (2000)
Red-tailed hawk (*Bueto jamaicensis*)	[d]Franson et al. (1996); [ad]Clark and Scheuhammer (2003); [a]Martin et al. (2008); [a]Stansley and Murphy (2011)
Ferruginous hawk (*Buteo Regalis*)	[a]Stephens et al. (2008)
Golden eagle (*Aquila chrysaetos*)	[b]Kramer and Redig (1997); [d]Wayland and Bollinger (1999); [ad]Clark and Scheuhammer (2003); [a]Domenech and Langer (2009); [b]Stauber et al. (2010); [a]Kelly and Johnson (2011); [d]Harmata et al. (2013)
Bald eagle (*Haliaeetus leucocephalus*)	[d]Langelier et al.(1991); [d]Elliott et al. (1992); [d]Scheuhammer and Norris (1996); [d]Wayland and Bollinger (1999); [ad]Clark and Scheuhammer (2003): [b]Strom et al. (2006); [bcd]Neumann et al. (2008); [b]Stauber et al (2010); [a]Harmata (2011); [b]Bedrosian et al. (2012); [b]Cruz-Martinez et al. (2012)
American kestrel (*Falco sparverius*)	[a]Martin and Barrett (2000); [a]Clark and Scheuhammer (2003)
Great horned owl (*Bubo virginianus*)	[ad]Clark and Scheuhammer (2003) ; [a]Martin et al. (2008)
Eastern screech owl (*Megascops asio*)	[a]Martin et al. (2008)
Common raven (*Corvus corax*)	[ab]Craighead and Bedrosian (2008, 2009)

[a] Lead exposure (detected in tissue)
[b] Lead exposure coinciding with hunting season
[c] Evidence of ingestion of lead ammunition
[d] Lead poisoning based on tissue concentrations

California condors, California and Arizona: Lead poisoning was diagnosed in 23 of 135 mortalities in the California condor population between 1993 and 2009 (Rideout et al. 2012). Collection of blood samples from free-flying condors found that an average of 71% of birds tested each year in the California population (1997 to 2010) and between 48% and 95% of birds from the Arizona population (2000 to 2007) contained lead levels above background (Parish et al. 2009, Finkelstein et al. 2012). Further details are provided in the California condor case study below.

Bald eagles, Great Lakes: Wildlife rehabilitators and diagnostic laboratories in the Great Lakes region have found significant morbidity and mortality of bald eagles from lead exposure. Between 1996 and 2009, 1,227 moribund bald eagles were processed in Minnesota, with 331 (27%) of the birds having lead concentrations consistent with lead poisoning in the blood (Cruz-Martinez et al. 2012). Between 2000 and 2008, 62 moribund bald eagles were processed with 39 (62.9%) of the birds having lead concentrations consistent with poisoning in the blood or liver (Neumann 2009). Between 2000 and 2008, 583 dead bald eagles were processed with 87 (15%) diagnosed with lead poisoning (Strom et al. 2009). Further details are provided in the bald eagle case study below.

Bald eagles, Maine: Mierzykowski et al. (2013) found that 19 out of 127 (15%) bald eagles found dead in the State of Maine between the years 2001 and 2012 had liver lead concentrations greater than the threshold for lead poisoning (6 ppm wet weight). The highest lead concentrations were detected in the eagles collected during the winter and early spring months.

Turkey vultures and golden eagles, California: Lead exposure was elevated in blood of 83 of 172 turkey vultures collected from California in 2008 and 2009 (>0.10 ppm; Kelly et al. 2011). In an intensely hunted area, average blood lead levels were twice as high during the hunting season as during the off-season, with 76% having elevated concentrations during the hunting season as compared to 36% prevalence outside of the hunting season. After the ban on lead ammunition, there was a reduction in blood lead concentrations in turkey vultures and golden eagles (Kelly

and Johnson 2011). Following the ban, non-migrant golden eagles had significantly lower blood lead concentrations than eagles of unknown residency status.

Golden eagles, Montana: Of 42 migrant golden eagles sampled in Montana ~~from~~ during the fall of 2006 and 2007, 58% had elevated blood lead levels (Domenech and Langner 2009). For all eagles, blood lead ranged from <0.005 to 4.81 ppm, with 18 exhibiting background levels (0-0.10 ppm), 19 subclinical exposure (0.10-0.60 ppm), 2 clinically exposure (0.60-1.00 ppm), and 3 acute exposure (>1.00 ppm).

Turkey vultures, Kentucky: In 2013, the USFWS (Anthony Velasco, personal communication, 2013) reported finding four live black vultures in Kentucky during January and February of 2013 with blood lead concentrations (0.255 ppm) sufficient to cause mortality or require veterinary intervention. These data have been reported by the USFWS but have not been written up for publication.

Ferruginous hawks, Golden eagles, Wyoming: In 2001, Stephens et al. (2008) investigated blood lead concentrations in nestling ferruginous hawks and golden eagles collected from Thunder Basin National Grasslands, Wyoming, due to local increases of lead poisoned raptors being admitted to wildlife rehabilitation facilities. Lead was detected in blood of all nestlings, but at less than sublethal concentrations. Ferruginous hawks and golden eagles are known to scavenge on black-tailed prairie dogs at this location that have been killed by shooters and not recovered. Concentrations of lead in nestlings were similar to those at a reference site where no hunting takes place. As scavenging on prairie dog carcasses is a known exposure pathway (bullet fragments were detected in 4 of 10 carcasses collected), the authors hypothesized that lead levels may have been low due to decreased shooting at the site that season and an increase in alternate food sources for raptors.

Common ravens, Wyoming: Craighead and Bedrosian (2008) found that of 302 ravens sampled in the southern Yellowstone River ecosystem during 2004 and 2005, 47% exhibited elevated blood lead levels (≥0.10 ppm) during the hunting season as compared to 2% during the nonhunting

season. Expanding upon this data, addition samples were collected through 2008 (Craighead and Bedrosian 2009a). For all samples pooled (N = 539), median blood lead levels were 0.10 ppm for the hunting season and 0.02 ppm during the nonhunting season. A significant relationship was detected between annual median blood lead levels and the combined large-game harvest success from the National Elk Refuge and Grand Teton National Park.

Bald eagles, golden eagles, Wyoming: Craighead and Bedrosian (2009b) also found that bald eagles and golden eagles accumulated more lead in blood samples during the hunting season (median 0.56 ppm) in the southern Yellowstone River ecosystem compared to the non-hunting season (median 0.277 ppm). Nine blood samples out of the 63 (14.3%) eagles had concentrations greater than the threshold for lead poisoning (1.0 ppm).

Discussion Points: Mortality and Morbidity of Scavengers

- Avian species continue to be exposed to be exposed to and poisoned by lead despite the 1991 ban for use of lead ammunition in waterfowl hunting.
- Lead exposure has been documented in numerous species of predatory and scavenging birds, and in many regions of North America.
- Some documented cases of elevated lead exposure include evidence suggestive of exposure to lead ammunition, such as co-occurrence with hunting seasons or detection of fragments in the gastrointestinal tract.
- Documented cases likely represent a much larger number of affected birds.

Temporal Trends of Mortality and Hunting Season

A rise in lead exposure events and mortality during or immediately after game hunting seasons was formerly observed in scavenging birds prior to the lead shot ban for waterfowl in the U.S. (Bloom et al. 1989, Wiemeyer et al. 1989). This temporal trend continues to be observed (*bald eagle*: Wayland and Bollinger 1999, Craighead and Bedrosian 2009b, Strom et al. 2009, Cruz-Martinez et al. 2012; *golden eagle*: Wayland and Bollinger 1999, Craighead and Bedrosian

2009b, Kelly and Johnson 2011; *California condor*: Hunt et al. 2007, Green et al. 2009, Parish et al. 2009; Stauber et al. 2010; *turkey vulture*: Kelly et al. 2011; c*ommon raven*: Craighead and Bedrosian 2008, 2009a).

In the upper Midwest, there are at least two studies describing a trend between hunting seasons and an increase in lead exposure events in wintering bald eagles. Deer hunting in this region occurs in the winter when waterways are frozen and the ground is covered in snow for an extended duration. This seasonality plays an important role in lead exposure as the diversity of food becomes limited, and bald eagles opportunistically forage on readily available sources such as hunter-killed deer and offal piles. A study that analyzed 1,277 bald eagles admitted for rehabilitation in Minnesota from 1996 - 2009 found that 27% were admitted with elevated lead concentrations, and that there were increased odds for elevated lead levels based on hunting season, deer hunting zones, and age of bird (Cruz-Martinez et al. 2012). Another study analyzed necropsy data from the Wisconsin Health Program of the Wisconsin Department of Natural Resources to investigate lead exposure in bald eagles from 2000-2007 (Strom et al. 2009). This study reported lead toxicity as the cause for 15% of the bald eagle deaths in Wisconsin, with an increase in mortality beginning in October and peaking in December. The Wisconsin deer hunting season overlaps during this time period.

In Arizona, California condor mortality associated with lead exposure and mortality has predominantly occurred in the fall and winter months and is associated with the big-game hunting seasons (Parish et al. 2009). Since 2002, an increase of condor blood lead concentrations has been found to correspond with deer hunting areas in southern Utah (USFWS 2012). Similarly, a study in the central region of the condor range in California found blood lead concentrations in turkey vultures to be significantly associated with big game hunting. This central region of the condor range has high wild pig hunting intensity (Kelly and Johnson (2011). Bald eagles in Utah were found to have lead shot in 71% of the casts collected and analyzed. The authors hypothesize the likely source to be associated with jackrabbit hunting in the region (Platt 1976).

The temporal association of lead exposure to scavenging birds is also documented outside of the U.S. Wayland and Bollinger (1999) reported lead exposure and poisoning in bald and golden eagles collected in the Canadian provinces. In this report, golden eagles that exhibited high lead exposure died closer to the hunting season as compared to eagles that exhibited low lead exposure. In Europe, raptors that forage on game species have been identified as susceptible to lead exposure from ammunition used in hunting as a result of their feeding habits (Eurasian sparrowhawk (*Accipiter nisus*), northern goshawk (*A. gentilis*), common buzzard (*Buteo buteo* in Pain et al. 1993) and lead exposure to marsh harriers (*Circus aeruginosus*) has been associated with timing of hunting season in France (Pain et al. 1997).

Discussion Points: Temporal Trends of Mortality and Hunting Season

- Several species of scavenging birds have a documented increase of exposure and mortality rates during hunting seasons.
- This temporal pattern is documented in the U.S. and elsewhere
- Bald eagles in the upper Midwest during the winter season have high incidences of lead exposure events and mortality overlapping with the timing of deer hunting.
- The temporal trend of high lead exposure rates in scavenging birds continues even after the lead shot ban of 1991.

Direct Evidence of Exposure: Presence or observation of ammunition

While many lines of evidence connect lead exposure in scavengers with ingestion of ammunition (e.g., diet, foraging behavior, seasonality), positive attribution of lead poisoning to ingested ammunition occurs when bullet fragments or shot can be recovered from birds at necropsy and removed surgically (Figure 7), present on radiographs (Figure 6), or when birds are observed feeding on contaminated carcasses. These lines of evidence in consort with lead concentrations in tissues and supporting clinical signs can be used to diagnose lead poisoning cases. While many cases lack this final direct line of evidence, there are numerous citations of such to complete the pathway, some examples of which are presented herein.

Figure 7. Retrieval of lead shot pellet from gastrointestinal tract of a bald eagle**.**

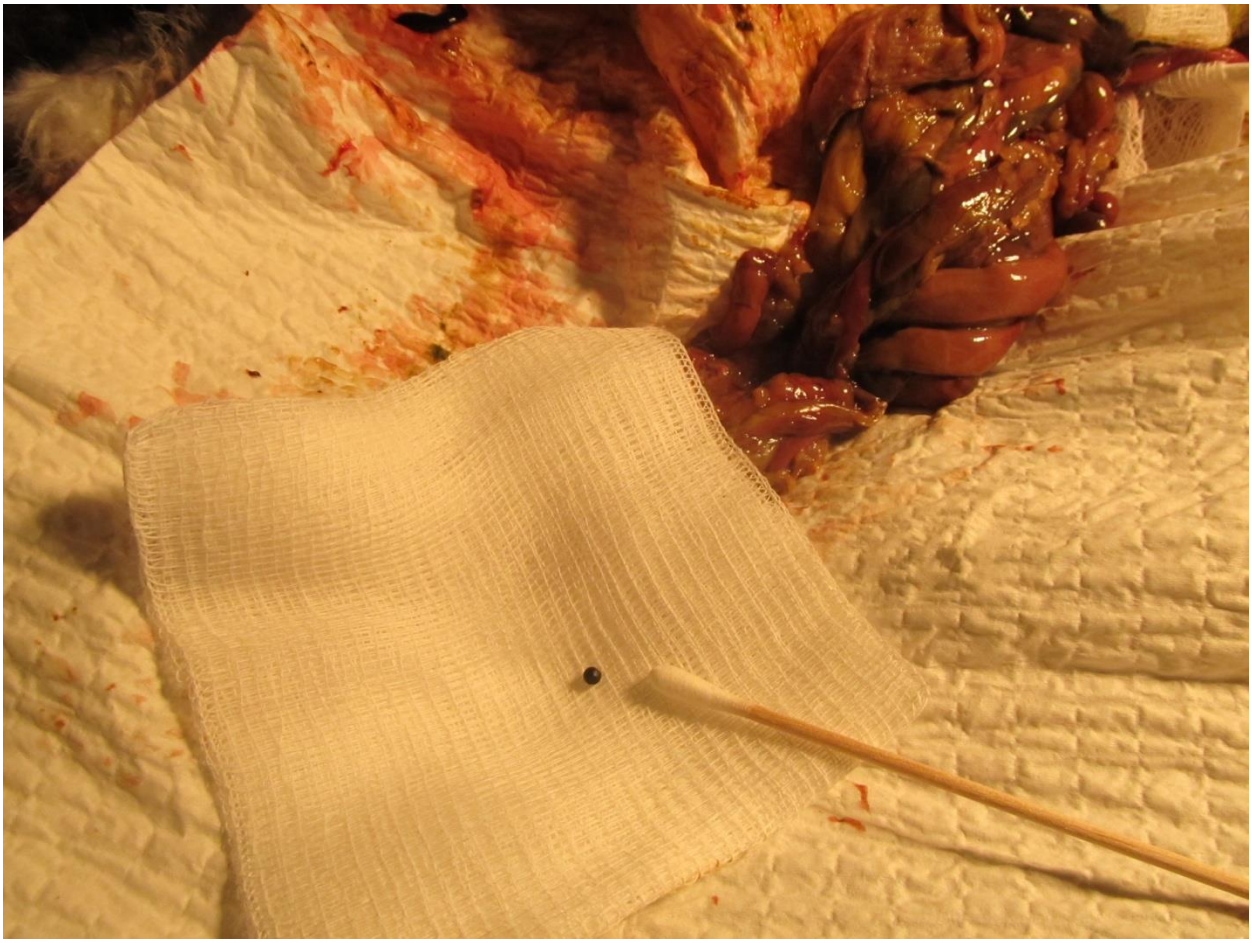


Photo courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

In a report submitted to the California Fish and Game assessing lead exposure cases in California condors from 1984-2001, Fry and Maurer (2003) report 7 condors suspect of having metallic fragments in their digestive systems. Two of these cases were positively confirmed by chemical analysis of the metallic particles. Rideout et al. (2012) reported that 6 California condors from the Arizona population had metallic fragments in a dataset that included mortality cases from 1996-2010. Three were confirmed to be lead ammunition and three were reported as metallic pellets. More recent data for the Arizona population, 2007-2011, indicate that radiographs continue to detect lead-poisoned condors with lead pellets and fragments in their digestive tracks,

as well as bullet fragments in deer and coyotes collected in the condor's range (USFWS 2012). Similarly, Neumann et al. (2009) reports that over 50% of bald eagles admitted to rehabilitation facilities have ingested ammunition (e.g., Figure 8).

