VETERINARY TOXICOLOGY

Lead Exposure from Backyard Chicken Eggs: A Public Health Risk?

Adrienne C. Bautista · Birgit Puschner · Robert H. Poppenga

Published online: 19 June 2014 © American College of Medical Toxicology 2014

Abstract Although the USA has made significant strides in reducing lead exposure, new and emerging sources are raising cause for public concern. Recent reports of finding lead in eggs from chickens raised in urban gardens has highlighted the need to consider the potential health risks of consuming eggs from backyard chickens. Following the detection of 0.33 μ g/g lead in the edible portion of eggs submitted for lead analysis from a backyard chicken owner, further investigation was conducted to determine the source and extent of lead exposure in the flock. Several birds, almost two dozen eggs, and environmental samples were submitted to the California Animal Health and Food Safety Laboratory for further testing. Lead was detected in the blood, liver, kidney, and bone at varying concentrations in all birds but was not detected in the muscle tissue. All egg shells contained detectable amounts of lead, while only a little over half of the edible portion of the eggs contained lead. The detected concentrations in the edible portion approached or exceeded the recommended threshold of lead consumption per day that should not be exceeded by young children if a child consumed one average-sized egg. Peeling paint from a wooded structure adjacent to the flock's coop was the likely lead source containing 3,700 µg/g lead. Thus, removal of the chickens from the source and periodic testing of eggs for lead were recommended. This case illustrates the need for consumers and health care workers to be

A. C. Bautista (⊠) · B. Puschner · R. H. Poppenga California Animal Health and Food Safety Laboratory System School of Veterinary Medicine, University of California, 620 West Health Sciences Drive, Davis, CA 95616, USA e-mail: acbautista@cahfs.ucdavis.edu

B. Puschner

aware of potential sources for lead exposure such as backyard chickens.

Keywords Lead · Chicken · Egg

Introduction

The Centers for Disease Control and Prevention (CDC) estimates that there are more than 500,000 children in the US aged 1-5 years with blood lead levels above 5 µg/dl, the reference level at which public health actions should be initiated [1]. In reality, no safe blood lead level in children has been identified. Thus, public health efforts have been aimed at preventing exposure to lead. Sources of exposure include air, water, and soil as well as concentrated lead sources such as lead-based paint, automotive batteries, fishing sinkers, shot gun pellets, sewage sludge, lead mine tailings, oil and gasoline, as well as lead contaminated pottery and jewelry, and lead-based inks used in food packaging such as candy wrappers [2, 3]. Much of the emphasis on prevention has been focused on lead-based paints since they are the primary source of childhood lead exposure and, recently, on the lead levels in candy products likely to be consumed frequently by small children [4]. However, the concern for lead exposure through the consumption of eggs was recently made public by the New York Times [5]. Although eggs bought in the supermarket are known to contain negligible amounts of lead [6], this may not be the case for eggs collected and then consumed from backyard poultry.

In California, a substantial increase in backyard poultry ownership has been noted based on the increase in backyard poultry submissions to the California Animal Health and Food Safety (CAHFS) laboratory [7]. In addition, over the past few years, the CAHFS laboratory has identified toxic liver lead concentrations in a number of backyard chickens submitted

Department of Molecular Biosciences, School of Veterinary Medicine, University of California, 1089 Veterinary Medicine Drive, Davis, CA 95616, USA

for routine diagnostic work-up involving necropsy and ancillary testing [8]. Nonetheless, the true frequency of clinical or subclinical lead poisonings in backyard chickens is probably underestimated since many of these birds do not routinely undergo ante- or postmortem diagnostic testing. Therefore, a more substantial risk may exist for the backyard poultry consumer than has previously been recognized. Follow-up testing of eggs from the backyard flocks has also uncovered detectable amounts of lead in the eggs which pose a potential public health risk to individuals repeatedly consuming them. Based on the concentration of lead detected in one of these eggs (72 µg/kg lead in mixed yolk and albumen), it has been estimated that consumption of one average size 60 g egg (roughly equivalent to a commercially labeled large egg) would approach the designated threshold of 6 µg of lead per day from all dietary sources that should not be exceeded by young children [8]. The case discussed herein highlights the importance of the need for consumers, veterinarians, and physicians to be aware of this risk and have access to testing resources and management guidelines for prevention.

