July 5, 2011

The Honorable John Tester
U.S. Senate
Washington, D.C. 20510

Re: S838 and HR 1558 and claims of "no credible science"

Dear Senator Tester:

The Raptor Research Foundation (RRF) is a non-profit scientific society whose primary goal is the accumulation and dissemination of scientific information about raptors (hawks, eagles, falcons and owls). This information is used to inform the public (both scientific and lay) about the role of raptors in nature, and to promote the conservation of raptors whose populations are threatened by human activities. The RRF's membership consists of academic researchers, government agency employees, students, and others interested in birds of prey. The RRF was organized in 1966 and started publishing a scholarly journal, The Journal of Raptor Research, in 1967. Though based in the United States, the RRF is an international organization with nearly 1,000 members, currently including members in over 50 countries.

We have enclosed a summary, with citations to the science, outlining what we know about the effects of lead from spent ammunition on wildlife, especially birds of prey.

We bring this to your attention as a result of misinformation issued in press releases and elsewhere related to bills S838 and HR1558 introduced by the Congressional Sportsman's Caucus. The claim of "no credible science" behind the conclusion that lead from spent ammunition is negatively impacting wildlife is deeply troubling to us a body of professional scientists.

Contrary to what you may have been told, lead from spent ammunition does indeed contaminate wildlife, often with debilitating and fatal consequences. For decades, scientists from around the world have been collecting scientific evidence that lead from spent ammunition causes death and misery in wildlife that could be prevented by switching to non-lead ammunition, such as solid copper bullets. California condors are affected at the population level. This species could recover from its endangered status once lead is absent from its food, which often includes the remains of hunter harvested game.

Please contact us if you have questions about the effects of lead ammunition in wildlife.

Sincerely yours,

Ruth Tingay, Ph.D.
President, Raptor Research Foundation
www.raptorresearchfoundation.org

RAPTOR RESEARCH FOUNDATION WHITE PAPER
Prepared by RRF Conservation Committee, July 2011

SCIENTIFIC RESEARCH SHOWS THAT LEAD (Pb) FROM SPENT AMMUNITION CAUSES DEATH AND SICKNESS TO BALD EAGLES, GOLDEN EAGLES AND OTHER WILDLIFE, AND LIMITS THE REESTABLISHMENT OF THE CALIFORNIA CONDOR POPULATION.

Much of our recent knowledge of lead exposure in wildlife derives from research in conjunction with efforts to restore California condors to the wilds of northern Arizona, southern Utah, and southern California. Following a long period of decline, the last 22 condors in existence were removed to captive breeding facilities in the 1980s to produce young for the reestablishment of self-sustaining populations. Releases in the Grand Canyon region began in 1996, and there are now 73 free-flying condors in Arizona, eight of which were produced by wild pairs.

The Arizona condor population began showing evidence of lead poisoning in the early 2000s when the birds began travelling more widely in the landscape and foraging on their own. Condor recovery biologists were able to trace the source to deer hunting areas on the Kaibab Plateau because each condor was radio-tagged (Hunt et al. 2007), and they surmised that the birds might be acquiring lead from the remains of gun-killed deer. X-rays of 38 deer carcasses showed that 94% contained bullet fragments; 90% of 20 gut piles contained fragments, and half of them showed at least 100 fragments (Hunt et al. 2006). Meanwhile, increasing numbers of condors were being exposed to lead, and lead poisoning had become the principle source of mortality in the population (Parish et al. 2006, 2009). Each year, biologists found it necessary to test (up to 100%) and treat substantial numbers (up to 70%) of condors to prevent sickness and death (Parish et al. 2007, 2009). Demographic analysis showed that a self-sustaining population could not be established without such intervention and a reduction in lead prevalence (Woods et al. 2007, Cade 2007, Green et al. 2008).

In response to these findings, the Arizona Game and Fish Department began a program in 2005 to reduce lead exposure by offering boxes of lead-free ammunition to hunters drawing big game permits in the condors' range (Sieg et al. 2009). The results have been encouraging in that hunter volunteer participation in the program has reached 90 percent, and the incidence of lead exposure and deaths deriving from the Arizona landscape is much reduced (Sieg et al. 2009, Sieg in. lit. 2009). However, the condors have since extended their range into Utah where such a program is only just beginning.

Prior to recent studies of bullet fragmentation, focus on lead contamination in wildlife centered on the use of lead shotgun pellets. These were banned for use in waterfowl hunting in the United States and Canada because large numbers of waterfowl were dying from ingesting lead shot, estimated at 1.4 million deaths per year (Anderson et al. 2000), and because of the secondary poisoning of bald eagles that ate crippled waterfowl (Kramer and Redig 1997). Ingestion of lead shotgun pellets continues even today in upland areas, however, as dove hunters annually deposit billions of lead pellets on the land surface, which the birds mistake for seeds and grit. According to experiments, most die as a result (Schulz et al. 2006a), and the total number dying each year from lead poisoning is estimated at 8.8 to 15 million doves (Schulz 2006b). Ingested shotgun pellets also poison quail, pheasants, and other birds (Friend et al. 2009, Fisher et al. 2006, see Watson et al. 2009) and may secondarily poison raptors and scavengers.