Figure 8. Bald eagle carcass displaying ammunition fragment in radiography**.**

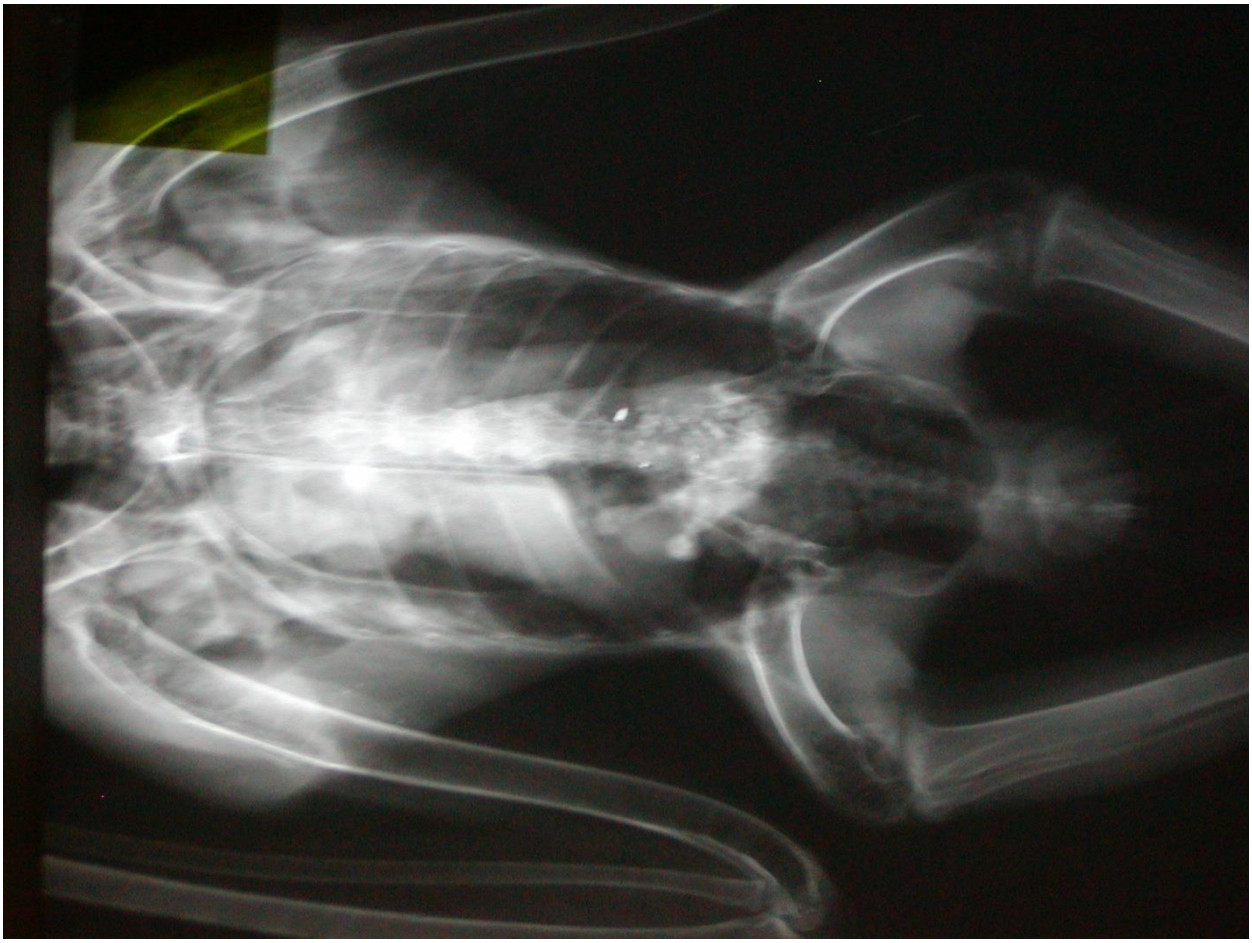


Photo courtesy of Kay Neumann, Saving Our Avian Resources, 25494 320th Str, Dedham, IA 51440

Despite cases of lead fragment detection/recovery or a direct observation of ingestion, the absence of such does not negate the possibility of ammunition as the source of exposure. As described previously, the frangibility of lead can produce fragments that are likely to be small enough to be completely dissolved in the gastrointestinal tract. Absorption of fragments is enhanced by the unique physiology of raptors that combines an extremely acidic environment with prolonged digestion. Fragments that are not completely absorbed may also be passed by

birds prior to death to capture, leaving no evidence in radiographs or post-mortem examination (Rideout et al. 2012). In laboratory studies of scavenging birds dosed with lead shot, retention of pellets varied by length of retention of shot and number of shot retained among individuals of the same species and under similar treatment regimens (Pattee et al. 1981, Carpenter et al. 2003, Pattee et al. 2006).

Discussion Points - Direct Evidence of Exposure: Presence or observation of ammunition

- Recovery of lead fragments or pellets from birds, its presence on radiographs, or observation of exposed birds feeding on contaminated carcasses provide direct evidence of ammunition as a source of lead exposure.
- While this evidence is not always available or sought by those investigating cases of lead poisoning, numerous examples exist in the literature that help to confirm this exposure pathway.
- The absence of a lead fragment or a direct observation of ingestion does not negate the possibility of ammunition as the source of exposure due to a bird's ability to completely absorb or pass the ingested object.

Use of stable isotopes to identify sources of lead in wildlife

Elements are defined by the number of protons contained in the nucleus, 82 in the case of lead. Some elements can exist with different numbers of neutrons in their nucleus, and each variation is called an isotope. Isotopes can be radioactive, and therefore subject to decay, or stable, either not subject to decay or decaying over such a long-half (e.g., millions of years) that they are considered stable. Lead has four stable isotopes that occur naturally: ^{204}Pb, ^{206}Pb, ^{207}Pb, and ^{208}Pb. Ores from which lead is mined are made up of different percentages of these four isotopes, and those percentages are maintained as lead is extracted and manufactured into products. The ratio of any two of these isotopes is sufficient to provide a unique identifier for the lead, with the ratio of ^{207}Pb:^{206}Pb most commonly reported. (all ratios are reported as such herein). Measuring these ratios in human or wildlife tissue and comparing them to ratios in

potential sources of lead (e.g., paint, ammunition, mine tailings) can help to elucidate how biota is exposed to this toxin.

In an attempt to identify sources of lead exposure for wildlife, several studies have examined isotopic signatures of ammunition for comparison with ratios measured in tissues of exposed individuals. Scheuhammer and Templeton (1998) measured ^{207}Pb:^{206}Pb ratios in 22 brands of lead shotshells, finding low variability within cartridges and boxes, but higher variability between brands. Ratios from tissues of lead-exposed birds (game birds, waterfowl, and bald and golden eagles) showed ranges and patterns similar to that measured observed for shot pellets (0.787 – 0.935). Few samples had ratios within the narrow range associated with environmental lead from gasoline (0.8658 – 0.8811), with the exception of bones from 10 lead-exposed herring gulls, a species that rarely ingests lead ammunition. None of the samples analyzed in this study were consistent with ratios associated with mine and smelter waste.

Church et al. (2006) compared ^{207}Pb:^{206}Pb isotopic ratios measured in ammunition, condor dietary items, and condor tissue. Ratios from 13 boxes of purchased ammunition and 9 individual rifle bullets donated from hunters ranged from 0.8054 to 0.8145, much narrower than that determined by Schuehammer and Templeton (1998). Lead isotope ratios in 7 samples of dietary items (0.8253 – 0.8394) were similar to background environmental ratios in California obtained from literature (0.8338 – 0.8453). Blood was collected from both captive (pre-release) and free-ranging condors. Lead levels and isotopic ratios were strongly inversely associated; as condors were exposed to greater lead concentrations, isotopic ratios declined. Isotopic ratios from pre-release condors (0.8296 – 0.8351) were significantly different than free-flying condors with blood lead above 0.0375 ppm (0.8101 – 0.8307), indicating that higher lead concentrations were associated with exposure to a novel source of lead and perhaps shifting downwards towards ratios associated with ammunition.

While Church et al (2006) contend that the narrow range of isotopic ratios measured in ammunition is representative for California condor exposure as samples were purchased in the local area, subsequent studies expanded this range by testing a more expansive set of

ammunition. Finkelstein et al. (2012) collected ammunition from an exchange program, hunters, or from shot carcasses and found an isotopic range of approximately 0.78 – 0.87 ^{207}Pb:^{206}Pb. Results from blood analyses were similar to Church et al. (2006) in that condors exposed to greater lead concentrations showed a decline in isotopic ratios. Isotopic ratios ranged from 0.8296 – 0.8483 in pre-release condors and 0.7602 – 0.9145 in wild condors. Lead exposure for 5 birds at the high end of the range (> 0.9) was attributed to lead-based paint from a fire lookout tower where condors had been observed nesting (for further discussion, see below: Alternative source of lead: Paint). Lead fragments recovered from 6 condors that were either lead-poisoned or observed feeding on a carcass shot with lead-based ammunition had isotopic ratios that were highly similar (≤0.22%) to blood collected from the same bird. The isotopic range of the fragment/blood pairs were approximately 0.81 – 0.83, overlapping slightly with ratios of pre-release condors. Nine of the 110 condors sampled had isotopic ratios that could not be explained by background, ammunition, or paint.

Lead concentrations and isotopic analysis in feathers

The use of feathers to monitor lead levels can provide a simple, non-invasive method to determine exposure in birds. Although many investigators have measured concentrations of lead in this manner, there have been a limited number of controlled exposure studies to validate this use and aid in the interpretation of such field data (Kendall and Scanlon 1981, Burger and Gochfeld 1990, Dauwe et al. 2002, Golden et al. 2003). These investigations have shown that under certain conditions feathers can be a reliable indicator of dietary exposure to lead that is associated with lead accumulation in internal organs and biochemical measures of effect. Golden et al. (2003) used ratios of lead concentrations found in these studies as well as those in juvenile birds collected from the field to calculate feather to tissue ratios for nestling or juvenile birds (1:10 for bone, 1:5 for kidney, and 1:2 for liver). They cautioned careful use of these ratios however, as ratios may vary with tissue collection technique (e.g., bone or feather type), and in situations of low or very high lead exposure.

Golden et al (2003) also cautioned that adult feathers appear to be a less reliable indicator of endogenous lead exposure, primarily due to complications from external deposition of lead.

Previous studies have found higher concentrations of lead in feather parts openly exposed to the environment compared to those components covered by other feathers, and a lack of correlation with concentrations of lead accumulated in internal organs (Goede and de Voogt 1985, Goede et al. 1986, Hahn et al. 1993). Rinsing of feathers does not necessarily remove external contamination, as electron micrographs revealed that feathers exposed to the environment for over a year retained numerous small particles despite extensive cleaning (Weyers et al. 1988). Dauwe et al. (2002) also found evidence that feathers may be subject to exogenous contamination of lead by excretion from the uropygial gland.

To examine lead concentrations and isotope ratios in condors, Church et al. (2006) collected one retrix feather that was partially grown at the time of death from the carcass of Condor 165. This condor had been in the wild approximately 2.5 years before its carcass was recovered and was subsequently diagnosed with lead poisoning. Since condor feathers can grow about ~5 mm per day, the 24 cm feather was likely to have been growing one to two months. Following rinsing in detergent, water, ethanol, and nitric acid, the feather was serially sampled along the length of the rachis and vane. As condors molt on an approximate 2-year cycle (Snyder et al. 1987), this new feather would have been subject to a relatively low extent of external contamination compared to feathers formed earlier. However, it was unknown how much time elapsed between the condor's death and its discovery that may have added to the feather's exposure to the environment. Results revealed the lowest lead concentrations in the oldest part of the feather, rising sharply in the youngest part of the feather to reach values several times the original concentrations. This pattern of lead concentrations is unlikely to have arisen from external contamination, which would produce a pattern opposite to that seen here, with lead concentrations highest in the outermost, or oldest part of the feather. In addition, the increase in lead levels corresponds with a distinct change in isotopic ratios, indicating an exposure to a novel source of lead during a specific time period. These ratios agree with those found in liver and kidney of the carcass, where exposure is limited to ingested lead. For these reasons, the source of lead measured in this feather is likely to be from ingestion.

Notable in this feather, isotopic ratios of ^{207}Pb:^{206}Pb rose with lead levels. Ratios associated with lead concentrations below 40 ppm ranged from approximately 0.720 – 0.790, markedly different than reported ratios from environmental sources or food items reported by the authors (0.8253 – 0.8394 and 0.8338 – 0.8453, respectively). A spike in lead levels from 50 – 90 ppm was associated with isotopic ratios of 0.800 – 0.810. While these values are consistent with those measured in ammunition, rises in isotopic ratios can also generally be associated with environmental ratios of lead as well as dietary items of condors. However, as lead shot pellets were recovered post-mortem from the stomach [or ventriculus] of this bird, this direct evidence of exposure to ammunition confirms its contribution to elevated lead levels in this individual.

Finkelstein et al. (2010) expanded upon the work of Church et al. (2006) with more extensive analysis of lead in the feathers, blood, and tissue of six condors chosen to represent three scenarios: a well-documented lead exposure event (condors observed feeding on a carcass from which lead bullets were recovered), mortalities attributed to lead poisoning, and routine exposure monitoring. For two condors with known exposure, feathers showed a spike in lead concentrations and a drop in isotopic ratios that correspond with the timing of the feeding event. Isotopic ratios of bullet fragments recovered from the scavenged carcass were similar to ratios in blood collected near the time of feeding. For the two condors that died from lead poisoning, feathers concentrations showed a spike at the time of poisoning that corresponds with a change in isotopic ratios. However, for one of the condors, isotopic ratios rose instead of declined, indicating that exposure may have derived from a source other than ammunition. In each case, isotopic ratios in blood, liver, and kidney corresponded with the acute exposure event, and ratios in bone – the site of long-term storage of lead – corresponded to pre-release ratios, indicating not only a change in concentration of lead, but in the source itself. Of three condor feathers collected during routine monitoring, two had increased lead concentrations that corresponded with shifts in isotopic ratios, while the third gave ambiguous results, showing decreasing lead concentrations from older to newer parts of the feather, with no corresponding change in isotopic ratios.

Where atmospheric deposition of lead is responsible for an increase in lead concentrations in feathers, we would expect to see not only a pattern of increasing concentrations in older parts of

feathers, but a corresponding isotopic signature in the lead. For most of the feathers analyzed in these studies, acute rises in concentration tended to correspond with ratios that were lower than environmental sources. Abiotic sources (dust, snow fed lake water, urban aeroseols, environmental lead, river water) measured in California cited by Church et al (2006) fell within the range of 0.8338 – 0.8453. This is similar to atmospheric concentrations measured in California cities in the 1990's (0.8418 – 0.8628) (Bollhöfer and Rosman 2001). While this represents only a small sampling of atmospheric lead, it is noteworthy that spikes in lead concentrations measured in feather tended to drive isotopic ratios below the ranges measured in these studies.

In a study of Andean condor feathers from the Argentine Patagonia region, Lambertucci et al. (2011) compared lead concentrations and isotopic ratios to seven types of ammunition obtained from retail stores and hunters in the study area. Molted feathers (N=152) were collected from the base of cliffs where condors congregate to roost. Isotopic ratios from ammunition fell into two ranges, averaging 0.817 for big game hunting, and 0.857 for hare hunting. The higher isotopic ratios were indistinguishable from background ratios for this region, and most feathers with low concentrations fell within this range. While isotopic ratios in ammunition were not predictive of lead concentrations in feathers, isotopic ratios differed between feathers with high (>4 ppm) and low (<4 ppm) concentrations, indicating that elevated lead resulted from a novel, possibly anthropogenic, source of lead. While feathers were rinsed prior to analysis, the possibility of external contamination was not examined by the authors.

Discussion Points: Use of stable isotopes to identify sources of lead in wildlife

- Isotope ratios can vary for any particular source of lead (e.g., ammunition, dietary items, paint) and measured ranges tend to increase with sample size.
- When used with other lines of evidence such as observations, behavioral ecology, or recovery of ingested items, isotopic ratios can help to confirm the source of the lead. For example, isotopic ratios in blood of exposed birds and a known source of exposure (e.g., bullet fragment or paint chip removed from bird) will be highly similar and can confirm

that lead was absorbed from that source into the bird's tissue, and where applicable, resulted in poisoning.

- Changes in isotopic ratios in avian tissues can reveal when a novel source of lead has been introduced into the diet. A noteworthy example is the decline in ^{207}Pb:^{206}Pb ratios associated with increased lead exposure as condors transition from captivity to the wild.
- Serially sampling of feathers can provide a more refined view of lead exposure in an individual over time, as changes in isotopic ratios can be useful to define when an elevation or decline in lead concentrations is associated with a change in the source of lead. When interpreted with caution, adult or fully grown feathers can be useful tools for analyzing changes in lead exposure particularly via the coupling of lead concentrations and isotopic ratios.

Alternate sources of lead as potential exposure routes

While multiple lines of evidence support the conclusion that scavenging birds are exposed to lead by ingesting spent ammunition, as previously noted, there are numerous sources of lead that contribute to its availability on the landscape. Herein, we examine several alternate sources of lead and evaluate the likelihood of exposure for scavenging birds.

Microtrash and other metal objects

The ingestion of metal objects by birds resulting in lead exposure that cannot be positively attributed to ammunition or fishing sinkers has been documented in only a limited basis. The first documentation of lead poisoning from the ingestion of a metal object of unidentified origin was in the Mississippi sandhill crane (*Grus Canadensis pulla*) (Franson and Hereford 1994).

One scavenging species that has been documented to ingest foreign anthropogenic material, or "microtrash," is the California condor. These objects can be collected from condor nests, where adults regurgitate ingested material for the consumption of nestlings. While it has been hypothesized that condors may not collect trash until feeding nestlings (Walters et al. 2008), a systematic search of roost sites for regurgitated pellets has not been conducted to look for

evidence of microtrash ingestion during non-breeding periods (Mee et al. 2007). However, inspection of trash from nest sites can elucidate the spectrum of materials collected by the birds.