Case Study

In January 2014, following publication of an article in a regional newspaper highlighting the potential for lead contamination of eggs in backyard poultry [9], a backyard chicken owner submitted three eggs to CAHFS for lead analysis. No history about the backyard flock from which the eggs were collected was given by the owner upon submission. The edible portions, the yolk and albumen, from each of the three eggs were pooled for lead analysis by graphite furnace atomic absorption spectrometry (GFAAS). This method allows for the detection of lead at or above 0.05 μ g/g. A lead concentration of 0.33 μ g/g wet weight was detected in the pooled edible contents. Based on the confirmed lead exposure in the submitted eggs, a recommendation was made to the owner to identify the source of the lead and to submit blood samples from the chickens in the flock to assess individual animal exposure. A field investigation was also undertaken to help identify a possible lead source. The flock consisted of ten chickens housed in an approximately 100 sq ft outdoor coop constructed of wood and chicken wire with one wall adjacent to an old shed with white flaking paint. The chickens had been purchased in April of the previous year and were continuously housed in the coop. The eggs were consumed by the owners who had no children in the household and excess eggs were given to neighbors. The owner reported no recent deaths and all chickens appeared in good health. No signs of lead poisoning such as weakness, diarrhea, or paralysis were noted [10, 11]. Several environmental samples, including soil, water, and painted wood found within the chickens' coop, were collected along with an additional 21 eggs. Interestingly, the chickens were noted to peck at the white paint flakes that fell to the ground during inspection of the painted wood wall. Four chickens with no clinical signs of lead toxicosis were also submitted for tissue, blood, and bone lead analysis. Tissue and bone lead concentrations were determined using inductively coupled plasma atomic emission spectrometry (ICP-AES), while blood and egg (shell and edible portion) lead analysis was conducted using GFAAS.

Tissue concentrations were quite variable ranging from non-detectable in both the breast and thigh muscle to as high as 490 μ g/g wet weight in the femoral bone (Table 1). All four chickens had detectable amounts of lead in the liver, kidney, and bone. Lead was not detected in any of the muscle tissues analyzed. Blood lead concentrations in all four chickens were above a concentration considered consistent with toxicosis (>0.35 µg/ml) [12], yet none showed any clinical signs of lead toxicosis prior to euthanasia. In addition, lead was detected in the shell of all 21 submitted eggs at concentrations ranging from 0.075 to 1.8 μ g/g wet weight. Interestingly, lead was detected in the edible portion of only 12 eggs; 8 eggs had non-detectable lead concentrations (Table 2). The results of the environmental samples showed the highest concentration of lead, $3,700 \ \mu g/g$ (0.37 %), in the wood paint as the most likely primary lead source. The coop soil, which contained flecks of the wood paint, also had moderately high concentrations of lead ranging from 410 to 560 µg/g. Lead analysis results of the environmental samples are summarized in Table 3.

Discussion

In this particular incident, the highest concentrations of lead were found in the femoral bone, followed by the kidney and liver. This is consistent with prior reports of lead toxicosis in chickens [13, 14]. Following the ingestion of lead, there are dose-related increases in the blood, kidney, liver, and bone [15]. Bone serves as a reservoir for lead and, in mammals, may be mobilized by physiological processes such as pregnancy and lactation [16]. Lead was not detected in the breast or thigh muscle of the chickens submitted for analysis despite the detection of high concentrations of lead in the blood and other tissues. Some studies have reported high concentrations of lead in the muscle [14], whereas others have reported very low to non-detectable levels [17, 18]. The variability in finding a detectable amount of lead in the muscle tissue could be due to individual genetic differences in the metabolism and distribution of lead, dietary influence, length of exposure, or timing of sample collection. In one study looking at the modification of lead toxicity by dietary sulfur amino acids, the addition of methionine or cystine to the diet resulted in the reduction of organ lead concentrations, including the muscle [19]. Another study looking at the toxic effects of ingested

