

Lead contamination in backyard chicken layer flocks in California

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Abstract. Backyard layer chickens may be exposed to a variety of metals in the environment, including lead. The potential public health concerns associated with lead exposure prompted us to systematically screen liver samples from backyard layers submitted to the diagnostic laboratory to estimate the prevalence of lead exposure. Over a period of 1 y, we tested 1,476 chicken livers, of which 45 were found to have lead concentrations of 0.9–41 µg/g. The lead-positive cases were investigated by follow-up questions to the bird owners on the environment, general management of the flock, and egg consumption of family members. Lead concentrations in 14 pooled egg samples were determined, and a conservative estimate of daily exposure of family members to lead was made based on egg consumption. In some cases, estimated daily lead intake exceeded the recommended limits for lead consumption in children. Analysis of feed, water, and environmental samples did not identify a source of exposure in most cases. Only 34% of owners of lead-positive birds submitted eggs or environmental samples, indicating a lack of interest or financial concerns. In most cases, neither the case history nor postmortem findings were indicative of lead intoxication; without systematically testing all birds, some cases could have been missed. Our study highlights the need for backyard chicken owners, veterinarians, and public health personnel to be aware of the risk of lead exposure and undertake preventive and surveillance measures.

Key words: backyard chickens; biomonitor; egg; lead.

Backyard layer chickens are becoming increasingly common as a source of eggs in both rural and urban areas with the widespread belief that home-raised chicken eggs are healthier, safer, and more sustainable alternatives to commercial store-bought eggs.¹¹ However, these birds are potentially exposed to heavy metals, pesticides, and other chemicals in the environment. Among the heavy metals that can be transferred to birds from the environment, lead is important because of its toxicity and potential to enter the human food chain through eggs and meat.^{14,16}

According to the Centers for Disease Control and Prevention (CDC), there are ~500,000 U.S. children 1–5 y old with blood lead concentrations >0.24 µmol/L (5 µg/dL), the reference level at which the CDC recommends public health actions be initiated.¹² With reports of lead poisoning in Michigan and California (Lead poisoning afflicts neighborhoods across California, 2017, <https://www.reuters.com/article/us-usa-lead-california-exclusive-idUSKBN16T18Y>), public awareness of the detrimental effect of lead has increased.⁷ Lead is ubiquitous in the environment and hence it is found at low concentrations in many organisms. Sources of lead contamination of soil, water, and air include lead-based paints, leaded gasoline, lead pipes, industrial effluents, alloy processing plants, and oil.³ Lead exposure also occurs from consuming plants and grains grown in lead-contaminated soil or consumption of tissues of lead-exposed animals.² However, one overlooked source of lead is backyard chicken

eggs if chickens are exposed as a result of foraging in lead-contaminated environments. The most likely source of lead in soils is assumed to be from lead-based paint if birds are kept near old buildings^{11,13,14} or, less likely, from spent lead shot from shooting activities.¹⁰ The potential for lead exposure through eggs was described in a study of chicken eggs collected from New York City community gardens.¹³ We published a case report on high concentrations of lead in eggs and tissues of a flock housed in a lead-contaminated environment in Sacramento, CA.¹ However, this was a case in a single household. Additionally, lead has been detected periodically in chickens submitted for postmortem during routine metal screening as part of our avian influenza (AI) surveillance program even when no clinical signs of lead toxicosis were noted. Based on this background, we sought to understand the extent to which lead contamination was prevalent in backyard chicken flocks of California. The California Animal Health and Food Safety (CAHFS) laboratory system consists of branches at Davis, Tulare, Turlock, and San Bernardino. Cases submitted for postmortem examinations

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to the 4 branches of the CAHFS were assumed to be generally representative of the State of California. We discuss herein our findings over a period of 1 y from October 1, 2015 to September 30, 2016, following up on cases in which lead was detected and estimating lead exposure to family members based on lead concentrations in eggs and estimated egg consumption.