Nests of seven condor pairs, representing 11 breeding attempts, were monitored from 2001 – 2005 (Mee et al. 2007). Nest-floor substrates were collected at the end of breeding attempts or opportunistically during nests visits using a fine-mesh window screen that allowed for collection of material greater than 1 mm^2. Of 650 microtrash items recovered, 148 (22.8%) were metallic, including items such as bottle-tops, washers, ammunition casings, and electrical wiring (Mee et al. 2007). No objects that could be associated with alternate sources of lead (e.g., wheel weight) where specifically identified. Necropsies were performed on thirteen dead nestlings from the California population between 2001 and 2009 (Rideout et al. 2012). Of these, six nestlings that were either diagnosed with trash ingestion as the cause of death, or containing microtrash, also had hepatic lead concentrations measured. None had detectable lead concentrations despite the presence of metallic objects such as nuts and washers (Rideout et al. 2012). Though these studies represent only a subset of condors, the analyses show no indication that condors are exposed to lead objects in the accumulation of microtrash.

The degree of trash ingestion by condors appears to vary between the southern California population and the Arizona population. One juvenile (5 years old) and one adult (11 years old) from the Arizona population were reported to have died from trash ingestion between 1996 and 2010, neither showing detectable levels of hepatic lead (Rideout et al. 2012). However, contrary to the California population, trash ingestion by chicks in Arizona is rare and generally does not contribute to mortality (Walters et al. 2008). The carcass of one chick collected in 2011 contained microtrash, but it was not believed to be the cause of death (USFWS 2012). The difference in trash ingestion is believed to be a function of foraging practices. Condors in the Arizona population forage more on natural food and further away from provisioning sites as compared to condors in the southern California population (Walters et al. 2008). These foraging sites are more remote from anthropogenic activity and believed to contain less trash. The necessity to travel further distances may also leave less time for trash collection (Walters et al. 2008). Yet both the California and Arizona populations suffer from mortality due to lead

exposure, which cannot be readily explained by the ingestion of microtrash. Although condors have been observed ingesting metal objects, these cases are a select few and cannot account for the widespread lead exposure that is occurs in the California and Arizona population.

Paint

Ingestion of lead-based paint from buildings has been documented as a source of lead exposure for birds. Perhaps the most widespread and studied instance of this exposure occurred on Midway Atoll, where lead-poisoned Laysan albatross (*Phoebastria immutabilis*) chicks were associated with proximity to historical buildings and discovered with paint chips in the proventriculus (Sileo and Fefer 1987, Work and Smith 1996). Finkelstein et al. (2003) performed lead isotopic ratio analysis on blood lead from asymptomatic and symptomatic chicks on Midway, as well as from soil and paint chips found in or near the nests of sampled chicks. While there was wide variation in isotopic composition of samples in both blood ($^{207}Pb/^{208}Pb$ = 0.838 - 0.908) and paint ($^{207}Pb/^{208}Pb$ =0.8240-0.887), ratios from blood of chicks exhibiting droopwing, a sign of lead toxicosis, were significantly related to the ratios of lead in paint chips collected from nests. No relationship was found between isotopic ratios of blood samples and soil samples. Few other examples exist of birds exposed to lead through this route. A group of 13 captive sandhill cranes (*Grus canadensis*) exhibited signs of lead poisoning after being moved to a facility that was later discovered to have lead-based paint on the walls, though this event occurred indoors with a non-wild population (Kennedy et al. 1977).

Lead-based paint from an inactive fire lookout tower has been identified as a source of lead exposure for a subset of California condors (N=5) (Finkelstein et al. 2012). These cases were confirmed both by observations of condors roosting on or near the tower, and by matching $^{207}Pb/^{208}Pb$ isotope ratios between blood of these condors and paint chips sampled from the tower (Finkelstien et al. 2012). Lead-based paint can have a wide range of lead isotopic ratios (Rabinowitz and Hall 2002, Finkelstein et al. 2003). However, by matching isotopic ratios from blood of lead-exposed birds to the specific source suspected of this exposure based upon condor movements and behavior, it is possible to narrow the range of isotopes to the paint used on the roosting tower. Outside of this unique exposure pathway for condors to lead-based paint, there is

no clear pathway for condors, which are highly studied and tracked through both radio transmitters and visual observation, to ingest other sources of lead-based paint. Condor association with fire lookout towers in central California is a rare occurrence, as indicated by tracking data (Finkelstein 2012), and reports are not available of condors associating with other historical structures. Though we cannot definitively dismiss the possibility, there ~~is~~ are no examples from the literature of birds achieving elevated lead concentrations from chronic low-level exposure to various sources of lead-based paint; each example has been tied to a known source providing the exposure (i.e., buildings on Midway, fire lookout tower).

Mine tailings

Lead mines are throughout the nation, with the largest concentration located in southeast Missouri, known as the "Old Lead Belt". The process of extracting lead from the earth produces waste tailings that are typically deposited on the landscape if environmental clean-up or storage measures are not followed or in place. Lead tailings have polluted water and sediment in lakes and rivers, and soil in terrestrial habitats. Lead exposure to wildlife can occur when there is direct contact with the tailings dermally or by ingestion, or when an animal eats another animal that has been exposed to the lead tailings.

One of the most published examples of lead poisoning to waterfowl from mine waste is in the Coeur d' Alene River basin in northern Idaho (Chupp et al. 1964). Lead from mining and smelting activities in the Coeur d' Alene River system in northern Idaho was associated with mortality to tundra swans (*Cygnus columbianus*) from the ingestion of the lead contaminated sediment and plants (Blus et al. 1991). A more recent publication suggested that lead contaminated sediment was the main cause of mortality in 77% of 285 waterfowl carcasses (mostly tundra swans) found sick or dead from 1992-1997, with 7% of deaths attributed to lead poisoning from ingested shotgun shells (Sileo et al. 2001).

Waterfowl are typically thought to be the most common species exposed to lead from mine waste because of their physiology and foraging behavior, however, passerines can be at risk through secondary exposure routes. Hansen et al. (2011) investigated lead exposure from mine

wastes and soil ingestion rates of three species of ground-feeding songbirds, American robin (*Turdus migratorius*), Swainson's thrush (*Catharus ustulatus*), and song sparrow (*Melospiza melodia*), at the Coeur d'Alene River basin at a reference area, moderately contaminated sites, and at highly contaminated sites. The highest soil ingestion rates and the highest blood lead concentrations were found in the American robin at the highly contaminated sites. At these sites, 18% of the 204 American robins had severe poisoning, 52% had clinical poisoning, and 24% had subclinical poisoning. Soil ingestion accounted for almost all of the species' exposure to lead.

Exposure from lead mine wastes to scavenging or predatory birds can originate from secondary exposure from dietary sources. For example, 218 blood samples were collected from Eurasian eagle owl chicks (*Bubo bubo*) between 2003 and 2007 at a reference and contaminated site from an abandoned mine. Blood lead levels were significantly higher in the chicks at the mine site compared to chicks at the reference site, suggesting dietary exposure to lead (Gomez-Ramirez et al. 2010). Similar exposure pathways for bald eagles or other raptors that feed on small mammals and birds cannot be ruled out, although there is no documentation of this in the literature. The likelihood of scavenging birds that normally feed on live or dead animal matter to directly ingest mine tailings is low.

Shooting ranges

Shooting ranges, either military or for recreation, are a source of spent lead ammunition and soil contamination. There are approximately 3000 military shooting ranges and 9000 nonmilitary sites in the United States (USEPA 2005). Birds foraging in these areas or in wildlife habitats adjacent to the shooting ranges can be directly exposed by the uptake of spent pellets incidental to grit ingestion, or by ingesting contaminated soil or food. A study investigating the toxicity of lead in soil collected from a small arms-range found that rock doves accumulated lead in the blood, tissues, and feathers when dosed with soil, with an accompanying increase in erythrocyte protoporphrin at blood levels > 0.50 ppm (Bannon et al. 2010).

Species most likely to be at risk through direct ingestion of spent lead or contaminated soil on or near shooting ranges are waterfowl, mourning doves, and other species that forage on the ground

or use grit to help aid in digestion (i.e., quail, grouse, pheasant). Roscoe et al. (1989) describes lead poisoning of northern pintails foraging in a tidal meadow contaminated with lead from a trap and skeet shooting range. Passerines that forage on the ground near a small-arms range are documented to have greater lead exposure compared to those in ~~a~~ wildlife habitat removed from the small-arms range (Vyas et al. 2000). Passerines collected in a woodlot adjacent to a small-arms range had significantly higher lead exposure, measured by free-erythrocyte protoporphyrin levels, compared to those at a reference site (Vyas et al. 2000). However, the likelihood of scavenging birds that normally feed on live or dead animal matter to directly ingest contaminated soil or spent lead shot from the ground is low. Scavenging birds such raptors can be exposed from eating food items (i.e. waterfowl and other bird species, small mammals, invertebrates) that have directly ingested spent lead shot or contaminated soil from shooting ranges.

Discussion Points: Alternate sources of lead as potential exposure routes

- Numerous sources of lead contribute to its availability on the landscape, including lead-based paints, mining operations, and shooting ranges.
- These routes cannot be ruled out as potential sources of lead exposure to scavenging birds, however, foraging and behavioral traits of scavenging birds make them less likely.
- There are few documented cases of lead poisoning to scavenging birds from alternate exposure routes as compared to lead ammunition.
- Studies to date have not found the ingestion of foreign anthropogenic material or "microtrash' to be a significant source of lead exposure.

Case Studies of Lead and Wildlife

Below we discuss two regionally specific case studies where lead poisoning from ammunition has been well-documented through multiple lines of evidence: the bald eagle in the Great Lakes, and the California condor throughout its range (Figure 9). In describing these cases, we apply the general concepts discussed above more specifically to the species under consideration.

Figure 9. Location in the U.S. of the two case study examples, the Great Lakes bald eagle population and the California condor population.

Case study 1 – Great Lakes bald eagles

Bald eagle populations in the United States are divided into two large populations; the Alaska population and the continental population (USFWS 2009). The continental population is divided

into management units that include the Great Lakes, Lower Mississippi, Mid-Atlantic, Northern Rocky Mountains, Pacific, Rocky Mountains and Plains, Southeast, and Southwest (USFWS 2009). The number of mapped nests of the breeding pairs for the continental population is 15,813 with 40% (6,375) in the Great Lakes management unit (USFWS 2009). The Great Lakes region is also an important habitat for bald eagles in the winter (Millsap 1986, Steenhof 2002) and some of the highest mid-winter bald eagle counts have been recorded for the central United States (Steenhof et al. 2008). During the winter season, bald eagles congregate and forage along lakes and tributaries to feed on fish and waterfowl. As winter progresses in the Midwest and ice freezes over many of the open waterways, bald eagles opportunistically search for other food sources that are available on the landscape. One readily available food source in the winter is discarded offal from hunter-killed white-tailed deer or deer that were shot and not retrieved (Harper et al. 1988, Ewins and Andress 1995, Lang et al. 2001, Cruz-Martinez 2012; e.g., Figure 10).

Figure 10. Picture of a juvenile bald eagle feeding on a white-tailed deer carcass with an adult bald eagle and an American crow at the Lost Mound Unit of the Upper Mississippi River National Wildlife and Fish Refuge.

Photo courtesy of USFWS Upper Mississippi River National Wildlife and Fish Refuge, 7071 Riverview Rd. Thomson IL 61285.

The diet of the bald eagle is broad and can vary by season. In summer, bald eagles feed ~~more~~ preferentially on fish and birds (Dunstand and Harper 1975). In winter, several studies have documented a shift in food preferences that take advantage of the greater availability of deer. On 949 feeding observations of bald eagles in New Brunswick, Canada, white-tailed deer and deer offal accounted for 40% and 30% of the diet, respectively (Stocek 2000). Of 339 feeding observations of bald eagles wintering in the lower Great Lakes basin, 47% were on carcasses of white-tailed deer (Ewins and Andress 1995). Analysis of eagle castings or pellets of items that are not fully digested and therefore regurgitated also support a dietary preference for deer in the

winter. White-tailed deer remains were found in 67-72% of the regurgitated castings of bald eagles wintering along the St. Lawrence River, representing the most frequently detected dietary item (Lang et al. 2001).

During the hunting season, it is common practice to field dress game and discard the internal organs and tissues on the landscape, leaving a lighter carcass to transport out of the field. It is well documented that lead fragments and particles in the offal discarded by deer hunters are available to bald eagles in the Midwest (Grund et al. 2010; Strom et al. 2006; Cruz-Martinez et al. 2012). The extent of the exposure from lead in offal varies according to the number of deer killed with lead ammunition that entered the visceral cavity and fragmented into the organs and tissues. The resulting exposure in bald eagle and other scavenging birds to lead from ammunition has been recognized as a problem in the United States (Friend et al. 2009; e.g., Table 5) and worldwide (Pain et al. 2009).

Lead poisoning progresses from inability to fly, starvation, and mortality (Franson and Pain 2011). Though many of the moribund or dead bald eagles likely go undetected (Vyas 1999), a number in the Great Lakes have been collected by the public and wildlife officials. Most of the collected bald eagles are sent to wildlife rehabilitation facilities where trained biologists attempt to treat the birds that are lead poisoned. Dead eagles can be sent to wildlife diagnostic laboratories where tissues are chemically analyzed and full necropsies performed to determine the cause of death.

Examples of Lead Exposed Bald Eagles in the Great Lakes

Minnesota: A major wildlife rehabilitation facility in Minnesota processed 1,227 moribund bald eagles from between 1996 and 2009 with 331 (27%) of the birds having lead concentrations in the blood that are consistent with poisoning (Cruz-Martinez et al. 2012).

Iowa: Wildlife rehabilitation facilities in Iowa processed 62 moribund bald eagles between 2000 and 2008 with 39 (62.9%) of the birds having concentrations >0.2 ppm in the blood or >6 ppm liver (Neumann 2009). The radiographs of seven of the 59 (12%) birds admitted in Iowa showed fragments in the digestive tract. Some of the birds were admitted for traumatic injuries and had

elevated lead exposure. While it is often difficult to establish a definitive relationship between the detection of sublethal concentrations of a contaminant and proximate cause of death, some sublethal effects may render birds more susceptible to other causes of death. Most of the lead poisoned birds were admitted between the months of September and April, overlapping with the deer hunting season.

Wisconsin: The State of Wisconsin diagnostic laboratory processed 583 dead bald eagles between 2000 and 2008 with 87 (15%) diagnosed with lead poisoning (Strom et al. 2009). This study associated bald eagle exposure rates with the hunting season in Wisconsin.

Upper Mississippi River and Surrounding Great Lakes States: An investigation conducted by the USFWS found that 35 of the livers from the 58 (60%) dead bald eagles found between 2009 and 2012 on the Upper Mississippi River National Wildlife and Fish Refuge (Refuge) and adjacent areas in the States of Iowa, Minnesota, and Wisconsin had detectable concentrations of lead and 22 (37.9%) had concentrations consistent with lead poisoning (>6 ppm wet weight) (Britton et al. 2013 *in review*). Of the bald eagles that had associated date of death information (month, day, and year) 41% (16) had a detection date overlapping with the hunting season or within one month after the season closed, and 4 of these bald eagles had concentrations consistent with lead poisoning. Due to concern about lead exposure to wildlife on the Refuge, a voluntary voucher program where hunters from controlled harvest events could obtain non-lead ammunition for use on the Refuge was tested. Most of the hunters obtained free vouchers for non-lead ammunition and many of them used non-lead ammunition.

Eagle Loss and Take Limits

The total number of bald eagles from the ad hoc reporting at wildlife rehabilitation facilities and wildlife diagnostic laboratories that died of lead poisoning between 1996 and 2012 was 585 or about 37 bald eagles per year from the Great Lakes management unit. Not all of the reporting for lead poisoning cases is provided or up to date and not all of the lead poisoned eagles are found or collected (Vyas 1999). Therefore, the value is likely an underestimation of the total number of bald eagles that die each year from lead poisoning (i.e, there are no data available from the State

of Illinois). The annual individual bald eagle take threshold which is authorized by the Bald and Golden Protection Act (16 U.S.C. 668c; 50 CFR 22.3) and set by the USFWS is 224 bald eagles per year for an estimated population size of 27,614 for the Great Lakes population. This population size does not necessarily include the total number of bald eagles that may winter in the Great Lakes management unit. This take value is based on the maximum sustainable yield that was developed for the eagle permit program of the USFWS (USFWS 2009).

The rate and trends of lead poisoning should be monitored against the backdrop of the other causes of mortality and permitted take for the management of continental bald eagle population. Information in USFWS (2009, Table C.3. page 143) may be used to help us understand if the level of bald eagle take from lead poisoning is significant for the continental bald eagle population. Moribund and dead eagles are considered to contribute to take, which is defined "to pursue, shoot, shoot at, poison, wound, kill, capture, trap, collect, destroy, molest, or disturb" under the Bald and Golden Eagle Protection Act (16 U.S.C. 668c; 50 CFR 22.3). The loss rate of dead birds may be up to 98% in the wild, depending on season, location, and species (Prosser et al. 2008). Due to the conspicuous nature of the bald eagle and its increased chances of being detected and reported, we assume a lower maximum loss rate of 70% as suggested by Ferrer (1991). Using this rate, a loss of 37 bald eagles per year due to lead poisoning based on tentative data from wildlife rehabilitation facilities and wildlife diagnostic laboratories may actually equal a loss of about 63 (37 + [37 X 0.70]) lead poisoned bald eagles per year. This annual loss rate of 63 bald eagles (plus from what is loss in Illinois) due to lead poisoning is about 28% of the annual maximum sustainable yield of 224 bald eagles.