	Liver (µg/g)	Kidney (µg/g)	Blood (µg/ml)	Breast muscle ($\mu g/g$)	Thigh muscle $(\mu g/g)$	Thigh bone $(\mu g/g)$
Bird 1	0.4	2.5	0.73	ND	ND	47
Bird 2	0.13	0.81	0.38	ND	ND	63
Bird 3	0.38	2.5	0.57	ND	ND	220
Bird 4	1.2	5	1.9	ND	ND	490

Table 1 Lead concentrations detected in various biologic samples (wet weight)

ND not detected (detection limit of 0.05 μ g/g)

lead shot over time found that after 1 week of exposure, appreciable concentrations of lead could not be detected in the breast muscle. However, 12 weeks after ingestion, a very low but detectable amount of lead was found in the breast muscle [17]. Thus, depending on the circumstances, the muscle may or may not contain detectable lead and become a potential source for exposure by the consumer.

Eggs accumulate lead in the shell as well as in the edible portion [14, 20]. Previous studies have reported that little to no lead can be detected in the albumen, while the yolk often contains much higher concentrations of lead [14, 17, 20]. We chose to combine the yolk and albumen for lead analysis in order to more closely simulate consumer exposure such as eating an egg for breakfast. The lead levels detected in the edible portion in this case were quite variable ranging from non-detectable to as high as 0.97 µg/g. A 2003 study on lead

Table 2 Lead concentrations detected in the shell and edible portion (yolk and albumen) of eggs (wet weight)

Egg number	Edible portion ($\mu g/g$)	Shell (µg/g)
18	0.19	1.8
1	0.39	1
6	0.23	0.83
19	0.16	0.78
11	ND	0.76
17	0.16	0.62
10	0.97	0.61
3	ND	0.58
2	0.12	0.5
14	0.33	0.46
5	0.14	0.35
21	0.27	0.3
12	0.12	0.28
8	0.056	0.22
13	ND	0.16
15	ND	0.16
20	ND	0.15
16	ND	0.14
4	ND	0.11
7	ND	0.076
9	ND	0.075

contamination in a small Iowa farm flock of chickens exposed to lead-based paint chips found a strong correlation between blood lead levels and the lead content of egg yolks [14]. In this case study, we could not determine the relationship between the concentration of lead in the blood and lead levels found in eggs, as the chicken of origin for each egg submitted was unknown. However, one would assume that the chickens with higher blood lead levels would have eggs with higher detectable concentrations of lead. There are currently no data on possible day-to-day variation in the amount of lead deposited in eggs following lead exposure and the potential influence diet, metabolism, and other factors may have on egg lead deposition.

Lead is consistently detected in egg shells when chickens are exposed to lead [14, 20]. In this case, lead was detected in every egg shell analyzed. The consistent finding of lead in the shell regardless of the amount of lead in the edible portion is intriguing and points to a continuous pathway for lead excretion in the chicken. Shell calcification utilizes Ca²⁺ from intestinal, dietary absorption as well as from mobilization of bone Ca²⁺ reserves [21]. In chickens exposed to lead, the divalent lead cation (Pb^{2+}) may utilize the pathway of calcium deposition in the egg shell. Further studies are needed to determine if lead found in egg shells is derived from the intestinal pool, which would be a reflection of recent dietary exposure, or from the bone storage pool, a reflection of chronic lead exposure or both. Nonetheless, the finding of lead in egg shells may represent another indirect source of lead exposure for humans. Many backyard gardeners will use compost to fertilize their gardens. If the lead-containing shells

Table 3 Lead concentrations detected in various environmental samples (wet weight)

ND not detected (detection limit of $0.05 \ \mu g/g$)

	Lead (µg/g)
Water	ND
Feed	1.1
Garden soil close to fence	120
Mid garden soil	31
Coop soil 1	560
Coop soil 2	430
Coop soil 3	410
Feces in coop	220
Paint from wood	3,700

are being placed in the compost pile and then being used on growing edible plants, there is the potential for uptake of the lead by the plants [22] as well as surface contamination of the plants. In addition, backyard chickens can be re-exposed to lead if the shells are consumed by them. Thus, it is imperative that chickens not be fed the leftover egg shells in order to prevent re-exposure.