Liver tissues from layer chickens were screened for metals (arsenic, cadmium, copper, iron, mercury, manganese, molybdenum, lead, and zinc) by inductively coupled plasma-optical emission spectrometry. Metal concentrations were reported on a wet-weight basis at a reporting limit of 1 $\mu\text{g/g}$. There are no established tissue lead concentrations that are definitive for lead toxicosis in chickens. For the purpose of our study, the liver lead concentrations described in wild birds were used to categorize the chickens into various exposure groups: positive ($\leq 2 \mu\text{g/g}$), significantly exposed (2–8 $\mu\text{g/g}$), or intoxicated ($> 8 \mu\text{g/g}$).⁸

Owners of chickens in which lead was detected were contacted by phone and asked a set of standard questions examining 1) the details of the premises (urban/suburban/rural, proximity to old buildings and shooting ranges); 2) number, ages, and pregnancy status of family members; 3) egg consumption habits (daily/weekly); and 4) management of the birds (feed, housing, vaccination, or treatment history). The owners were encouraged to submit eggs and environmental samples (feed, water, and soil) for lead analysis in an attempt to identify sources of exposure. We also suggested checking blood lead concentrations of other birds in the flock. Blood lead in family members were also checked by consulting with their primary care providers in some cases.

In lead-positive flocks, if owners were interested in testing eggs, a pool of 3–4 eggs laid on different days was collected and submitted for analysis. The egg contents (yolk and albumen, without the shell) were homogenized and analyzed, assuming a person would consume both the whites and yolks. The concentration of lead in eggs was determined by graphite furnace-atomic absorption spectrometry (GF-AAS). The lead concentrations of the shells were also determined in some cases because shells are sometimes fed back to birds or used in composting where there is a potential for plants to absorb lead. Feed and soil samples were prepared and analyzed similarly. The concentration of lead in blood was also analyzed by GF-AAS. The method detection limit for all matrices was 0.005 $\mu\text{g/g}$, and the limit of quantification was 0.05 $\mu\text{g/g}$.

Data were entered into a spreadsheet (Excel, Microsoft, Redmond, WA). The percentage of cases in which lead was detected (the estimate of prevalence) was calculated: (no. of chickens with lead detected in the liver/total no. of chickens tested) $\times 100\%$. An estimate of branch-wise prevalence was calculated: (no. of positive cases in each laboratory)/(total no. of backyard submissions in each laboratory) $\times 100\%$. The percentage of cases in different categories based on their liver lead concentrations was calculated: (no. of cases in

each exposure category)/(total no. of lead-positive cases) $\times 100\%$. The total lead intake through egg consumption was estimated from the lead concentration in $\mu\text{g/g}$ in the eggs, the estimated weight of an egg (50 g average), and number of eggs consumed in 1 d. Data on lead concentrations in liver and in chicken eggs from the same premises were available for 14 flocks. Logistic regression was used to determine the relationship between lead concentrations in liver and the presence or absence of lead in eggs. Data were assessed for normality using the Shapiro–Wilk test for normality. For normally distributed data, linear regression was used to determine whether the quantity of lead in liver was associated with the quantity of lead in eggs. For non-normally distributed data, quantile regression was used. Statistical analyses were performed (Stata 14, StataCorp, College Station, TX).

Of the 1,476 chicken livers that were analyzed for lead, 45 were positive (3.04%). Lead concentrations were 0.9–41 $\mu\text{g/g}$ wet weight. Thirty-eight percent of the positive chickens had $< 2 \mu\text{g/g}$ of lead; 40% and 22% had significant and toxic exposures, respectively. Only 27 (67%) owners responded to follow-up calls and answered the set of predetermined questions. To see if there was a difference in lead exposure from different regions and environments, the locations of the positive flocks were categorized into urban, suburban, and rural areas based on the client's answer to the questionnaire or physical address provided on the submission form when follow-up was not possible. Most of the cases were from urban ($n = 18$) locations followed by suburban ($n = 11$) and rural ($n = 11$) areas. The percentage of cases at each of the branch laboratories was also tabulated. The Davis branch had the highest percentage of lead-positive cases ($n = 33$) followed in descending order by San Bernardino ($n = 8$), Tulare ($n = 2$), and Turlock ($n = 2$) branches (Table 1). Most of the flocks had < 10 birds; only 2 flocks had > 30 birds. Most owners indicated that the purpose of birds was as a source of eggs and as pets. The birds were on the premises for as little as 6 mo to as long as 8 y, with most of them for 2–3 y. The majority of the premises had no obvious source of lead exposure except for 8 households having an old building and 2 households having a shooting range nearby. The birds were kept in coops, allowed to free range in the yard, and given commercial feeds (organic in many cases) or table scraps. Egg production varied among flocks, and the production was 4–5 eggs per wk in flocks with < 10 birds. Eggs from smaller flocks were consumed primarily by family members or occasionally supplied to neighbors, whereas eggs from 2 larger flocks were supplied to community markets.