Discussion Points: Case study 1 - Great Lakes bald eagles

- There is an alignment of conditions in the central United States that contribute to increased mortality rates for the bald eagle (habitat and seasonal variables, timing of hunting season).
- In the winter months, a significant portion of the bald eagle continental population plus birds that have migrated from Canada compete for food in the Great Lakes.

- Scavenging on deer offal is common for wintering bald eagles.
- Data from the wildlife rehabilitation facilities and wildlife diagnostic laboratories confirm that lead exposure make up a significant portion of moribund and dead eagles show annually.
- Data show a temporal trend between bald eagle admittance to a rehabilitation facility or diagnostic laboratory and deer hunting seasons.
- The rate of lead poisoning in recovered moribund bald eagles is likely an underestimation of the problem, because many moribund and dead birds go undetected by the public and wildlife officials.
- Lead poisoning alone may account for over 25% of the annual maximum sustainable yield of 224 bald eagles for the Great Lakes population.

Case study 2 - California condor

The California condor, once distributed throughout North America, is now considered one of the rarest bird species in the world. In the 1950s, the population of California condors only existed in central California. By 1982, there were 23 individuals left in the wild based on field surveys and an intensive captive rearing program was initiated. The cause for the initial decline remains unknown, but documentation of mortality from intentional poisoning, egg collecting, intentional shooting, and lead poisoning are possible contributors (Grantham 2007). By 1987, all birds had been removed from the wild to initiate the captive rearing program. The captive rearing was successful in producing enough individuals to be released at historic condor range sites in southern California in 1992. The first condors were released in Arizona in 1996, in central California in 1997, and in Baja Mexico in 2003 (Grantham 2007).

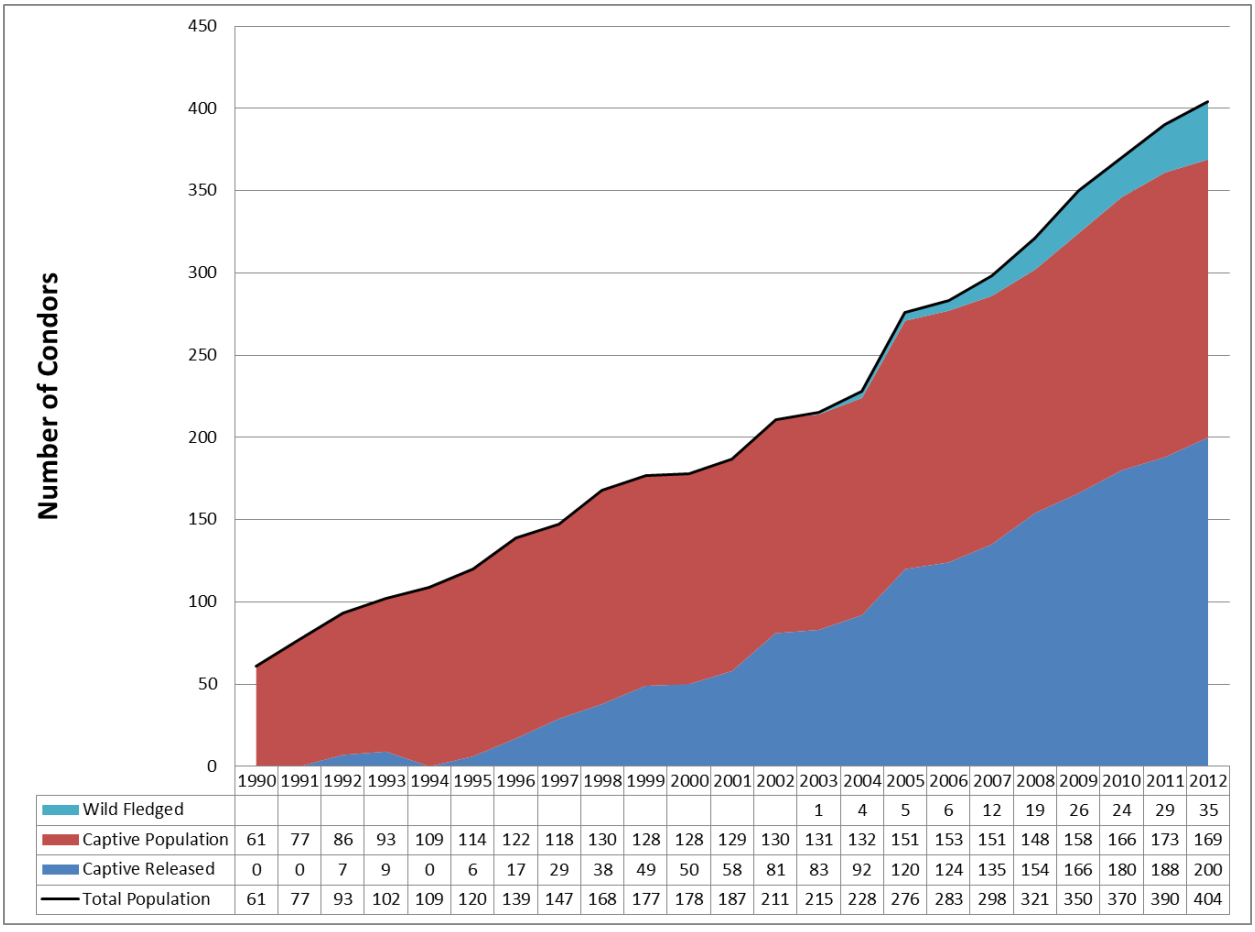
The population's reproductive rates remain low at all of the release sites. Extensive conservation efforts towards recovery continue, including annual captive rearing and release, bi-yearly trapping and extracting of blood for lead testing, treatment of condors that exhibit elevated lead levels with chelation therapy, supplemental feeding, radio tracking, and daily monitoring. The population remains endangered despite these efforts due to the high mortality that surpass the low productivity rates. Although the wild free-flying population is supplemented with captive

raised individuals and lead exposed condors are treated with survivors released back into the free flying population, the wild population has yet to exhibit positive growth (USFWS 2012).

California condor population status

The condor population in California and Arizona has steadily increased over the past two decades, chiefly from the propagation and release of captive-reared individuals. As of December 2012 there were 404 individuals in total (235 free-flying birds in the wild, 169 in captivity). The first wild fledged condor occurred in 2003 in Arizona, 17 years after the inception of the recovery program there, and at the end of 2012, 35 wild hatched chicks survive (USFWS 2013e).

Figure 10. Year-end California condor population figures from1990 – 2012 (USFWS 2012, 2013e).



	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012
Wild Fledged														1	4	5	6	12	19	26	24	29	35
Captive Population	61	77	86	93	109	114	122	118	130	128	128	129	130	131	132	151	153	151	148	158	166	173	169
Captive Released	0	0	7	9	0	6	17	29	38	49	50	58	81	83	92	120	124	135	154	166	180	188	200
Total Population	61	77	93	102	109	120	139	147	168	177	178	187	211	215	228	276	283	298	321	350	370	390	404

Recovery plan objectives

Free-flying condors in the wild consist of 129 in California with 12 active breeding pairs, 78 in Arizona with 6 active breeding pairs, and 28 birds in Baja California (USFWS 2012, 2013e). Although the population as a whole has experienced an increase in wild-hatched condors, it is still not considered reproductively sustainable as defined in the USFWS Recovery Plan. The Recovery Plan lists conditions that must be met in order for the species to be down listed from endangered to threatened status. The minimum criterion is the maintenance of at least two wild populations and one captive population (USFWS 2012). The five conditions that are required from the Recovery Plan, in addition to the minimum criterion, are that two of the free-flying populations:

(1) must each number at least 150 individuals;

(2) must each contain at least 15 breeding pairs;

(3) must be reproductively self-sustaining with a positive rate of population growth.

(4) must be spatially disjunct and noninteracting; and

(5) must contain individuals descended from each of the 14 founders.

The Recovery Plan was written in 1996 and predicted that the criteria for down listing would be met by 2010 (USFWS 1996); a goal that is far from being achieved. The current level of mortality is documented to be from anthropogenic causes with the leading cause lead exposure and poisoning (Rideout et al. 2012). Microtrash ingestion, predation, depredation, and exposure to DDT/DDE in the coastal population provide additional mortality events. The population's high rate of mortality and low reproductive success has prevented the California condor from achieving recovery goals.

Lead exposure pathway for condor

If vulnerability to a contaminant can be described by the exposure potential and sensitivity of a species or individual, the California condor may be uniquely susceptible to poisoning from lead ammunition. Lead is available to the free flying California condors from a variety of sources (Fry and Maurer 2003). The sources include past deposition from vehicle exhaust when leaded fuels

were in use, atmospheric deposition from industrial air emissions, lead based paint remaining on roads or structures, mining operations, waste disposal sites, and spent ammunition from hunting. The kinds of spent ammunition include shotgun pellets and rifle projectiles and may be available in the environment at shooting ranges, embedded in wounded or crippled game that later die, and in the carcasses or offal piles from field dressed game discarded by hunters.

While exposure to other sources of lead cannot be definitively ruled out, and some have been documented in select cases (e.g., lead paint; Finkelstein et al. 2012), the diet and foraging behavior of the California condor makes it particularly vulnerable to exposure to ammunition as a source of lead, especially from hunter-killed carcasses left in the field. As obligate scavengers, California condors feed only on carcasses and often choose large mammals, including those which have been killed by hunters. Condors in interior habitats rely heavily on mule deer (*Odocoileus hemionus*), tule elk, pronghorn antelope (*Antilocapra americana*), feral hogs, and domestic ungulates, but may also take smaller mammals such as rabbits (USFWS 2012, 2013e). For condors residing in coastal areas, marine mammals such as whales (Order *Cetacea*) and sea lions (*Zalophus californianus*) are included in the diet field (USFWS 2013e). While condors may feed individually, their reliance on visual clues to locate food, especially the presence of other scavengers at a carcass, predisposes them to group feeding, which is a documented part of their foraging strategy. Group feeding can result in a single carcass containing lead fragments, or another contaminant, affecting multiple scavenging individuals. Condors maintain wide-ranging foraging patterns throughout the year, allowing for opportunistic feeding in accordance with food supplies. Currently, California condors predominately forage in open terrain of foothill grassland and oak savanna habitats, but have also been observed less commonly feeding in more wooded areas.

The total harvest of game and varmints from the counties in the range of the free flying California condors in California can be quite high and was estimated at 106,049 animals in 2001 (Fry and Maurer 2003). That total included 30,000 cottontail rabbits, nearly 20,000 wild pigs, over 20,000 coyotes, over 15,000 jackrabbits, several thousand tree squirrels, several thousand deer, plus an uncounted number of ground squirrels. Carcasses from these kills may remain in the field, embedded with the ammunition used in the hunt. However, in recent years, measures

have been taken to reduce the amount of lead ammunition available to scavengers from this pathway in the range of the free flying California condor populations (USFWS 2013e). In 2003, the State of Arizona instituted a voluntary program for non-toxic ammunition or the removal of offal if harvested with lead ammunition. In 2012, the State of Utah instituted a program similar to Arizona. In 2007, the State of California passed a law that banned the use of lead ammunition in the range of the California condor for big game and varmint hunting. While likely to have contributed to a decline in availability to scavengers, lead ammunition may still be present in the range of the condor from non-covered uses (upland game hunting, nuisance animal depredation, and dispatching domestic livestock), poaching, and non-compliance. From studies of other scavengers foraging on contaminated carcasses, even very small numbers of afflicted carcasses (<1%) can affect disproportionately large numbers of foraging birds (Green et al. 2004). Thus, anything less than complete removal of lead ammunition from the condor's habitat could result in a high frequency of exposure in the population. Accordingly, no decline in blood lead concentrations was found in condors residing in California between the pre-lead ammunition ban years 2006 and 2007 compared to the post-lead ammunition ban years of 2009 and 2010 (Finkelstein et al. 2012).

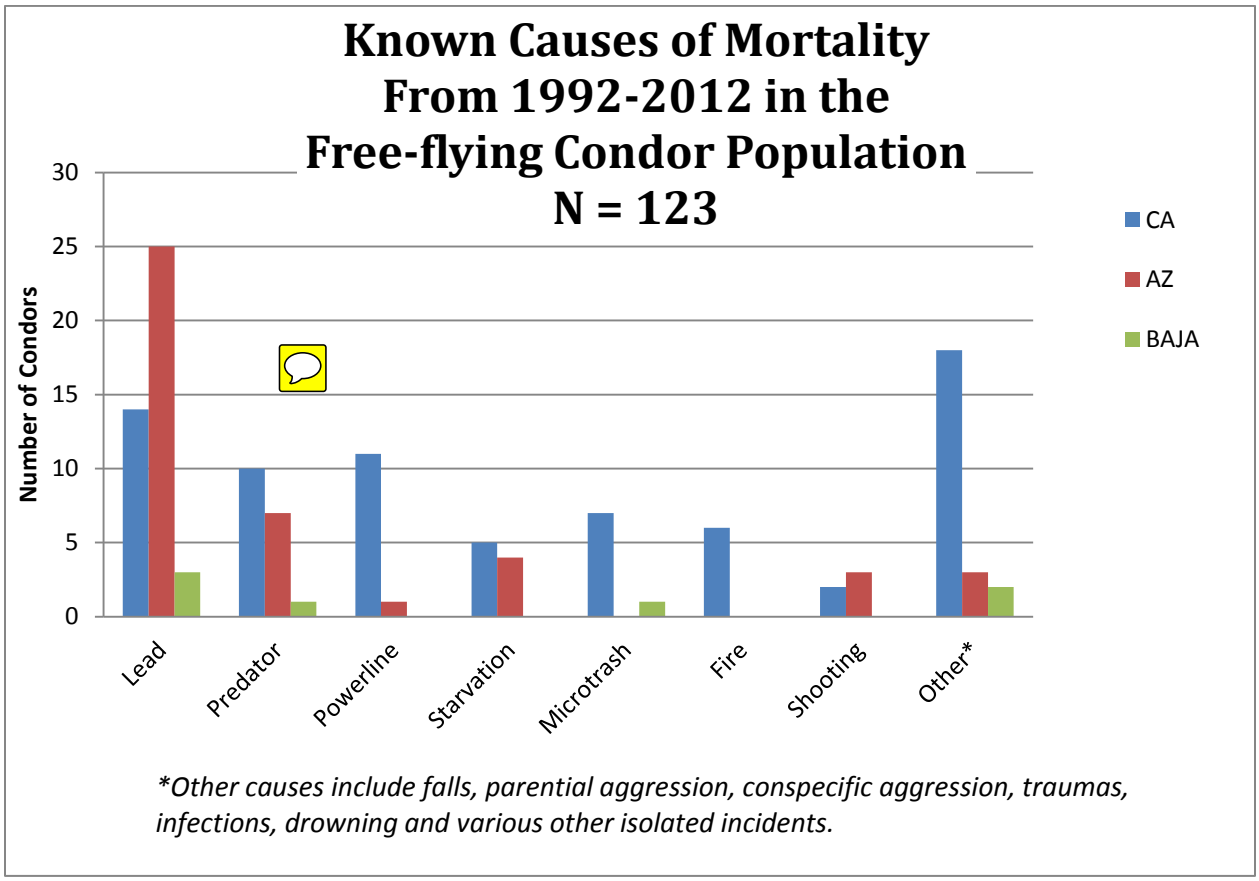
Once exposed to lead fragments or particles while scavenging, the digestive system of birds promotes their absorption into the circulatory system. Raptors species contain physiological traits such as low stomach acid and enhanced digestion that may increase absorption of lead from the gastrointestinal tract (Duke 1995, 1997). Condors may possess even more inherent sensitivity than other scavenging species, as evidenced by controlled laboratory studies of the closely related Andean condor (Pattee et al. 1981). Absorbed lead can cause sublethal effects that may enhance vulnerability to other threats, or cause direct poisoning that progresses from inability to fly to adverse physiological effects, starvation, and ultimately mortality (Franson and Pain 2011).

Lead-related mortality in condors

Rideout et al. (2012) summarized condor mortality cases for the California and Arizona population from the inception of the reintroduction project in December of 1992 to 2009 (Figure 11). At that time there had been 135 deaths out of a total population that (in 2009) consisted of

352 individuals (38%). Since 2009, there have been more condor deaths; however, in most cases data are not published and readily available about the details of the mortalities. The Rideout (2012) paper summarizes 17 years of data where 98 carcasses submitted for necropsy included adults and juveniles (85), and nestlings (16) of which 76 had a definitive mortality cause. Out of these cases, 53 (70%) were attributed to anthropogenic causes. From the 85 juvenile and adults that were submitted for necropsy, 65 had known mortality causes. Anthropogenic causes attributed to 44 of the 65 cases, with 23 (52%) confirmed cases of lead toxicosis with 9 cases in California, 12 known mortality cases in Arizona, and 3 cases in Baja California. In these cases, lead toxicosis was diagnosed either by blood levels (antemortem) >0.50 ppm or post-mortem liver or kidney concentrations of >6.0 ppm wet weight. Some cases had additional supporting evidence such as lead ammunition or fragments in gastrointestinal tracts and histological findings such as Kupffer cell erythrophagocytosis and hemosiderosis.