Death by lead poisoning is a cruel process. Lead substitutes in the body for calcium and other metals and is thus incorporated into nerve tissue throughout the body. Condors and eagles dying of lead poisoning often become emaciated as the nerves controlling muscle contraction and food movement in the crop and stomach are inactivated. Other manifestations include depression, anemia, vomiting, diarrhea, ataxia, blindness, and seizures (Kramer and Redig 1997). As the bird grows weak, it loses its ability to fly and may hide and starve in ground-cover where it is unlikely to be found and diagnosed. For that reason, lead poisoning deaths are thought to be grossly under-reported.

Lead-exposed birds also experience sub-lethal effects (Redig et al. 1991). Herring gulls dosed with lead showed impacts on growth, motor coordination, behavioral development, thermoregulation, depth perception, and individual recognition in both the laboratory and the wild (Burger and Gochfield 2000). None of this is surprising, given the central importance of calcium in nerve function and the propensity of lead to replace it.

Based on recent studies in human populations, the implications of even miniscule levels of sub-lethal lead exposure to wildlife are relevant. In the mid-20th Century, the amount of lead in the bloodstream of a child considered in need of medical intervention was considered to be 60 micrograms per deciliter (µg/dl), whereas today it is 10 µg/dl. Children sustain permanent cognitive damage when they show an average of only 7.5 µg/dl in blood before the age of five (Lanphear et al. 2005). Prenatally exposed fetuses are impacted as well (Schnaas et al. 2006). Lead impairs motor function (Cecil et al. 2008), causes attention dysfunction (Braun et al. 2006), and has been implicated in causing criminal behavior (Needleman et al. 2002, Wright et al. 2008). Lead is also implicated in decreased growth (Hauser et al. 2008), decreased brain volume (Cecil et al. 2008), spontaneous abortion (Borja-Aburto et al. 1999), kidney damage (Ekong et al. 2006), cancer, and cardiovascular disease (Menke et al. 2006, Lustberg and Silbergeld 2002). Many other studies show that lead exposure is harmful and that even very small amounts of lead can have permanent, debilitating, sub-lethal effects. The consensus among medical researchers is that there is no safe level of lead exposure in young children (CDC 2005).

Lead from ammunition is a threat to condor populations. By itself, lead may not threaten eagle populations, but understanding that mortality can be additive, and that each human-caused mortality factor moves the demographic balance point closer to the threshold of decline, the precautionary principle dictates that any human caused mortality that can be avoided, should be avoided. The deaths and misery unnecessarily inflicted on eagles, condors, and other wildlife by lead poisoning from spent bullets and shot should not be ignored.

Effective substitutes for lead shotgun pellets and bullets exist. Research shows that switching to non-lead ammunition would benefit condor populations, and improve survival and reduce unnecessary suffering in eagles and other wildlife.

References

Anderson, W. L., S. P. Havera, and B. W. Zercher. 2000. Ingestion of lead and nontoxic shotgun pellets by ducks in the Mississippi Flyway. Journal of Wildlife Management 64:848-857.

Borja-Aburto, V. H., I. Hertz-Picciotto, M. R. Lopez, P. Farias, C. Rios, and J. Blanco. 1999. Blood lead levels measured prospectively and risk of spontaneous abortion. American Journal of Epidemiology 150:590-597.

.

Braun, J. M., R. S. Kahn, T. Froehlich, P. Auinger, and B. P. Lamphear. 2006. Exposure of environmental toxicants and attention deficit hyperactivity disorder in U.S. children. Environmental Health Perspectives 114:1904-1909.

Burger, J. and M. Gochfield. 2000. Effects of lead on birds (Laridae): a review of laboratory and field studies. Journal of Toxicology and Environmental Health, Part B 3:59-78.

Cade, T. J. 2007. Exposure of California condors to lead from spent ammunition. Journal of Wildlife Management 71:2125-2133.

CDC. 2005. Preventing lead poisoning in young children. Centers for Disease Control and Prevention, Atlanta, USA.

Cecil, K. M., C. J. Brubaker, C. M. Adler, K. N. Dietrich, M. Altaye, J. C. Egelhoff, S.Wessel, I. Elangovan, R. Hornung, K. Jarvis, and B. Lanphear. 2008. Decreased brain volume in adults with childhood lead exposure. PLoS Medicine 5:741-750.