Most of the households had 4 members with 2 adults and 2 children. The consumption of eggs varied, with most of the families eating eggs 2–3 times per wk and 2 families with daily consumption. Owners of 14 flocks (30% of positive flocks) submitted a pool of 3–4 eggs collected over 7–10 d for testing. Lead concentrations varied 0.096–0.26 $\mu\text{g/g}$ of egg wet weight. Based on the detected egg lead concentrations

Table 1. California Animal Health and Food Safety branch-wise prevalence of lead-positive backyard chicken cases and corresponding range of detected lead concentrations.

Branch location	No of lead-positive birds	Total birds tested	Percentage (%)	Ranges of liver lead concentrations ($\mu\text{g/g}$)
Davis	33	815	4.05	1.8–27
Tulare	2	165	1.21	6.4, 41
Turlock	2	114	1.75	1.6, 1.6
San Bernardino	8	381	2.10	1.0–41

and average egg size of 50 g, the estimated lead content was 3–13 μg per egg pool. Logistic regression analysis suggested that the concentration of lead in the liver of a dead bird may be associated (odds ratio = 1.19; 95% confidence interval: 0.96–1.47) with the presence or absence of lead in eggs from the same premises, but the association was not statistically significant ($p = 0.12$). Neither lead concentrations in liver nor in eggs were normally distributed ($p < 0.01$). Quantile regression did not identify a significant association between quantity of lead in liver and quantity of lead in eggs from the same premises ($p = 0.32$).

Only 6 owners (14 %) of positive flocks submitted environmental samples. The concentration of lead in soils was 6–375 $\mu\text{g/g}$ wet weight, and 3 water samples tested negative for lead. In one case, a feed sample and paint chips were also tested, and they contained 0.18 and 14 $\mu\text{g/g}$ wet weight, respectively. Whole blood from 5 chickens belonging to this flock were also tested and found to have lead concentrations of 0.053–0.44 $\mu\text{g/dl}$.

Owners were asked if family members were tested to assess exposure following our recommendation upon receiving positive egg results. Three owners tested blood lead levels (BLL) of members in the family; all levels were below the blood lead action level of 0.24 $\mu\text{mol/L}$ (5 $\mu\text{g/dL}$). Two of these families consumed eggs 3 times per wk, and 1 family consumed eggs on a daily basis. Eighty-eight percent of the owners mentioned that they were aware of testing environmental samples or BLLs. However, financial constraints deterred the majority of them from pursuing extensive follow-up testing, and the rest were uninterested or thought that the concentrations were too low to pose a risk.

Many owners were aware of the postmortem services provided by CAHFS through their veterinarians and outreach publications of the California Department of Food and Agriculture. However, it is possible that owners who did not have knowledge of these services may not have submitted birds, thereby biasing the representation. Additionally, given that the initial samples were derived only from birds that were dead and submitted as a part of AI surveillance, the total percentage of lead-positive birds is only an estimate and not a true prevalence, given potential sample bias.

Clinical signs or postmortem findings of lead intoxication specific to chickens have not been described extensively in the literature or in 2 of the previously reported cases.^{1,14} The

lack of clinical signs or postmortem lesions is the result of the relative resistance of chickens to lead intoxication compared to other avian species.⁵ However, clinical signs have been described in waterfowl, and these include anorexia, lethargy, green diarrhea that stains the vent feathers, muscular weakness, crop impaction, and inability to fly or walk.⁸ Additionally, microscopic changes such as edema and degenerative changes in brain and peripheral nerves, widespread hemosiderosis of internal organs, acid-fast intranuclear inclusion bodies in kidney tubules, and necrosis of gizzard muscles have been noted.⁸ Because of the neurotoxicity of lead, similar clinical signs may be noted in chickens in cases of severe toxicosis. In our cases, even though 60% of the positive chickens had lead concentrations that were considered significant-to-toxic, the majority of the birds did not show any clinical signs or postmortem lesions suggestive of lead intoxication. Three birds had neurologic signs, such as head wobbling, incoordination, swollen crop, or inability to walk. Five lead-positive birds had various postmortem histologic lesions such as a dilated crop and proventriculus, biliary stasis, ulcerative necrotizing ingluvititis, diffuse severe microangiopathy in the cerebellum, and marked hemosiderosis in liver, kidney, and spleen. However, both the lead-positive and lead-negative birds had concurrent disease conditions such as Marek's disease and ovarian carcinomas, making it difficult to attribute postmortem findings solely to lead toxicosis. The lack of characteristic clinical signs or postmortem lesions presents a significant challenge for early detection and diagnosis in the absence of routine metal screening.