Of great concern from a public health standpoint was the lack of any sign of illness in the chickens from this particular incident. It is well known that the severity of clinical signs does not necessarily correlate with the concentration of lead detected in blood. Thus, relatively high concentrations can be associated with rather mild clinical signs whereas lower concentrations can be associated with more severe signs [20, 23]. This can be problematic from a public health standpoint. Backyard chickens may not show signs of illness with high levels of lead in the blood; lead is then incorporated into the egg which the owner in all likelihood will consume, sometimes on a daily basis. The US Food and Drug Administration (FDA) has not established regulatory limits for concentrations of lead in edible tissues of poultry with the exception of arsenic. In 2006, the FDA recommended a maximum level of 0.1 μ g/g of lead in candy likely to be consumed by children [24]. In addition, it has been suggested that children not consume more than 6 µg of lead per day from all sources [25]. From eggs collected in this case, the consumption of one egg could result in an oral exposure of lead ranging from just below (4.5 μ g) to 18 times as much as the recommended level (108 µg). The adverse health effects of lead exposure, especially in pregnant women and young children, have been well documented [3, 26]. Prenatal and childhood lead exposure has been associated with behavioral disorders, ADHD, and IQ deficits [27, 28]. In addition, childhood lead exposure has been associated with decreased brain volume in adults, indicating that lifelong effects persist [29]. Therefore, consumers need to be made aware of potential sources, including their own backyard chickens, in order to prevent exposure and potentially lifelong health effects.

The following recommendations may help backyard chicken owners reduce their exposure to lead. In backyard chickens with no prior history of lead toxicosis and in which exposure to lead is not known by the owner, we recommend having the eggs tested for lead. Initially, several eggs may be pooled for testing to assess the potential risk. If lead is detected, further investigation should be conducted to determine the source to prevent further exposure to the chickens. In addition, after consulting with a veterinarian, the owner may consider submitting whole blood samples from individual chickens for lead testing, in order to determine which birds may have been exposed. Follow-up testing of eggs at periodic intervals is also recommended to ensure that the consumption of lead contaminated eggs at levels potentially harmful to children and adults does not occur. In addition, we advise that owners not use the egg shells for composting or re-feed them to the chickens. For information regarding lead abatement programs, owners should contact their local or state public health departments. In addition, the Centers for Disease Control and Prevention has (www.cdc.gov) many resources including current recommendations on screening, prevention, and abatement of lead.

The incidence of lead exposure has significantly decreased over the last few decades, mainly through the phasing out of the use of lead in gasoline and paint as well as the reduction of lead in industrial emissions, water, and consumer products [30]. However, lead exposure remains a serious public health problem. The increase in backyard chicken ownership, especially in urban and suburban areas, brings with it another potential source for lead exposure through the consumption of eggs or the use of the egg shells for fertilizing backyard gardens. Thus, consumers as well as health care workers need to be aware of new and emerging sources such as backyard chickens.

Acknowledgments The authors would like to thank Ian Holser for his contribution to this case report.