Figure 11. Known causes of mortality among free-flying condors since reintroduction into the wild in 1992 until 2009 (Rideout et al. 2012) and through 2012 (USFWS 2012).



In California, 3 of the 9 lead poisoning cases were based on liver lead concentrations of 11, 21, and 26.4 ppm wet weight. Five cases were for condors that received chelation therapy but did not recover. Chelation therapy results in lowered lead concentrations in tissues, and as such, these condors had blood lead concentrations below the threshold level for toxicosis at the time of death (all under 3.7 ppm wet weight) as expected. Data is not publically available on the blood lead concentrations at the time of admittance for these 5 condors, so it is unknown how elevated the original levels were. In 3 of these cases, visceral gout was associated with the chelation treatment. Lastly, condor 422 was diagnosed as having lead toxicosis and did not recover after receiving therapy. Based on feather analysis, this condor was exposed to lead four times since it

was released 180 days earlier. One of these exposure events was severely elevated with blood lead levels at 3.00 ppm.

In Arizona, 7 of the 12 lead poisoning cases were based on liver lead concentrations ranging from 17 - 62 ppm wet weight. Metallic fragments (2 lead shotgun fragments, 1 lead ammunition fragment) were identified in the gastrointestinal tract of 3 of these individuals. The remaining 5 lead poisoning cases were for condors that received chelation therapy but did not recover. These condors had blood lead concentrations below the threshold level for toxicosis (all under 5.2 ppm wet weight) at the time of death as a result of chelation therapy. Data is not publically available on the blood lead concentrations at the time of admittance for these 5 condors, so it is unknown how elevated the original levels were. Metallic fragments were detected in the gastrointestinal tracts of 3 of these condors, though were not identified as lead or another metal.

In Baja, one condor that died of lead poisoning ingested a 22-caliber bullet after being fed a donkey carcass that unknowingly had the bullet embedded in it. The other condor was brought in having high lead levels and treated with chelation therapy but did not survive. Visceral gout was associated with the chelation therapy.

Updated information from the USFWS Five Year Reviews for the California and Arizona population summarizes data up until 2011 or 2012. In California, since the start of the re-introduction program in 1992 to December of 2012, there have been 42 condor deaths out of 123 (34%) where a cause of death is known and the diagnosis is lead poisoning. Lead poisoning is the leading cause of mortality and is twice as high as the next mortality cause, predation (USFWS 2013e).

In Arizona, condors were first released in December of 1996 and by the end of 2011, 134 condors had been released into the wild and 15 chicks had been wild-hatched (USFWS 2012). Sixty-nine of these birds have died and 21 deaths have been attributed to lead poisoning (8 carcasses contained bullet fragments, 1 had a whole bullet, and 5 had shot pellets). Shotgun pellets and rifle bullet fragments in animal carcasses have been the primary sources of lead exposure to condors in Arizona and southern Utah. By the end of 2011, 73 free-flying condors existed in southwest population (USFWS 2012).

Lead exposure and morbidity in condors

Because California condors are being monitored and treated, simply analyzing lead-related mortality rates in the population does not fully capture the extent of lead exposure or poisoning that would occur without intervention. Instead, the rate of exposure and morbidity must be considered.

Finkelstein et al. (2012) tested blood samples multiple times throughout the year from up to 150 free flying condors in the California population between 1997 and 2010. This resulted in the collection of 1,154 blood samples that were tested for lead and stable lead isotope ratios. An average of 71% of the birds tested each year were exposed to lead above the selected background concentration of 0.10 ppm (determined by multiplying the mean blood lead concentration of pre-release birds, 0.0303 ppm, by a factor of 3). Thirty percent of blood samples collected each year indicated exposure to lead ≥0.20 ppm, indicative of subclinical health effects (>60 percent inhibition of ALAD). About 20% of the birds tested each year were exposed to lead concentrations ≥0.450 ppm, the threshold selected for chelation therapy to avert morbidity or mortality. Over the period between 1997 and 2010, 48% of the free flying birds had blood lead concentrations over this threshold. The use of isotopic analysis to identify possible sources of lead exposure revealed that 79% of the 150 free flying California condors contained lead with ratios that overlapped with those measured in ammunition by the researchers (Finkelstein et al. 2012). Other samples contained isotope ratios consistent with leaded paint from a tower where condors were observed roosting. The remaining isotopic ratios were not consistent with known sources of lead

Parish et al. (2009) tested blood samples for lead exposure multiple times throughout the year from up to 64 free flying condors in the Arizona population between 2000 and 2007. Depending on the year, an average of 48% to 95% of the birds tested was exposed to lead (threshold concentrations used not provided). Birds were held and monitoring when blood lead concentrations were >0.30 ppm. Chelation therapy was administered to between 4% and 70% of condors each year above this threshold whose blood lead concentrations continued to increase, or those with concentrations >0.60 ppm.

There was not a reduction in blood lead concentrations in the Arizona population of California which is similar to the observations in the California population of California condors after reduction in the use of lead ammunition, but potentially for different reasons (Parish et al. 2009). The potential reason for the continued lead exposure in the Arizona population of California condors provides an opportunity to see the temporal relationship to big game hunting with lead ammunition and the behavior of the California condors in that population. The California condors from Arizona would move into Utah highlands for feeding on the livestock carcasses that died naturally and would stay for the mule deer and elk hunting season for the available carrion from the hunter's activities. This resulted in a spike of blood lead concentrations during and just after the big game hunting season in Utah.

Monitoring of blood lead is likely to underestimate the frequency of exposure in the condor population. Blood is a short-term storage compartment for lead, with an elimination half-life of about 13 days (Finkelstein et al. 2012). Therefore blood sampling that occurs in intervals of weeks or months is likely to miss lead that has moved out of blood and into other tissues (e.g., liver or kidney). Furthermore, sampling of this nature is even more likely to miss peak values, as concentrations in blood may either be rising or falling at the time of collection, with no indication based on a single sample.

Discussion Points: Case study 2 – California condor

- The California condor population remains endangered despite extensive conservation efforts. High mortality surpasses the low productivity rates and the wild population has yet to exhibit positive growth.
- California condors feed only on carcasses including those which have been killed by hunters.
- Lead exposure to other sources cannot be ruled out; however, the diet and foraging behavior of the California condor makes it particularly vulnerable to exposure to ammunition as a source of lead, especially from hunter-killed carcasses left in the field.

- In Arizona and southern Utah, shotgun pellets and rifle bullet fragments in animal carcasses have been the primary sources of lead exposure to condors.
- In California, over the period between 1997 and 2010, 48% of the free flying birds had blood lead concentrations over the lead poisoning threshold.
- Measures to reduce the amount of lead ammunition used have likely contributed to a decline in lead availability, but lead ammunition may still be present in the range of the condor from upland game hunting, nuisance animal depredation, and dispatching domestic livestock, as well as poaching, and non-compliance of lead ban regulations.
- Mortality rates for condors may be even higher without current levels of intervention (monitoring and chelation therapy).
- Monitoring of blood lead is likely to underestimate the frequency of exposure in the condor population.

Toxicity of alternative metals used in ammunition

One way to break the exposure pathway of scavenging birds to lead ammunition is via the use of alternative metals in ammunition, provided that ammunition derived from these metals is nontoxic (i.e., does not cause sickness and death when ingested by migratory birds) (USFWS 2013a).

Nontoxic shot approval process

To determine the types of ammunition that are nontoxic and safe to use for waterfowl hunting, a comprehensive testing method was developed for the registration of alternative shot and shot coatings (Rattner and Morehouse 1994). The protocol, formalized and implemented in 1997, requires manufacturers to abide by a three-tiered test method in order for the USFWS to consider approval of any proposed nontoxic candidate material (USFWS 1997). Tier 1 requires existing data to be compiled on (1) the physical and chemical characterization of the shot or coating, (2) any existing ecological risk assessments and toxicity information of the candidate material, and (3) available tests that determined the effects on the reproduction of water birds. Tier 2 requires

erosion rate testing and acute toxicity testing on mallards, invertebrates, and early-life stage vertebrates to assess potential impacts on waterfowl habitat. Tier 3 requires chronic exposure tests to mallards under adverse environmental conditions to determine effects on reproduction. Based on the Tier 1 information, the USFWS can approve or deny the candidate material or require further testing in the Tier 2 and Tier 3 requirements (USFWS 1997).

Based on toxicity testing and results from the shot approval process, the USFWS established a maximum environmentally acceptable level of lead in shot as trace amounts of <1 percent (USFWS 1995) and steel was the first nontoxic shot approved following the three tiered testing requirement (USFWS 1999). To date, the testing has resulted in 12 approved nontoxic shot types, including different combinations of tungsten, bismuth, tin, iron, copper, nickel, and bronze (Table 6), illustrating that there are suitable alternatives to lead that present limited threats to waterfowl (USFWS 2013a). The testing requirements remain the current method used to approve the registration of nontoxic shot for hunting purposes (US FWS 2013b).

Table 6. The shot types that are approved as nontoxic for waterfowl hunting in the U.S. are the following.

Approved shot type*	**Percent Composition by Weight**
Bismuth-tin	97 bismuth, and 3 tin
Iron (steel)	iron and carbon
Iron-tungsten	any proportion of tungsten, and ≥1 iron
Iron-tungsten-nickel	≥1 iron, any proportion of tungsten, and up to 40 nickel
Tungsten-bronze	51.1 tungsten, 44.4 copper, 3.9 tin, and 0.6 iron, or 60 tungsten, 35.1 copper, 3.9 tin, and 1 iron
Tungsten-iron-copper-nickel	40-76 tungsten, 10-37 iron, 9-16 copper, and 5-7 nickel
Tungsten-matrix	95.9 tungsten, 4.1 polymer
Tungsten-polymer	95.5 tungsten, 4.5 Nylon 6 or 11
Tungsten-tin-iron	any proportions of tungsten and tin, and ≥1 iron
Tungsten-tin-bismuth	any proportions of tungsten, tin, and bismuth.
Tungsten-tin-iron-nickel	65 tungsten, 21.8 tin, 10.4 iron, and 2.8 nickel
Tungsten-iron-polymer	41.5-95.2 tungsten, 1.5-52.0 iron, and 3.5-8.0 fluoropolymer

* Coatings of copper, nickel, tin, zinc, zinc chloride, and zinc chrome on approved nontoxic shot types also are approved.

Toxicity of alternative metals

The tiered testing protocol for the approval of nontoxic ammunition provides comparable data on the toxicity of different kinds of metal alloys to mallards. In general, the results of these tests found combinations of tungsten, tin, bismuth, iron, and copper to provide limited threats based on a number of health metrics. Studies found no adverse effects on mallards dosed with bismuth-tin and steel (Sanderson et al. 1997), tungsten-iron, tungsten-polymer, and steel (Kelly 1998), and tungsten, tin, and bismuth (Ringelman et al. 1993). Additionally, Sanderson et al. (1997) found no effects on blood chemistries, body and kidney weight, livers, gonads, or gizzards for mallards dosed with bismuth-tin and steel. However, mild impairment of bile flow was observed in mallards in a different study that tested tungsten alloys (tungsten-iron and tungsten-polymer) (Kelly 1998). Ringelman et al. (1993) suggested that there is no uptake of tungsten, tin, or bismuth to mallard tissues and study results found no changes in 23 hematology and blood parameters measured. For toxicity tests with iron, mallard mortality rates were low and weight losses were not significantly different from control groups (Irby et al. 1967). Locke et al. (1967)

investigated the histopathological response of mallards dosed with uncoated and coated iron shot and also found limited effects. Mallards fed iron, molybdenum coated iron, and zinc coated iron developed hemosiderosis of the liver (Locke et al. 1967).

The mixture of tungsten-tin-bismuth has been tested in scavenging birds, with similar results as those found in the mallard toxicity tests. Risebrough (2001) tested a composite of tungsten-tin-bismuth on turkey vultures that were trapped from the wild. Tungsten and bismuth did not accumulate in tissues, although tin concentrations increased in the blood. There was no change in body mass, hematology, plasma biochemistries, or histopathology between dosed and control vultures. Krone et al. (2009) investigated the toxicity of brass and zinc to Pekin ducks (*Anas platyrhynchos domestica*) and found no mortality or organ alterations; however, the ducks dosed with zinc showed a higher weight loss compared to ducks dosed with brass and zinc showed the highest solubility in duck gizzards.

Birds are generally tolerant to metallic copper and multiple dosing studies found no mortality or clinical signs of toxicity and limited biochemical effects (Bellrose 1965, Irby et al. 1967, Locke et al. 1967, Krone et al. 2009, Bannon et al. 2011, Franson et al. 2012). Toxicity tests on mallards with metallic copper found no mortality in the dosed group (Irby et al. 1967). Locke et al. (1967) investigated the histopathological response of mallards dosed with copper shot, finding no lesions in tissues, no renal acid-fast inclusion bodies, and no hemosiderin in the liver. Bellrose (1965) tested different weights of single copper shot and the dosed mallards lost 15-20% of their initial weight by day 35 but recovered by day 110. The mallards in the copper tests were given doses over the limit of what they would normally be exposed to in the wild and several times the dose used in previous lead shot tests, suggesting that the quantities of normally ingested copper in the wild would rarely produce mortality. Bannon et al. (2011) investigated the toxicity of copper to rock doves that were exposed to small arms-range soil contaminated by spent ammunition from copper jacketed bullets with a lead core, reporting no copper retention in the tissues or any adverse effects. Similarly, Krone et al. (2009) investigated the toxicity of copper to Pekin ducks and found no mortality or organ alterations in the dosed group. Additionally, the

ducks dosed with copper did not show weight loss even after four weeks of shot retention in the gizzard.

Tests of metallic copper on raptors and scavenging species found similar non-significant results as the mallard and other avian toxicity tests suggest. Franson et al. (2012) tested copper pellets on captive raised American kestrels (*Falco sparverius*) and found no mortality or clinical signs of toxicity in the dosed birds. In addition, biochemistries, hematocrit values, and copper concentrations in the kidney and plasma did not differ between dosed and control birds. Hepatic copper concentrations were greater in dosed birds and hepatic copper and metallothionein concentrations significantly correlated. The authors suggested that birds sharing similar regurgitation frequency as American kestrels are not likely to be adversely affected by copper ingestion. Risebrough (2001) investigated the toxicity of copper to turkey vultures that were trapped from the wild. The vultures were fed food dosed with copper at a level that was considered equivalent to the ingestion of two copper bullets. The study found no mortality; however, the copper levels were greater in the livers of dosed vultures compared to those in the control group.

Although metallic copper does not seemingly present a health threat to birds, the salts of copper have been found to exhibit toxicity in selected cases. Kobayashi et al. (1992) investigated a die-off of free-ranging mute swans with high copper concentrations in liver tissue, and attributed the deaths to copper poisoning of unknown source. For example, Henderson and Winterfield (1975) recorded two cases of copper poisoning in Canada geese, suggesting that copper sulfate used as an algicide was the cause.

Tissue concentrations of copper can vary widely among individuals and species, and can reach seemingly elevated levels without detectable effects to individuals. For example, copper concentrations in the livers of California condors ranged from 2.2 to 531 ppm wet weight at necropsy (Rideout et al. 2012). None of these birds were diagnosed with copper toxicosis. In American kestrels dosed with copper pellets the mean liver concentration was 20.7 ppm dry weight (Franson et al. 2012). Multiple studies report much higher liver copper concentrations

specifically in mute swans and common eiders. The copper concentration in the livers of three mute swans found dead in Mamaroneck Harbor, NY, ranged from 1562-5857 µg/g dry weight, while hepatic concentrations in two captive mute swans were much lower, 64 and 121 µg/g dry weight (Molnar 1983). The copper contamination was suspected to be from antifouling paint released in water through the scraping of vessel hulls. Clausen and Wolstrup (1978) and Kobayashi et al. (1992) also detected high concentrations of copper in the liver of mute swans and other studies have found high copper concentrations in the livers of the common eider over 1000 ppm wet weight (Norheim and Borch-Iohnsen 1990; Hollmén et al. 1998; Franson et al. 2000; Stout et al. 2002). Because of this variability, interpretation of copper concentrations in tissue should be done with caution, as high levels do not necessarily signify adverse effects.

Discussion Points: Toxicity of alternative metals used in ammunition

- A comprehensive testing method exists in the U.S. to ensure alternative metals used to hunt waterfowl do not cause sickness and death when ingested by migratory birds. These data can be extrapolated to ammunition used for other forms of hunting.
- The nontoxic shot approval process has resulted in the approval of suitable alternatives to lead that present limited environmental threats.
- The types of nontoxic metals available and approved for use offer the public many options. After the 1991 lead ban, steel became the number one alternative to lead and is still widely used.
- Copper, the primary alternative currently used in bullets, exhibits low toxicity to birds in its metallic form.

Conclusion

While scavenging birds may be exposed to multiple sources of lead in the environment, scientific evidence points to lead ammunition as the most frequent cause of mortality in lead exposure cases. A suite of evidence exists to support this conclusion, including the behavioral ecology and physiology of scavenging birds, their sensitivity as exhibited in controlled dosing studies, the

bioavailability of lead ammunition due to fragmentation, recovery of ingested lead fragments or pellets from exposed birds, observations of birds feeding on contaminated carcasses, isotopic analyses relating tissue concentrations to ammunition, patterns of mortality coincident with hunting seasons, diagnosis of lead poisoning by well-established tissue thresholds and clinical signs, and the lack of abundant evidence for other sources of lead. While few cases of mortality and morbidity may exhibit all of these lines of evidence, there are numerous documented cases of lead- poisoned scavengers in the literature and many are be supported by one or more lines of evidence.