The main source of lead exposure for backyard poultry is likely to be contaminated soil. Lead-contaminated soils are common, especially in urban areas with older buildings with flaking lead-based paint and proximity to roadways.¹⁵ A closer analysis of the physical location of cases showed that most of the lead-positive submissions to the Davis branch laboratory originated from San Francisco/Oakland urban areas, and the owners reported the presence of old structures on the premises. However, noticeable peeling of paint and consumption by birds were not reported in most cases. Three owners located in rural areas mentioned the presence of shooting ranges nearby. Interestingly, only one bird had a lead pellet recovered from the gizzard during autopsy. A source of lead exposure in other positive cases was not determined.

There is a potential health risk for people consuming eggs from lead-exposed chickens. The U.S. Food and Drug Administration recommendations for the provisional tolerable daily intake (TDI) for lead from all sources in children <6 y old, pregnant women, and adults is 6, 25, and 75 μg , respectively.⁴ At the highest estimate of lead concentration measured (13 $\mu\text{g}/\text{g}$), consumption of 1 egg per day would double the recommended TDI in children <6 y old. Although most families consumed eggs on an average of 3 times per wk, frequent exposure to such high concentrations may lead to adverse health effects in children, such as behavioral disorders, attention-deficit/hyperactivity disorder, decreased brain volume, and IQ deficits.^{6,9} In fact, no threshold for adverse effects has been clearly identified for lead.⁹ Therefore, owners of backyard chickens must be aware of the risk of repeated consumption of lead-contaminated eggs even at concentrations that they perceive to be insignificant.

It is important to point out that backyard chickens serve as a biomonitor of environmental lead contamination. If chickens are found to be positive for lead, there is a risk that children are exposed to the primary source of lead contamination (e.g., soils) in addition to lead-contaminated eggs, given that they tend to play in the soils and spend more time outdoors for activities.

Interestingly, there exists great variability in lead exposure even when the birds are in the same environment. In one of the flocks in which BLLs were determined for 5 birds, the values varied from <0.0024 $\mu\text{mol}/\text{L}$ (0.05 $\mu\text{g}/\text{dL}$) to as high as 0.021 $\mu\text{mol}/\text{L}$ (0.44 $\mu\text{g}/\text{dL}$) with the latter concentration consistent with toxicosis (>0.017 $\mu\text{mol}/\text{L}$ [0.35 $\mu\text{g}/\text{dL}$]).⁵ This variability may be attributed to the difference in foraging and pecking behavior of individual birds. Even in a single bird, BLLs tend to vary between days and do not follow a specific trend, posing an important limitation in predicting the pattern.¹⁴ We assume that this fluctuation is because of the deposition in storage tissues such as bone and subsequent release following calcium mobilization during egg shell formation. A positive correlation between whole blood and egg yolk lead concentrations has been reported¹⁴; egg yolk lead concentrations were measured on 1, 5, and 9 days post-lead exposure, and yolk lead concentrations tended to increase. However, no correlation between liver and blood lead concentrations has been determined for chickens. Given that the owners could not identify the individual chickens that laid the eggs, it was not possible to determine a relationship in positive birds. Although there seemed to be an association between lead in the liver of birds and eggs collected from the same premises, the association was not significant, probably because of the small sample size. In general, eggs with higher lead concentrations were submitted from flocks that had dead birds with significant-to-toxic lead exposures.

One of the main limitations of our study is that the number of backyard flocks tested for lead in blood and eggs was

limited because of financial constraints of the owners. Additionally, there was a lack of information about the BLLs of individuals that consumed eggs from flocks with high lead concentrations, further limiting the possibility of extrapolating the information. Even in 1 of the 3 families in which BLLs were obtained, the low value did not truly reflect their frequent egg consumption, probably because of the varied lead concentrations in eggs. Although similar follow-up information was collected from all owners, we assume that there may have been biased responses.

Lead has been detected in backyard chickens in both rural and urban areas, and exposure to the public is a potential health concern. The lack of obvious clinical signs in chickens suggestive of lead exposure or toxicosis makes early detection and diagnosis difficult. Therefore, a safer alternative is to raise awareness of chicken owners, veterinarians, and public health agencies of the problem and likely sources of exposure. In higher risk environments (e.g., older urban homes), testing of environmental samples prior to bird placement is recommended and post-placement monitoring of birds and eggs should be considered.

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