Conflicts of Interest The author(s) declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

- CDC (2013) Blood lead levels in children aged 1–5 years—United States, 1999–2010. Morb Mortal Wkly Rep 62(13):245–248
- Mahaffey KR (1977) Quantities of lead producing health effects in humans: sources and bioavailability. Environ Health Perspect 19: 285–295
- Bushnell PJ, Jaeger RJ (1986) Hazards to health from environmental lead exposure: a review of recent literature. Vet Hum Toxicol 28(3): 255–261
- Levin R et al (2008) Lead exposures in US children, 2008: implications for prevention. Environ Health Perspect 116(10):1285–1293
- Scelfo J (2012) High lead found in city-sourced eggs. In: The New York Times. New York Times Company, New York
- Pagan-Rodriguez D et al (2007) Cadmium and lead residue control in a hazard analysis and critical control point (HACCP) environment. J Agric Food Chem 55(4):1638–1642
- Stinson S, Mete A (2013) Popular backyard flock program reduces biosecurity risks of amateur production. Calif Agric 67(4):203–209
- Roegner A et al (2013) Public health implications of lead poisoning in backyard chickens and cattle: four cases. Vet Med Res Rep 4:11– 20
- 9. Pierce P (2013) Keeping the lead out of eggs. In: San Francisco Chronicle. Hearst Communications, Inc, San Francisco
- Mazliah J et al (1989) The effects of long-term lead intoxication on the nervous system of the chicken. Neurosci Lett 101(3):253–257
- Thomas EF, Shealy AL (1932) Lead arsenate poisoning in chickens. J Agric Res 45(5):317–319

- Gwaltney-Brant S (2004) Lead. In: Plumlee KH (ed) Clinical veterinary toxicology. Mosby, St. Louis, pp 204–210
- Vengris VE, Mare CJ (1974) Lead poisoning in chickens and the effect of lead on interferon and antibody production. Can J Comp Med 38(3):328–335
- Trampel DW et al (2003) Lead contamination of chicken eggs and tissues from a small farm flock. J Vet Diagn Investig 15(5):418–422
- Bakalli RI, Pesti GM, Ragland WL (1995) The magnitude of lead toxicity in broiler chickens. Vet Hum Toxicol 37(1):15–19
- (ATSDR), A.f.T.S.a.D.R (2007) In: U.S.D.o.H.a.H. Services (ed) Toxicological profile for lead. Public Health Service, Atlanta
- Hirai M, Kawamoto T, Kodama Y (1991) Toxic effects of ingested lead shots in domestic fowls. Biol Trace Elem Res 30(3):291–308
- Havera SP, Wood SG, Georgi MM (1992) Blood and tissue parameters in wild mallards redosed with lead shot. Bull Environ Contam Toxicol 49(2):238–245
- Latta DM, Donaldson WE (1986) Modification of lead toxicity and organ distribution by dietary sulfur amino acids in chicks (Gallus domesticus). Comp Biochem Physiol C 84(1):101–104
- Mazliah J et al (1989) The effect of chronic lead intoxication in mature chickens. Avian Dis 33(3):566–570

- Bar A (2009) Calcium transport in strongly calcifying laying birds: mechanisms and regulation. Comp Biochem Physiol A Mol Integr Physiol 152(4):447–469
- 22. Pourrut B et al (2011) Lead uptake, toxicity, and detoxification in plants. Rev Environ Contam Toxicol 213:113–136
- Carpenter JW et al (2003) Experimental lead poisoning in turkey vultures (Cathartes aura). J Wildl Dis 39(1):96–104
- 24. Administration, U.S.F.a.D (2006) Lead. In: U.S.D.o.H.a.H. Services (ed) Candy likely to be frequently consumed by small children: recommended maximum level and enforcement policy. Center for Food Safety and Applied Nutrition, College Park
- 25. Register, F. p. 33860
- 26. Bellinger DC (2004) Lead. Pediatrics 113(4 Suppl):1016-1022
- Bellinger DC (2011) The protean toxicities of lead: new chapters in a familiar story. Int J Environ Res Public Health 8(7):2593–2628
- Wright JP et al (2008) Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. PLoS Med 5(5):e101
- Cecil KM et al (2008) Decreased brain volume in adults with childhood lead exposure. PLoS Med 5(5):e112
- CDC (1997) Update: blood lead levels—United States, 1991–1994. Morb Mortal Wkly Rep 46(7):141–146