In order to reduce the adverse effects of lead ammunition to scavenging birds, it must be removed from the exposure pathway. This need not necessitate a reduction in hunting, simply a change in practices. Carcasses or gut piles can be made unavailable to scavengers by removal from the field. Lead can be replaced in ammunition by alternative metals that are currently available and present limited environmental threats. Other avenues or points on the exposure pathway can be explored for further solutions. While reducing exposure to lead ammunition will not result in an end to all lead exposure to avian scavengers, we believe that analysis of the scientific literature shows this to be the largest pathway of such exposure and thus will result in the greatest beneficial effect to species.

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References

Anderson WL, Havera SP, Zercher BW. 2000. Ingestion of lead and nontoxic shotgun pellets by ducks in the Mississippi flyway. *The Journal of Wildlife Management* 64:848-857.

Bagley GE, Locke LN, Nightingale GT. 1967. Lead poisoning in Canada geese in Delaware. *American Association of Avian Pathologists* 11:601-608.

Bannon DI, Parsons PJ, Centeno JA, Lal S, Xu H, Rosencrance AB, Dennis WE, Johnson MS. 2010. Lead and copper in pigeons (*Columbia livia*) exposed to a small arms–range soil. *Archives of Environmental Contamination and Toxicology* 60:351-360.

Barrett MW, Karstad LH. 1971. A fluorescent erythrocyte test for lead poisoning in waterfowl. *The Journal of Wildlife Management* 35:109-119.

Batha C, Lehman P. 2011. How good are copper bullets, really???. Final Technical Report. The Wisconsin Department of Natural Resources, Madison, WI.

Bedrosian B, Craighead D. 2009. Blood lead levels of bald and golden eagles sampled during and after hunting seasons in the Greater Yellowstone Ecosystem. Extended Abstract. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA, pp 219-220. Available: http://www.peregrinefund.org/subsites/conference-lead/PDF/0209%20Bedrosian.pdf

Bedrosian B, Craighead D, Crandall R. 2012. Lead exposure in bald eagles from big game hunting, the continental implications and successful mitigation efforts. *PLos ONE* 7: e51978.

Bellrose FC. 1951. Effects of ingested lead shot upon waterfowl populations. *North American Wildlife Conference Transactions* 16:125-133.

Bellrose FC. 1959. Lead poisoning as a mortality factor in waterfowl populations. *Illinois Natural History Survey Bulletin* 27:235-288.

Bellrose FC. 1965. Wasted waterfowl. Final Report. Exhibit F. Mississippi Flyway Council Planning Committee.

Berglund ÅMM, Ingvarsson PK, Danielsson H, Nyholm NEI. 2010. Lead exposure and biological effects in pied flycatchers (*Ficedula hypoleuca*) before and after the closure of a lead mine in northern Sweden. *Environmental Pollution* 158:1368-1375.

Beyer WN, Franson JC, Locke LN, Stroud RK, Sileo L. 1998. Retrospective study of the diagnostic criteria in a lead-poisoning survey of waterfowl. *Archives of Environmental Contamination and Toxicology* 35:506-512.

Bloom PH, Scott JM, Pattee OH, Smith MR. 1989. Lead contamination of golden eagles (*Aquila chrysaetos*) within the range of the California condor (*Gymnogyps californianus*). Proceedings of the 3rd World Conference on Birds of Prey and Owls, Eliat, Israel. In Meyburgh BU, Chancellor RD, eds, *Raptors in the Modern World*, pp 481-482. Available from: http://www.raptors-international.org/book/raptors_in_the_modern_world/Bloom_Scott_1989_481-482.pdf

Blus LJ, Henny CJ, Hoffman DJ, Grove RA. 1991. Lead toxicosis in tundra swans near a mining and smelting complex in northern Idaho. *Archives of Environmental Contamination and Toxicology* 21:549-555.

Bollhöfer B, Rosman KJR. 2001. Isotopic source signatures for atmospheric lead: The Northern Hemisphere. *Geochimica et Cosmochimica Act* 65:1727-1740.

Buckley, NJ. 1999. Black Vulture (*Coragyps atratus*), The Birds of North America Online, Poole A, ed. Ithaca: Cornell Lab of Ornithology. Available from: the Birds of North America Online: http://bna.birds.cornell.edu/bna/species/411

Buehler, DA. 2000. Bald Eagle (*Haliaeetus leucocephalus*), The Birds of North America Online, Poole A, ed, Ithaca: Cornell Lab of Ornithology. Available from: The Birds of North America Online: http://bna.birds.cornell.edu/bna/species/506

Buerger TT, Mirarchi RE, Lisano ME. 1986. Effects of lead shot ingestion on captive mourning dove survivability and reproduction. *Journal of Wildlife Management* 50:1-8.

Burger J, Gochfeld M. 1990. Tissue levels of lead in experimentally exposed herring gull (*Larus argentatus*) chicks. *Journal of Toxicology and Environmental Health* 29:219-233.

Burger J. 1993. Metals in avian feathers bioindicators of environmental pollution. In Hodgson E, ed, *Review of Environmental Toxicology*. Vol 5. Toxicology Communications Inc. Raleigh, NC, USA, pp 203-311.

Burger J, Lavery MH, Gochfeld M. 1994. Temporal changes in lead levels in common tern feathers in New York and relationship of field levels to adverse effects in the laboratory. *Environmental Toxicology and Chemistry* 13:581-586.

Burger J, Gochfeld M, Jeitner C, Snigaroff D, Snigaroff R, Stamm T, Volz C. 2008. Assessment of metals in down feathers of female common eiders and their eggs from the Aleutians: Arsenic, cadmium, chromium, lead, manganese, mercury, and selenium. *Environmental Monitoring and Assessment* 143:247-256.

Cade TJ. 2007. Exposure of California condors to lead from spent ammunition. *Journal of Wildlife Management* 71:2125-2133.

Carpenter JW, Pattee OH, Fritts SH, Rattner BA, Wiemeyer SN, Royle JA, Smith MR. 2003. Experimental lead poisoning in turkey vultures (*Cathartes aura*). *Journal of Wildlife Diseases* 39:96-104.

Chupp NR, Dalke PD. 1964. Waterfowl mortality in the Coeur D'alene river valley, Idaho. *Journal of Wildlife Management* 28:692-702.

Church ME, Gwiazda R, Risebrough RW, Sorenson K, Chamberlain CP, Farry S, Heinrich W, Rideout BA, Smith, DR. 2006. Ammunition is the principal source of lead accumulated by California condors re-introduced to the wild. *Environmental Science and Technology* 40:6143-6150.

Clark AJ, Scheuhammer AM. 2003. Lead poisoning in upland-foraging birds of prey in Canada. *Ecotoxicology* 12:23-30.

Clausen B, Wolstrup C. 1978. Copper load in mute swans (*Cygnus olor*) found in Denmark. *Norisk Veterinary Medicine* 30:260-266.

Cornatzer WE, Fogarty EF, Cornatzer EW. 2009. Qualitative and quantitative detection of lead bullet fragments in random venison packages donated to the Community Action Food Centers of North Dakota, 2007. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA, pp 154-156. Available from: http://www.peregrinefund.org/subsites/conference-lead/PDF/0111%20Cornatzer.pdf

Craighead D, Bedrosian B. 2008. Blood lead levels of common ravens with access to big-game offal. *Journal of Wildlife Management* 72:240-245.

Craighead D, Bedrosian B. 2009. A relationship between blood lead levels of common ravens and the hunting season in the southern Yellowstone Ecosystem. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of lead from spent ammunition: Implications for wildlife

and humans. The Peregrine Fund, Boise, Idaho, USA, pp 1-4. Available from: http://www.peregrinefund.org/subsites/conference-lead/PDF/0206%20Craighead.pdf

Cruz-Martinez L, Redig PT, Deen J. 2012. Lead from spent ammunition: A source of exposure and poisoning in bald eagles. *Human-Wildlife Interactions* 6:94-104.

Dauwe L, Bervoets R, Blust M, Ee T. 2002. Tissue levels of lead in experimentally exposed zebra finches (*Taeniopygia guttata*) with particular attention on the use of feathers as biomonitors. *Archives of Environmental Contamination and Toxicology* 42:88-92.

DeMent SH, Chisolm JJ Jr, Eckhaus MA, Strandberg JD. 1987. Toxic lead exposure in the urban rock dove. *Journal of Wildlife Diseases* 23:273-278.

Dieter MP, Finley MT. 1978. Erythrocyte δ-aminolevulinic acid dehydratase activity in mallard ducks: Duration of inhibition after lead shot dosage. *The Journal of Wildlife Management* 42:621-625.

Dobrowolska A, Melosik M. 2008. Bullet-derived lead in tissues of the wild boar (*Sus scrofa*) and red deer (*Cervus elaphus*). *European Journal Wildlife Research* 54:231-235.

Domenech R, Langer H. 2009. Blood-lead levels of fall migrant golden eagles in west-central Montana. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA, pp 221-222. Available from: http://www.peregrinefund.org/subsites/conference-lead/PDF/0210%20Domenech.pdf

Douglas-Stroebel E, Hoffman DJ, Brewer GL, Sileo L. 2004. Effects of lead-contaminated sediment and nutrition on mallard duckling brain growth and biochemistry. *Environmental Pollution* 131:215-222.

Duke GE, Jegers AA, Loff G, Evanson OA. 1975. *Comparative Biochemistry and Physiology* 50A:649-656.

Duke GE. 1997. Gastrointestinal physiology and nutrition in wild birds. *Proceedings of the Nutrition Society* 56:1049-1056.

Dunlap TR.1998. Sport hunting and conservation 1880 -1920. *Environmental History Review* 12:51-60.

Dunstan TC, Harper JF.1975. Food habits of bald eagles in north-central Minnesota. *Journal of Wildlife Management* 39:140-143.

Dusek GL, Mackie RJ, Herriges JD, Compton BB. 1989. Population ecology of white-tailed deer along the lower Yellowstone River. Wildlife Monographs ISSN:00840173. *The Wildlife Society* 104:1-68.

Dykstra CR, Meyer MM, Stromburd KL, Warnke KD, Bowerman WW, Best DA. 2001. Association of low reproductive rates and high contaminant levels in bald eagles on Green Bay, Lake Michigan. *Journal of Great Lakes Research* 27:239-251.

Edens FW, Benton E, Bursian SJ, Morgan GW. 1976. Effect of dietary lead on reproductive performance in Japanese quail, *Coturnix coturnix japonica*. *Toxicology and Applied Pharmacology* 38:307-314.

Eisler R. 2000. Lead. In Eisler R, ed, *Handbook of Chemical Risk Assessment: Health Hazards to Humans, Plants, and Animals*, Vol 1-Metals. Lewis Publishers. Boca Raton, FL, USA, pp 201-311.

Elliott JE, Langelier KM, Scheuhammer AM, Sinclair PH, Whitehead PE. 2002. Incidence of lead poisoning and lead shot in waterfowl gizzards from British Columbia, 1988-91. Canadian Wildlife Service Progress Notes No. 200, Ottawa, Canada.

Ewins PJ, Andress RA. 1995. The diet of bald eagles, *Haliaeetus Leucocephalus*, wintering in the Lower Great Lakes Basin, 1987-1995. *Canadian Field-Naturalist* 109:418-425.

Farner DS. 1960. Digestion and the digestive system. In Marshall AJ, ed, *Biology and Comparative Physiology of Birds*, Academic Press, NY, pp 411-468.

Ferrer M, de la Riva M, Castroviejo J. 1991. Electrocution of raptors on power lines in southwestern Spain. *Journal of Field Ornithology* 62:181-190.

Finkelstein ME, Gwiazda RH, Smith DR. 2003. Lead poisoning of seabirds: Environmental risks from leaded paint at a decommissioned military base. *Environmental Science and Technology* 37:3256-3260.

Finkelstein ME, George D, Scherbinski S, Gwiazda R, Johnson M, Burnett J, Brandt J, Lawrey S, Pessier AP, Clark M. 2010. Feather lead concentrations and 207Pb/206Pb ratios reveal lead exposure history of California condors (*Gymnogyps californianus*). *Environmental Science and Technology* 44:2639-2647.

Finkelstein ME, Doak DF, George D, Burnett J, Brandt J, Church M, Grantham J, Smith DR. 2012. From the cover: Lead poisoning and the deceptive recovery of the critically

endangered California condor. *Proceedings of the National Academy of Sciences* 109:11449-11454.

Finley MT, Dieter MP, Locke LN. 1976. δ-Aminolevulinic acid dehydratase: Inhibition in ducks dosed with lead shot. *Environmental Research* 12:243-249.

Finley MT, Dieter MP. 1978. Erythrocyte δ-aminolevulinic acid dehydratase activity in mallard ducks: Duration of inhibition after lead shot dosage. *The Journal of Wildlife Management* 42:621-625.

Franson JC, Sileo L, Pattee OH, Moore JF. 1983. Effects of chronic dietary lead in American kestrels (*Falco Sparverius*). *Journal of Wildlife Diseases* 19:110-113.

Franson JC, Haramis G. Perry M, Matthew C. and Moore, John F. 1986. Blood protoporphyrin for detecting lead exposure in canvasbacks. Final Report. Lead Poisoning in Wild Waterfowl - A Workshop, National Wildlife Federation, Washington, DC, pp 32-37.

Franson JC, Hereford SG. 1994. Lead poisoning in a Mississippi sandhill crane. *The Wilson Bulletin* 106:766-768.

Franson JC, Hohman,WL, Moore JL, Smith MR. 1996. The efficacy of protoporphyrin as a predictive biomarker for lead exposure in canvasback ducks: Effect of sample storage time. *Environmental Monitoring and Assessment* 43:181-188.

Franson JC, Hollmén T, Poppenga RH, Hario M, Kilpi M. 2000. Metals and trace elements in tissues of common eiders (*Somateria mollissima*) from the Finnish Archipelago. *Ornis Fennica* 77:57-63.

Franson JC, Pain D. 2011. Lead in birds. In Beyer WN, Meador JP, eds, *Environmental Contaminants in Biota, Interpreting Tissue Concentrations*, 2nd ed. Taylor & Francis Group LLC, Boca Raton, FL, USA, pp 563-593

Franson JC, Lahner LL, Meteyer CU, Rattner BA. 2012. Copper pellets simulating oral exposure to copper ammunition: Absence of toxicity in American kestrels (*Falco sparverius*). *Archives of Environmental Contamination and Toxicology* 62:145-153.

Friend M. 2009. Biological and societal dimensions of lead poisoning in birds in the USA. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans, The Peregrine Fund, Boise, Idaho, USA, pp 34-60.

Available From: http://www.peregrinefund.org/subsites/conference-lead/PDF/0104%20Friend.pdf

Fry M, Maurer J. 2003. Assessment of lead contamination sources exposing California condors. Final Report. California Department of Fish and Game, Habitat Conservation Planning Branch. Sacramento, CA.

Gangoso L, Álvarez-Lloret P, Rodríguez-Navarro AAB, Mateo R, Hiraldo F, Donázar JA. 2009. Long-term effects of lead poisoning on bone mineralization in vultures exposed to ammunition sources. *Environmental Pollution* 157:569-574.

Gasparik J, Venglarcik J, Slamecka J, Kropil R, Smehyl P, Kopecky J. 2012. Distribution of lead in selected organs and its effect on reproduction parameters of pheasants (*Phasianus colchicus*) after an experimental per oral administration. *Journal of Environmental Science and Health, Part A: Toxic/Hazardous Substances and Environmental Engineering* 49:1267-1271.

Goede AA, de Voogt P. 1985. Lead and cadmium in waders from the Dutch Wadden Sea. *Environmental Pollution Series A, Ecological and Biological* 37:311-322.

Goede AA, De Bruin M. 1986. The use of bird feathers for indicating heavy metal pollution. *Environmental Monitoring and Assessment* 7:249-256.

Golden NH, Rattner BA. 2003. Ranking terrestrial vertebrate species for utility in biomonitoring and vulnerability to environmental contaminants. *Reviews in Environmental Contamination and Toxicology* 176:67-136.

Golden NH, Rattner BA, Cohen JB, Hoffman DJ, Ottinger MA. 2003. Lead accumulation in feathers of nestling black-crowned night-herons (*Nycticorax nycticorax*) experimentally treated in the field. *Environmental Toxicology and Chemistry* 22:1517-1524.

Gómez-Ramírez P, Martínez-López E, María-Mojica P, León-Ortega M, García-Fernández AJ. 2010. Blood lead levels and δ-ALAD inhibition in nestlings of Eurasian eagle owl (Bubo bubo) to assess lead exposure associated to an abandoned mining area. *Ecotoxicology* 20:131-138.

Grand JB, Franson JC, Flint PL, Petersen MR. 2002. Concentrations of trace elements in eggs and blood of spectacled and common eiders on the Yukon-Kuskokwim Delta, Alaska, USA. *Environmental Toxicology and Chemistry* 21:1673-1678.

