Lead exposure to children from consumption of backyard chicken eggs

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Abstract

Backyard chicken ownership is rapidly increasing in urban areas in the United States, largely as a way to provide eggs for household consumption. Despite elevated levels of environmental lead contamination in many US cities, the role of backyard chicken eggs as a pathway for lead exposure, particularly for children, has received limited scrutiny. To characterize lead exposure from consumption of backyard chicken eggs for children and predict related effects on blood lead level (BLL), we conducted a cross-sectional study of backyard chicken owners in the Greater Boston area (n=51). We interviewed participants regarding egg consumption by household members and collected backyard eggs (n=201) and coop soil samples (n=48) for analysis. Inductively coupled plasma mass spectrometry (ICP-MS) was used to evaluate lead concentration in homogenized eggs and an X-ray fluorescence (XRF) portable device was used to assess soil lead levels in the laboratory. We used the USEPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) to assess the relative contribution of backyard egg consumption to aggregate BLL in children. Four scenarios were developed in the IEUBK model to address variability in egg consumption rates and egg lead contamination. Lead was detected in egg samples from 98% of the households that provided egg samples. Mean household lead concentration was 0.10 ug/g (SD: 0.18). Egg lead concentrations ranged from below the limit of detection (0.0014ug/g) to 1.798 ug/g (<1.4–1198 ppb). Egg lead levels were strongly positively correlated with lead concentration in coop soil (r=0.64; p<0.001). In modeled scenarios where a child <7 years frequently ate eggs highly contaminated with lead, BLLs are predicted to increase by 0.9–1.5 ug/dL. In three other scenarios reflecting more moderate egg lead contamination and consumption rates, BLLs were predicted to increase from 0.1 to 0.8 ug/dL. Consumption of

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backyard chicken eggs can contribute to lead exposure in children. Soil lead remediation prior to chicken ownership may reduce lead exposure from backyard eggs.

Keywords

Lead; eggs; urban health; poultry; chickens; child health

1. Introduction

Backyard chicken (BYC) ownership is increasingly popular in the United States, especially in urban areas.[1–4] A 2010 study of backyard chicken ownership in Denver, Los Angeles, Miami and New York City reported that approximately 1% of all households in these cities owned chickens (ranging from 0.2% in New York City to 1.7% in Miami), and 4% of households without chickens intended to own them within the next 5 years.[5] A central motivation for backyard chicken ownership is consumption of chicken eggs, which flock owners often believe to be more nutritious, safer and tastier than their commercially available counterparts.[5,6] While a body of research highlights risks of zoonotic infections from contact with live poultry, the study of chicken egg consumption as a pathway for human exposure to environmental contaminants has received comparatively less attention.[7–12] In particular, the increase in poultry ownership in urban areas with heightened environmental lead contamination compels an investigation into backyard egg consumption as a source of lead exposure, particularly for children.

Environmental lead contamination is ubiquitous in the United States due to the historical use of leaded gasoline and paint.[13] A well-established literature documents the association between exposure to lead contaminated paint, dust, and soil and elevated blood lead levels in children.[14–18] Urban gardening is a documented source of lead exposure, both from accidental ingestion of leaded soil and from uptake or adherence of leaded soil on produce. Environmental lead exposure is associated with neurological impairment in children, including reduced IQ, as well as attention-related behavior problems and reduced academic achievement, at increasingly low levels of exposure.[19–23] According to the US Centers for Disease and Prevention, there is no safe blood lead level for children, indicating that any source of exposure poses health risks.[24]

In addition to consumption of contaminated eggs, human caretakers of chickens may be exposed to lead in soil from incidental ingestion of soil following bird handling or outdoor activity in the yard. Poultry owners may also inhale lead contaminated dust while caretaking or playing with birds, and may track contaminated soil into the home. In poultry, lead is preferentially deposited in the eggshell and egg yolk over the albumin, and lead is excreted in the feces.[25] In the context of households with BYC, composted lead-containing chicken feces and egg shells may be used to fertilize vegetable gardens, with the potential for lead to adhere or be taken up by homegrown produce, although lead contribution from this pathway is likely small for children.[26–28] Pathways of lead exposure for chickens living in backyards are unclear, but likely include behavioral and dietary factors. Chickens are exposed to lead in contaminated soil through poultry behaviors, such as foraging, roosting, preening, fighting,

and dust bathing. Chickens may also be exposed to lead through their diet; lead bio-accumulates in some chicken feeds and supplements, including worms and invertebrates and bone meal and oyster shells.[29–31] While the full depiction of the chicken-human lead pathway in the context of backyard chicken production is beyond the scope of this paper, we note that egg consumption is likely one of multiple pathways of potential lead exposure associated with backyard poultry husbandry.