Ekong, E. B., B. G. Jaar, and V. M. Weaver. 2006. Lead-related nephrotoxicity: a review of the epidemiologic evidence. Kidney International 70:2074-2084.

Fisher, I. J., D. J. Pain, and V. G. Reynolds. 2006. A review of lead poisoning from ammunition sources in terrestrial birds. Biological Conservation 131:421-432.

Friend, M., J. C. Franson, and W. L. Anderson. 2009. Biological and societal dimensions of lead poisoning in birds in the USA. *In* Watson, R.T., 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. DOI 10.4080/ilsa.2009.0104

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):e4022.

Hauser, R., O. Sergeyev, S. Korrick, M. M. Leem B. Revich, E. Gitin, J. S. Burns, and P. L.Williams. 2008. Association of blood lead levels with onset of puberty in Russian boys. Environmental Health Perspectives 116:976-980.

Hunt, W. G., W. Burnham, C. N. Parish, K. Burnham, B. Mutch, and J. L Oaks. 2006. Bullet fragments in deer remains: implications for lead exposure in scavengers. Wildlife Society Bulletin 34:168-171.

Hunt, W. G., C. N. Parish, S. C. Farry, T. G. Lord, and R. Sieg. 2007. Movements of introduced California condors in Arizona in relation to lead exposure. In: A. Mee, L. S. Hall (eds.). California Condors in the 21st Century.Washington DC: Special Publication of the American Ornithologists' Union and Nuttall Ornithological Club:79–96.

Hunt, W. G., R. T. Watson, J. L. Oaks, C. N. Parish, K. K. Burnham, R. L. Tucker, J. R. Belthoff, and G. Hart. 2009. Lead bullet fragments in venison from rifle-killed deer: potential for human dietary exposure. PLos ONE 4(4):c5330.

Kramer, J. L. and P.T. Redig. 1997. Sixteen years of lead poisoning in eagles, 1980-95: An epizootiologic view. Journal of Raptor Research 31:327-332.

Lanphear, B. P., R. Hornung, J. Khoury, K. Yolton, P. Baghurst, et al. 2005. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environmental Health Perspectives 113:894-899.

Lustberg M. and E. Silbergeld. 2002. Blood lead levels and mortality. Archives of Internal Medicine 162:2443-2449.

Menke, A., P. Muntner, V. Batuman, E. K. Silbergeld, and E. Guallar. 2006. Blood lead below 0.48 µmol/L (10µg/dL) and mortality among US adults. Circulation 114:1388-1394.

Needleman H. L., C. McFarland, R. B. Ness, S. E. Fienberg, and M. J. Tobin. 2002. Bone lead levels in adjudicated delinquents: a case control study. Neurotoxicol Teratol. 24:711-717.

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.

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. Pp. 259-265 *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

Redig, P. T., Lawler, E. M., Schwartz, S., Dunnette, J. L., B. Stephenson, and G. E. Duke. 1991. Effects of chronic exposure to sublethal concentrations of lead acetate on heme synthesis and immune function in red-tailed hawks. Archives of EnvironmentalContamination and Toxicology 21:72–77.

Schnaas, L., S. J. Rothenberg , M-F. Flores, S. Martinez, C. Hernandez, C. Osorio, et al. 2006. Reduced intellectual development in children with prenatal lead exposure. Environmental Health Perspectives 114:791-797.

Schulz, J. H., J. J. Millspaugh, A. J. Bermudez, X. Gao, T. W. Bonnot, L. G. Brit, and M. Paine. 2006a. Lead toxicosis in mourning doves. Journal of Wildlife Management 70:413-421.

Schulz, J. H., P. I. Padding, and J. J. Millspaugh. 2006b. Will mourning dove crippling rates increase with nontoxic-shot regulations? Wildlife Society Bulletin 34:861-865.

Sieg, R., K.A. Sullivan, and C.N. Parish. 2009. Voluntary lead reduction efforts within the Northern Arizona range of the California condor. *In* Watson, R.T., 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. DOI 10.4080/ilsa.2009.0309

Watson, R.T., M. Fuller, M. Pokras, and W.G. Hunt. 2009. Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. Conference Proceedings, The Peregrine Fund, Boise, Idaho.

Woods C. P., W. R. Heinrich, S. C. Farry, et al. 2007. Survival and reproduction of California condors released in Arizona. In: A. Mee and L. S. Hall (eds). California Condors in the 21st Century. Washington DC: Special Publication of the American Ornithologists' Union and Nuttall Ornithological Club: 57–78.

Wright, J.P., K. N. Dietrich, M. D. Ris, R. W. Hornung, S. D. Wessel, B. P. Lanphear, et al. 2008. Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. PLoS Med 5:732-740.