Grantham J. 2007. Reintroduction of California condors into their historic range: The recovery program in southern California. In Mee A, Hall LS, eds, California Condors in the 21st Century. Nuttall Ornithological Club, Cambridge, MA, and American Ornithologists' Union, Washington, DC, pp 123-139.

Grasman KA, Scanlon PF. 1995. Effects of acute lead ingestion and diet on antibody and T-cell-mediated immunity in Japanese quail. *Archives of Environmental Contamination and Toxicology* 28:161-167.

Green RE, Newton I, Shultz S, Cunningham AA, Gilbert M, Pain DJ, Prakash V. 2004. Diclofenac poisoning as a cause of vulture population declines across the Indian subcontinent. *Journal of Applied Ecology* 41:793-800.

Green R, Hunt WG, Parish CN, Newton I. 2009. Effectiveness of action to reduce exposure to free-ranging California condors in Arizona and Utah to lead from spent ammunition. In RT Watson, M Fuller, M Pokras, G. Hunt, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans. The Peregrine Fund, Boise, Idaho, USA, pp 240-253. Available from: http://www.peregrinefund.org/subsites/conference-lead/PDF/0218%20Green.pdf

Griffin CR, Baskett TS, Sparrowe RD. 1980. Bald eagles and the management program at Swan Lake National Wildlife Refuge. *Transactions of the 45th North American Wildlife and Natural Resources Conference* 45: 253-262.

Grinnell GB. 1894. Lead-poisoning. *Forest and Stream* 42:117-118.

Grue CE, Hoffman DJ, Beyer WN, Franson LP. 1986. Lead concentrations and reproductive success in European starlings (*Sturnus vulgaris*) nesting within highway roadside verges. *Environmental Pollution Series A, Ecological and Biological* 42:157–182.

Grund MD, Cornicelli L, Carlson LT, Butler EA. 2010. Bullet fragmentation and lead deposition in white-tailed deer and domestic sheep. *Human-wildlife Interactions* 4:257-265.

Hahn E, Hahn K, Stoeppler M. 1993. Bird feathers as bioindicators in areas of the German Environmental Specimen Bank--bioaccumulation of mercury in food chains and exogenous deposition of atmospheric pollution with lead and cadmium. *Science of the Total Environment* 140:259-270.

Hansen JA, Audet D, Spears BL, Healy KA, Brazzle RE, Hoffman DJ, Dailey A, Beyer WN. 2011. Lead exposure and poisoning of songbirds using the Coeur d'Alene River Basin, Idaho, USA. *Integrated Environmental Assessment and Management* 7:587-595.

Harmata AR, Restani M. 2013. Lead, mercury, selenium, and other trace elements in tissues of golden eagles from southwestern Montana, USA. *Journal of Wildlife Diseases* 49:114-124.

Harper RG, Scott D, Dunstan TC. 1988. Nonfish prey of wintering bald eagles in Illinois. The Wilson Bulletin 100:688-690.

Henderson BM, Winterfield RW. 1975. Acute copper toxicosis in the Canada goose. *Avian Diseases* 19:385-387.

Hoffman DJ, Pattee OH, Wiemeyer SN, Mulhern B. 1981. Effects of lead shot ingestion on δ-aminolevulinic acid dehydratase activity, hemoglobin concentrations, and serum chemistry in bald eagles. *Journal of Wildlife Diseases* 17:423-431.

Hoffman DJ, Franson JC, Pattee OH, Bunck CM, Anderson, A. 1985. Survival, growth, and accumulation of ingested lead in nestling American kestrels (*Falco sparverius*). *Archives of Environmental Contamination and Toxicology* 14:89-94.

Hollmén T, Franson JC, Poppenga RH, Hario M, Kilpi M. 1998. Lead poisoning and trace elements in common eiders (*Somateria mollissima*) from Finland. *Wildlife Biology* 4:193-203.

Hough E. 1894. Lead-poisoned ducks. *Forest and Stream* 42:117-118.

Hunt WG, Burnham W, Parish CN, Burnham KK, Mutch B, Oaks JL. 2006. Bullet fragments in deer remains: Implications for lead exposure in avian scavengers. *Wildlife Society Bulletin* 34:167-170.

Hunt WG, Parish CN, Farry SC, Lord TG, Sieg R. 2007. Movements of introduced California condors in Arizona in relations to lead exposure. In Mee A, Hall L, eds, California Condors in the 21st Century. Series in Ornithology No. 2, Nuttall Ornithological Club and the American Ornithologists' Union, pp 79-96. Available from: http://peregrinefund.org/docs/pdf/research-library/2007/2007-Hunt-condors-lead.pdf

Hunt WG, Watson RT, Oaks JL, Parish CN, Burnham KK, Tucker RL, Belthoff JR, Hart G. 2009. Lead bullet fragments in venison from rifle-killed deer: Potential for human dietary exposure. *PLoS ONE* 4:e5330. Doi:10.1371/journal.pone.0005330.

Iqbal S, Blumenthal W, Kennedy C, Yip FY, Pickard S, Flanders WD, Loringer K, Kruger K, Caldwell KL, Jean Brown M. 2009. Hunting with lead: Association between blood lead levels and wild game consumption. *Environmental Research* 109:952-959.

Irby HD, Locke LN, Bagley GE. 1967. Relative toxicity of lead and selected substitute shot types to game farm mallards. *The Journal of Wildlife Management* 31:253-257.

Jackson AJ, Ruxton GD, Houston DC. 2008. The effect of social facilitation on foraging success in vultures: A modeling study. *Biology Letters* 4:311-313.

Jacobson E, Carpenter JW, Novilla M. 1977. Suspected lead toxicosis in a bald eagle. *Journal of American Veterinary Medical Associates* 171:952-954.

Jordan JS, Bellrose FC. 1951. Lead poisoning in wild waterfowl. Illinois Natural History Survey, Biological Notes No. 26, Urbana, IL.

Kelly ME, Fitzgerald SD, Aulerich RJ, Balander RJ, Powell DC, Stickle RL, Stevens W, Cray C, Tempelman RJ, Bursian SJ. 1998. Acute effects of lead, steel, tungsten, iron, and tungsten-polymer shot administered to game farm mallards. *Journal of Wildlife Diseases* 34:673-687.

Kelly A, Kelly S. 2005. Are mute swans with elevated blood lead levels more likely to collide with overhead power lines? *Waterbirds* 28:331-334.

Kelly TR, Bloom PH, Torres SG, Hernandez YZ, Poppenga RH, Boyce WM, Johnson CK. 2011. Impact of the California lead ammunition ban on reducing lead exposure in golden eagles and turkey vultures. *PLoS ONE* 6:e17656.

Kelly TR, Johnson CK. 2011. Lead exposure in free-flying turkey vultures is associated with big game hunting in California. *PLos ONE* 6:e15350.

Kendall RJ, Scanlon PF. 1981. Effects of chronic lead ingestion on the reproductive characteristics of ringed turtle doves *Streptopelia risoria* and on tissue lead concentrations of adults and their progeny. *Environmental Pollution Series A, Ecological and Biological* 26:203-213.

Kendall RJ, Lacker TE, Bunck C, Daniel B, Driver C, Grue CE, Leighton F, Stansley W, Watanabe PG, Whitworth M. 1996. An ecological risk assessment of lead shot exposure in non-waterfowl avian species: Upland game birds and raptors. *Environmental Toxicology and Chemistry* 15: 4-10.

Kennedy S, Crisler JP, Smith E, Bush M. 1977. Lead poisoning in sandhill cranes. *Journal of the American Veterinary Medical Association* 171:955-958.

Kenntner N, Tataruch F, Krone O. 2001. Heavy metals in soft tissue of white-tailed eagles found dead or moribund in Germany and Austria from 1993 to 2000. *Environmental Toxicology and Chemistry* 20:1831-1837.

Kirk DA, Mossman MJ. 1998. Turkey Vulture (*Cathartes aura*), The Birds of North America Online, Poole A, ed, Ithaca: Cornell Lab of Ornithology. Available from: Retrieved from the Birds of North America Online: http://bna.birds.cornell.edu/bna/species/339

Knopper LD, Mineau P, Scheuhammer AM, Bond DE, McKinnon DT. 2006. Carcasses of shot Richardson's ground squirrels may pose lead hazards to scavenging hawks. *Journal of Wildlife Management* 70:295-299.

Knot J, Gilbert J, Green RE, Hoccom DG. 2009. Comparison of the lethality of lead and copper bullets in deer control operations to reduce incidental lead poisoning; Field trails in England and Scotland. *Conservation Evidence* 6:71-78.

Kobayashi Y, Shimada A, Umemura T, Nagai T.1992. An outbreak of copper poisoning in mute swans (*Cygnus olor*). *Japanese Veterinary Medical Science* 54:229-233.

Kochert MN., Steenhof K, Mcintyre CL, Craig EH. 2002. Golden Eagle (*Aquilachrysaetos*), The Birds of North America Online, Poole A, ed, Ithaca: Cornell Lab of Ornithology, Available from: The Birds of North America Online: http://bna.birds.cornell.edu/bna/species/684 doi:10.2173/bna.684

Koranda J, Moore K. 1979. Dietary effects on lead uptake and trace element distribution in mallard ducks dosed with lead shot. Final Report. California Fish and Game Department, Wildlife Investigations Lab, Sacramento, CA.

Kozie KD, Anderson RK. 1991. Productivity, diet, and environmental contaminants in bald eagles nesting near the Wisconsin shoreline of Lake Superior. *Archives of Environmental Contamination and Toxicology* 20:41-48.

Kramer JL, Redig PT. 1997. Sixteen years of lead poisoning in eagles, 1980-95: An epizootiologic view. *Journal of Raptor Research* 31:327-332.

Krementz DG, Ankney CD. 1995. Changes in total body calcium and diet of breeding house sparrows. *Journal of Avian Biology* 26:162-167.

Krone O, Kenntner N, Trinogga A, Nadjafzadeh M, Scholz F, Sulawa J, Totschek K, Schuck-wersig P, Zieschank R. 2009. Lead poisoning in white-tailed sea eagles: Causes and approaches to solutions in Germany. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans, The Peregrine Fund, Boise, Idaho, USA, pp 1-13. Available from: http://www.nps.gov/pinn/naturescience/upload/Krone_2009_Lead%20Poisoning%20in%20White-Tailed%20Sea%20Eagles%20-%20Causes%20and%20Approaches%20to%20Solutions%20in%20Germany.pdf

Lambertucci SA, Donázar JA, Huertas AD, Jiménez B, Sáez M, Sanchez-Zapata JA, Hiraldo F. 2011. Widening the problem of lead poisoning to a South-American top scavenger: Lead concentrations in feathers of wild Andean condors. *Biological Conservation* 144:1464-1471.

Lang AL, Andress RA, Martin PA. 2001. Prey remains in bald eagle (*Haliaeetus leucocephalus*) pellets from a winter roost in the Upper St. Lawrence River, 1996-1997. *Wildlife Medicine* 24:21-26.

Leonzio C, Massi A. 1989. Metal biomontoring in bird eggs: A critical experiment. *Bulletin of Environmental Contamination and Toxicology* 43:401-406.

Locke LN, Bagley GE, Irby HD. 1966. Acid-fast intranuclear inclusion bodies in the kidneys of mallards fed lead shot. *Bulletin of Wildlife Disease Association* 2:127-132.

Locke LN, Irby HD, Bagley GE. 1967. Histopathology of mallards dosed with lead and selected substitute shot. *Journal of Wildlife Diseases* 3:143-147.

Locke LN, Bagley GE. 1967. Lead poisoning in a sample of Maryland mourning doves. *Journal of Wildlife Management* 31:515-518.

Locke LN, Thomas NJ. 1996. Lead poisoning of waterfowl and raptors. In Fairbrother A, Locke LN, Huff GL, eds, Noninfectious diseases of wildlife, 2nd edition. Iowa State University Press, Ames, Iowa, USA, pp108-117.

Martin PA, Barrett G. 2000. Exposure of terrestrial raptors to environmental lead-determining sources using stable isotopes. Society of Toxicology and Chemistry 21st Annual Meeting, Abstract. Nashville TN. Available from: http://abstracts.co.allenpress.com/pweb/setac2000/document/19846

Martin PA, Campbell D, Hughes K, McDaniel. 2008. Lead in tissues of terrestrial raptors in southern Ontario, Canada, 1995-2001. *Science of the Total Environment* 391:96-103.

Mazliah J, Barron S, Bental E, Reznik I .1989. The effects of chronic lead intoxication in mature chickens. *Avian Diseases* 33:566-570.

McBride TJ, Smith JP, Gross HP, Hopper MJ. 2004. Blood-lead and ALAD activity levels of cooper's hawks (*Accipiter cooperii*) migrating through the southern Rocky Mountains. *Journal of Raptor Research* 38:118-124.

Mee A, Rideout BA, Hamber JA, Todd JN, Austin G, Clark M, Wallace MP. 2007. Junk ingestion and nestling mortality in a reintroduced population of California condors, *Gymnogyps californianus*. *Bird Conservation International* 17:119-130.

Merendino MT, Lobpries DS, Neaville JE, Ortego JD, Johnson WP. 2013. Regional differences and long-term trends in lead exposure in mottled ducks. *Wildlife Society Bulletin* 33:1002-1008.

Mierzykowski SE, Todd CS, Pokras MA, Oliveira RD. 2013. Lead and mercury levels in livers of bald eagles recovered in New England. Final Report. U.S. Fish and Wildlife Service, US Department of interior, Special Project Report FY13-MEFO-2-EC. Maine Field Office. Orono, ME. Available from: http://www.fws.gov/northeast/mainecontaminants/pdf/PbHg_BAEA_LiversNewEngland2013.pdf

Millsap BA. 1986. Status of wintering bald eagles in the conterminous 48 states. Wildlife Society Bulletin 14:433-440.

Molnar JJ. 1983. Copper storage in the liver of the wildlife mute swan (*Cygnus olor*). Its possible relation to pollution. *Archives of Pathology* 107:629-632.

Morgan GW, Edens FW, Thaxton P, Parkhurst CR. 1975. Toxicity of dietary lead in Japanese quail. *Poultry Science* 54:1636-1642.

Morrison JL, Dwyer JF. 2012. Crested Caracara (*Caracara cheriway*), The Birds of North America Online, Poole A, ed, Ithaca: Cornell Lab of Ornithology. Available from: The Birds of North America Online: http://bna.birds.cornell.edu/bna/species/249

Moulton DW, Frentress CD, Stutzenbaker CD, Lobpries DS, Brownlee WC. 1988. Ingestion of shotgun pellets by waterfowl in Texas. In Weller M, ed, *Waterfowl in Winter*, University of Minnesota press, Minneapolis, MN, USA, pp 597-607.

Nain S, Smits JEG. 2011. Subchronic lead exposure, immunotoxicology and increased disease resistance in Japanese quail (*Corturnix coturnix japonica*). *Ecotoxicology and Environmental Safety* 74:787-792.

Neumann K. 2009. Bald eagle lead poisoning in winter. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of lead from spent ammunition: Implications for wildlife and humans, The Peregrine Fund, Boise, Idaho, USA, pp 210-218. Available from: https://www.peregrinefund.org/subsites/conference-lead/PDF/0119%20Neumann.pdf

Nixon CM, Hansen LP, Brewer PA, Chelsvig JE, Esker TL, Etter D, Sullivan JB, Koerkenmeier RG, Mankin PC. 2001. Survival of white-tailed deer in intensively farmed areas of Illinois. *Canadian Journal of Zoology* 79:581-588.

Norheim G, Borch-Iohnsen B. 1990. Chemical and morphological studies of liver from eider (*Somateria mollissima*) in Svalbard with special reference to the distribution of copper. *Journal of Comparative Pathology* 102:457-466.

Ochiai K, Jin K, Itakura C, Goryo M, Yamashita K, Mizuno N, Fujinaga T, Tsuzuki T. 1992. Pathological study of lead poisoning in whooper swans (*Cygnus cygnus*) in Japan. *Avian Diseases* 36:313-323.

O'halloran J, Myers AA, Duggan PF. 1989. Some sub-lethal effects of lead on mute swan *Cygnus olor*. *Journal of Zoology* 218:627-632.

Onalaja AO, Claudio L. 2000. Genetic susceptibility to lead poisoning. *Environmental Health Perspective* 108:23-28.

Pain DJ, Rattner BA. 1988. Mortality and hematology associated with the ingestion of one number four lead shot in black ducks, *Anas rubripes*. *Bulletin of Environmental Contamination and Toxicology* 40:159 -164.

Pain DJ, Amiard-Triquet C. 1993. Lead poisoning of raptors in France and elsewhere. *Ecotoxicology and Environmental Safety* 25:183-192.

Pain DJ. 1995. Lead in the environment. In Hoffman DJ, Rattner BA, Burton GAJr, Cairns JJr, eds, *Handbook of Ecotoxicology*. CRC Press Inc., Boca Raton, FL, USA, pp 356-391.

Pain DJ, Bavoux C, Burneleau G. 1997. Seasonal blood lead concentrations in marsh harriers *Circus aeruginosus* from Charente-maritime, France: Relationship with the hunting season. *Biological Conservation* 81:1-7.

Pain DJ, Meharg AA, Ferrer M, Taggart M, Penteriani V. 2005. Lead concentrations in bones and feathers of the globally threatened Spanish imperial eagle. *Biological Conservation* 121:603-610.

Pain D, Fisher IJ, Thomas, VG. 2009. A global ipdate of lead poisoning in terrestrial birds from ammunition sources. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA, pp 99-118. Available from: http://www.peregrinefund.org/subsites/conference-lead/PDF/0108%20Pain.pdf

Parish CN, Hunt WG, Feltes E, Seig R, Orr K. 2009. Lead exposure among reintroduced population of California condors in northern Arizona and southern Utah. In Watson RT, Fuller M, Pokras M, Hunt G, eds. Ingestion of lead from spent ammunition: Implications for wildlife and humans, The Peregrine Fund, Boise, Idaho, USA, pp 259-264. Available: http://www.peregrinefund.org/subsites/conference-lead/PDF/0217%20Parish.pdf.

Pattee OH, Wiemeyer SN, Mulhern BM, Sileo L, Carpenter JW. 1981. Experimental lead-shot poisoning in bald eagles. *The Journal of Wildlife Management* 45:806-810.

Pattee OH, Hennes SK. 1983. Bald eagles and waterfowl: The lead shot connection. *Transactions of the 48th North American Wildlife and Natural Resources Conferences* 48:230-237.

Pattee OH, Bloom PH, Scott JM, Smith MR. 1990. Lead hazards within the range of the California condor. *The Cooper Ornithological Society* 92:931-937.

Pattee OH, Carpenter JW, Fritts SH, Rattner BA, Wiemeyer SN, Royle JA, Smith MR. 2006. Lead poisoning in captive Andean condors (*Vultur gryphus*). *Journal of Wildlife Diseases*. 42:772-779.

Pauli JN, Buskirk SW. 2007. Recreational shooting of prairie dogs: a portal for lead entering wildlife food chains. *The Journal of Wildlife Management* 71:103-108.

Peterson MN. 2004. An approach for demonstrating the social legitimacy of hunting. *Wildlife Society Bulletin* 32:310-321.

Platt JB. 1976. Bald eagles wintering in a Utah desert. *American Birds* 30:783-788.

Prosser P, Nattrass C, Prosser C. 2008. Rate of removal of bird carcasses in arable farmland by predators and scavengers. *Ecotoxicology and Environmental Safety* 71:601-608.

Quarterman, J. 1986. Lead. In Mertz W, ed, *Trace Elements in Human and Animal Nutrition.* 5th ed, Vol 2. Academic Press, NY, USA, pp 281-317.

Quortrup ER, Shillinger JE. 1941. 3,000 Wild bird autopsies on western lake areas. *Journal of American Veterinary Medical Association* 99:382-387.

Rabinowitz MB, Hall GS. 2002. Isotopic characterization of six major brands of white basic lead carbonate paint pigments. *Bulletin of Environmental Contamination and Toxicology* 69:617-623.

Rattner BA, Morehouse KA.1994. Ecological tiered testing protocol proposed for registration of shot used to hunt waterfowl. Proceedings of the Fifteenth Annual meeting of the Society of Environmental Toxicology and Chemistry. Abstract 90.

Rattner BA, Franson JC, Sheffield SR, Goddard CI, Leonard NJ, Stang D, Wingate PJ. 2008. Sources and implications of lead ammunition and fishing tackle on natural resources. Technical Review 08-01, The Wildlife Society and American Fisheries Society, Committee on Lead in the Environment, pp 1-62.

Redig PT, Lawler EM, Schwartz S, Dunnette JL, Stephenson B, Duke GE. 1991. Effects of chronic exposure to sublethal concentrations of lead acetate on heme synthesis and immune function in red-tailed hawks. *Archives of Environmental Contamination and Toxicology* 21:72-77.

Rideout BA, Stalis I, Papendick R, Pesser A, Puschner B, Finkelstein ME, Smith DR, Johnson M, Mace M, Stroud R, Brandt J, Burnett J, Parish C, Petterson J, Witte C, Stringfield C, Orr K, Zuba J, Wallace M, Grantham J. 2012. Patterns of mortality in free-ranging California condors (*Gymnogyps californianus*). *Journal of Wildlife Diseases* 48:95-112.

Ringelman JK, Miller MW, Andelt WF. 1992. Effects of ingested tungsten-bismuth-tin shot on captive mallards. *Journal of Wildlife Management* 57:725-732.

Risebrough RW. 2001. Absence of demonstrable toxicity to turkey vultures, *Cathartes aura* of copper and tungsten-tin-bismith-composite pellets. Final Report. U.S. Fish and Wildlife Service, California Condor Recovery Program, Ventura, CA.

Rocke TE, Samuel MD. 1991. Effects of lead shot ingestion on selected cells of the mallard immune system. *Journal of Wildlife Diseases* 27:1-9.

Roscoe DE, Nielsen SW. 1979. A simple quantitative test for erythrocytic protoporphyrin in lead-poisoned ducks. *Journal of Wildlife Diseases* 15:127-136.

Roscoe DE, Widjeskog L, Stansley W. 1989. Lead poisoning of northern pintail ducks feeding in a tidal meadow contaminated with shot from a trap and skeet range. *Bulletin of Environmental Contamination and Toxicology* 42:226-233.

Samuel MD, Bowers EF, Franson JC.1992. Lead exposure and recovery rates of black ducks banded in Tennessee. *Journal of Wildlife Diseases* 28:555-561.

Samuel MD, Bowers EF. 2000. Lead exposure in American black ducks after implementation of non-toxic shot. *Journal of Wildlife Management* 64:947-953.

Sanderson GC, Bellrose FC. 1986. Review of the problem of lead poisoning in waterfowl. Natural History Survey. Special Publication, Champaign, IL. 4. 34 p.

Sanderson GC. 1992. Lead poisoning mortality. Lead Poisoning in Waterfowl. Proceedings of an IWRB Workshop, Brussels, Belgium, 13-15 June. Edited by Deborah J. Pain. 14-18.

Sanderson GC, Anderson WL, Foley GL, Skowron LM, Brawn JD, Seets JW. 1997. Acute toxicity of ingested bismuth alloy shot in game-farm mallards. *Illinois Natural History Survey Bulletin* 35:183-215.

Scheuhammer AM. 1987. The chronic toxicity of aluminum, cadmium, mercury, and lead in birds: A review. *Environmental Pollution* 46:263-295.

Scheuhammer AM, Norris SL. 1996. The ecotoxicology of lead shot and lead fishing weights. *Ecotoxicology* 5:279-295.

Scheuhammer AM, Templeton DM. 1998. Use of stable isotope ratios to distinguish sources of lead exposure in wild birds. *Ecotoxicology* 7:37-42.

Schulz JH, Millspaugh JJ, Bermudez AJ, Gao X, Bonnot TW, Britt LG, Paine M. 2006. Acute lead toxicosis in mourning doves. *Journal of Wildlife Management* 70:413-421.

Shillinger JE, Cottam CC. 1937. The Importance of lead Poisoning in Waterfowl. Division of Wildlife Research, Bureau of Biological Survey. American Wildlife Institute. *Transactions of the Second North American Wildlife Conference*. Jefferson, St. Louis, Missouri, USA.

Sileo L, Fefer SI. 1987. Paint chip poisoning of Laysan albatross at Midway Atoll. *Journal of Wildlife Diseases* 23:432-437.

Sileo LH, Creekmore DJ, Audet L. 2001. Lead poisoning of waterfowl by contaminated sediment in the Coeur d'Alene River. *Archives of Environmental Contamination and Toxicology* 41:364-368.

Simpson VR, Hunt AE, French MC. 1979. Chronic lead poisoning in a heard of mute swans. *Environmental Pollution* 18:187-202.

Snyder NFR, Johnson EV, Clendenen DA. 1987. Primary molt of California condors. *The Condor 89*:468–485.

Stauber E, Finch N, Talcott PA, Gay JM. 2010. Lead poisoning of bald (Haliaeetus leucocephalus) and golden (*Aquila chrysaetos*) eagles in the US inland Pacific/Northwest - An 18 year retrospective study 1991-2008. *Journal of Avian Medicine and Surgery* 24:279-287.

Steenhof KL, Bond, K, Bates K, Leppert LL. 2002. Trends in midwinter counts of bald eagles in the contiguous United States, 1986–2000. Bird Populations 6:21-32.

Steenhof KL, Bond K, Dunn LL. 2008. The midwinter bald eagle survey results and analysis 1986-2005. Final Report. US Geological Survey, National Biological Information Infrastructure, and Northwest Alliance for Computational Science and Engineering. Available from: http://ocid.nacse.org/nbii/eagles/

Stendell RC, Smith RI, Burnham KP, Christensen RE. 1979. Exposure of waterfowl to lead: A nationwide survey of residues in wing bones of seven species, 1972-73. Final Report. US Fish Wildlife Service Special Science Report, Wildlife No. 223. pp1-12.

Stephens RM, Johnson AS, Plumb RE, Dickerson K, McKinstry MC, Anderson SH. 2009. Risk assessment of lead poisoning in raptors caused by recreational shooting of prairie dogs. *Intermountain Journal of Sciences* 13:116-123.

Stevenson AL, Scheuhammer AM, Chan HM. 2005. Effects of nontoxic shot regulation on lead accumulation in ducks and American woodcock in Canada. *Archives of Environmental Contamination and Toxicology* 48:405-413.

Stocek R. 2000. Diet of wintering bald eagles, *Haliaeetus leucocephalus*, In New Brunswick. *Canadian Field Naturalist* 114:605-611.

Strom SM, Langenberg JA, Businga NK, Batten JK. 2009. Lead exposure in Wisconsin birds. In Watson RT, Fuller M, Pokras M, Hunt G, eds, Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA, pp194-201. Available: http://www.peregrinefund.org/subsites/conference-lead/PDF/0205%20Strom.pdf

Stormer FA, Kirkpatrick CM, Hoekstra TW. 1979. Hunter inflicted wounding of white-tailed deer. *Wildlife Society Bulletin* 7:10-16.

Stout JH, Trust KA, Cochrane JF, Suydam RS, Quakenbush LT. 2002. Environmental contaminants in four eider species from Alaska and arctic Russia. *Environmental Pollution*. 119:215-226.

Snyder NFR, Johnson EV, Clenden, DA. 1987. Primary molt of California condors. *The Condor* 89:468-485.

Thiboldeaux R. 2008. The potential for ingestion exposure to lead fragments in venison in Wisconsin. Final Report. Wisconsin Department of Health and Family Services, Madison, WI, pp 1-20.

Thomas VG, Guitart R. 2013. Transition to non-toxic gunshot in Olympic shooting: Policy implications for IOC and UNEP in resolving and environmental problem. *AMBIO* DOI 10.1007/s13280-013-0393-7.

Tranel MA, Kimmel RO. 2009. Impacts of lead ammunition on wildlife, the environment, and human health-A literature review and implications for Minnesota. In Watson RT, Fuller M, Pokras M, Hunt WG, eds, Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. Available: http://www.peregrinefund.org/subsites/conference-lead/PDF/0307%20Tranel.pdf

Trust KA, Miller MW, Ringelman JK, Orme IM. 1990. Effects of ingested lead on antibody production in mallards (*Anas platyrhynchos*). *Journal of Wildlife Diseases* 26:316-322.

USFWS (U.S. Fish and Wildlife Service). 1986. Final Supplemental environmental Impact Statement. Use of lead shot for hunting migratory birds in the United States. US Department of Interior Fish and Wildlife Service. FES 86-16, pp 1-559.

USFWS (U.S. Fish and Wildlife Service). 1995. 50 CFR 20 Migratory Bird Hunting. §20.21, page 10, What hunting methods are illegal? pp 1-36.

USFWS (U.S. Fish and Wildlife Service). 1996. California Condor Recovery Plan, Third Revision. Portland, Oregon, 62 pp.

USFWS (U.S. Fish and Wildlife Service). 1997. Migratory Bird Hunting: Revised Test Protocol for Nontoxic Approval Procedures for Shot and Shot Coating. Federal Register Vol. 62, No. 230 Monday December 1 1997. 50 CFR Part 20 RIN 1018-AB80. Final Rule. 63608-63610. Washington, DC.

USFWS (U.S. Fish and Wildlife Service). 1999. Subpart M –Criteria and schedule for implementing nontoxic shot zones for the 1987-1988 and subsequent waterfowl hunting seasons. RST 50 CFR 7, 23,1999 page 32 §20.143. Final Rule. US Fish and Wildlife Service, Washington, DC, pp 31-36.

USFWS (U.S. Fish and Wildlife Service). 2006. Deer Hunting in the United States: Demographics and trends. Addendum to the 2006 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation. Report 2006-10. Accessed from: http://digitalmedia.fws.gov/cdm/ref/collection/document/id/207

USFWS (U.S. Fish and Wildlife Service). 2009. Final Environmental Assessment Proposal to Permit Take as Provided Under the Bald and Golden Eagle Protection Act. US Fish and Wildlife Service, Division of Migratory Bird Management. Available from: http://www.fws.gov/migratorybirds/CurrentBirdIssues/Management/BaldEagle/

USFWS (U.S. Fish and Wildlife Service). 2012. A review of the third five years of the California condor Reintroduction program in the southwest (2007-2011). Final Report. US Fish and Wildlife Service, Pacific Southwest Office (Region 8), Sacramento, CA.

USFWS (U.S. Fish and Wildlife Service). 2013a. Non-toxic shot regulations for hunting waterfowl and coots in the U.S. Available from: http://www.fws.gov/migratorybirds/CurrentBirdIssues/nontoxic.htm

USFWS (U.S. Fish and Wildlife Service). 2013b. Migratory Bird Hunting; Revisions of Language for Approval of Nontoxic Shot for Use in Waterfowl Hunting. Federal Register/Vol.78, No. 42/Monday, March 4, 2013. 50 CFR Part 20, [Docket No. FWS-R9-MB-2011-0077; FF09M21200-134-FXMB1231099BPP0]. RIN 1018-AY59. Washington, DC.

USFWS (U.S. Fish and Wildlife Service). 2013c. About the Wildlife and Sport Fish Restoration Program. Available from: http://wsfrprograms.fws.gov/Subpages/AboutUs/AboutUs1.htm

USFWS (U.S. Fish and Wildlife Service). 2013d. News and activities non-toxic shot approved for upcoming waterfowl season. Available from: http://www.fws.gov/contaminants/DisplayNews.cfm?NewsID=4DAA500C-3E21-4564-87AA714E9E301C9E

USFWS (U.S. Fish and Wildlife Service). 2013e. California condor (*Gymnogyps californianus*) 5-year review: Summary and evaluation. Final Report. US Fish and Wildlife Service Pacific Southwest Region, Sacramento, CA.

Veit HP, Kendall RJ, Scanlon PF. 1983. The effect of lead shot ingestion on the testes of adult ringed turtle doves (*Streptopelia risoria*). *Avian Diseases* 27:442-452.

Verbeek NA, Caffrey C. 2002. American Crow (*Corvus brachyrhynchos*), The Birds of North America Online, Poole A, ed, Ithaca: Cornell Lab of Ornithology. Available from: The Birds of North America Online: http://bna.birds.cornell.edu/bna/species/647

Vyas NB. 1999. Factors influencing estimation of pesticide-related wildlife mortality. *Toxicology and Industrial Health* 15:186-191.

Vyas NB, Spann JW, Heinz GH, Beyer WN, Jaquette JA, Mengel-Koch JM. 2000. Lead poisoning of passerines at a trap and skeet range. *Environmental Pollution* 107:159–166.

Walters JR, Derrickson SR, Fry DM, Haig SM, John M, Wunderle JM. 2008. Status of the California condor and efforts to achieve its recovery. Final Report. Prepared by the AOU Committee on Conservation, California Condor Blue Ribbon Panel, A Joint Initiative of The American Ornithologists' Union and Audubon California. Available from: https://wiki.umn.edu/pub/ESPM3241W/S11TopicSummaryTeamSeventeen/AOUreport.pdf

Wayland M, Bollinger T. 1999. Lead exposure and poisoning in bald eagles and golden eagles in the Canadian prairie provinces. *Environmental Pollution* 104:341-350.

Wetmore A. 1919. Lead Poisoning in Waterfowl. Final Report No. 793-19. United States Department of Agriculture Bulletin, Washington, DC, USA.

Weyers B, Gluck E, Stoeppler M. 1988. Investigation of the significance of heavy metal contents of blackbird feathers. *Science of the Total Environment* 77:61-67.

Wiemeyer SN, Scott JM, Anderson MP, Bloom PH, Stafford CJ. 1988. Environmental Contaminants in California condors. *Journal of Wildlife Management* 52:238-247.

Wobeser GA. 1997. Diseases of wild waterfowl. In Wobeser G, ed, 2nd Edition. Western College of Veterinary Medicine, University of Saskatchen, Saskatoon, Saskatchewan, Canada. Plenum Press, 233 Spring Street, New York, NY, USA.

Work TM, Smith MR. 1996. Lead exposure in Laysan albatross adults and chicks in Hawaii: Prevalence, risk factors, and biochemical effects. *Archives of Environmental Contamination and Toxicology* 31:115-119.

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