A limited number of studies have documented detectable levels of lead in backyard eggs to date, including lead concentrations higher than detected in commercially-available eggs. [25,32–37] Backyard egg lead concentrations in studies in the US, Belgium and Australia ranged from as low as 0.003 ug/g in Belgium to as high as 0.97 ug/g in California. Rubio et al. documented higher levels of toxic metals in home-grown hen eggs compared to free range eggs in Spain, although noted lower concentrations of lead compared to other toxic metals.[38] Lead was also detected in backyard eggs in an industrially polluted region in Italy and in low levels in egg products in Turkey.[39,40] To our knowledge, only one prior study, focused on New York City's community garden coops, has evaluated change in BLL from backyard chicken egg consumption.[32] The New York City study observed increases in BLL in children as a result of egg consumption, but used data derived from national studies on commercial egg consumption rather than those specific to household producers, the latter of which we hypothesized to be higher.

To evaluate lead contamination in backyard chicken eggs and identify associated changes to BLL resultant from backyard egg consumption for children, we conducted a study of backyard poultry owners in Greater Boston, Massachusetts. We used established methods for lead characterization and exposure assessment, and determined whether and under which scenarios consumption of backyard eggs may increase children's BLL. We also assessed whether egg lead was correlated with soil lead in the backyard environment to identify possible sources of remediation.

2. Materials and Methods

2.1. Study design

We recruited households who keep chickens in their backyards in the Greater Boston area of Massachusetts in the CLUC ("Chickens Living in Urban Coops") Study from 2016–2017. A cross-sectional, convenience sampling design was used. Participants were recruited by word of mouth and flyers and emails distributed through a local backyard poultry organization and posted at feed stores and urban agriculture events. Potential participants were eligible if they were older than age 18 years and housed at least one chicken on their property.

Enrollment activities were conducted at the participant's home by a trained team. Study participation consisted of a 45-minute interview including the following topics: poultry husbandry and biosecurity practices, poultry contact by each family member, egg consumption practices, and household demographics. To quantify exposure to lead from consumption of backyard chicken eggs for this study, questionnaire respondents answered the following questions regarding members of their household: "Do you and/or your household members consume eggs from your flock? If yes, on average how many eggs/week

does each toddler (<3 years) or young child (3–7 years) consume?" Participants also provided at least one egg sample, and were offered the option to provide more samples at their discretion. Whole eggs (in shell) were refridgerated within two hours of collection and stored until laboratory analysis. Up to three soil samples from the chicken coop were collected from a depth of 1–3 inches by a member of the study team and combined into a Ziploc bag. Samples were airdried, sealed, and stored in ambient conditions until analysis.

All study materials were labeled with a unique study ID and identifying data (name, address) were stripped from the dataset for analysis. Results regarding egg lead concentrations were reported back to participating households in writing accompanied by a document entitled "Understanding Your Test Results: Lead in Soil and Chicken Eggs" which was developed by the Healthy Soils, Healthy Communities project led by Cornell University, New York State Department of Health and partners ([http://cwmi.css.cornell.edu/ UnderstandingTestResultsLeadSoilsEggs.pdf](http://cwmi.css.cornell.edu/UnderstandingTestResultsLeadSoilsEggs.pdf)). Participants provided informed consent prior to joining the study. All aspects of study design and conduct were approved by the Social, Behavioral & Educational Research Institutional Review Board at Tufts University.

2.2. Laboratory analysis

Backyard chicken egg samples were analyzed for lead concentrations using Inductively Coupled Plasma Mass Spectrometry (ICP-MS) at the ICP-MS & Laser Ablation Lab at the Boston University Department of Earth & Environment. Samples were prepared by digesting ~0.4 g of homogenized egg (egg yolk and egg white) in 5 mL of concentrated HNO_3 in closed Teflon vials at 70 °C for 24 hours. Following digestion, a 0.1 mL aliquot was removed and diluted to a total volume of 5 mL using high-purity water. This procedure resulted in a total dilution factor of the egg of between ~650 to 700 times. The resulting solutions were analyzed for lead concentrations on a VG Plasma Quad ExCell ICP-MS, using a Meinhard-C concentric nebulizer at a flow rate of ~1 mL/min. Instrumental drift was monitored and corrected for by analyzing a 1 ng/g Pb standard at various times throughout the run (every 5 analysis items). A calibration curve was generated by analyzing lead standards of varying concentrations (from 0.01 ng/g to 50 ng/g) interspersed throughout the analytical run, and this curve had an r2 value of 0.999. To assess the reproducibility of this procedure, several samples were digested in triplicate and concentrations agreed within 5%. Method blanks were run at the beginning and end of each analytical session and were subtracted from the signal. Concentrations in the original egg were calculated by multiplying the concentration in the solution analyzed by the dilution factor of the homogenized egg.

Up to three soil samples were collected from the coop at a depth of 1–3 inches. These samples were aggregated and air-dried prior to storage and analysis. Soil samples were analyzed for lead using the Innov-X Alpha Series portable handheld X-Ray fluorescence analyzer (XRF) (Olympus NDT, Waltham MA) in a stationary stand at the Boston University School of Public Health Exposure Biology Laboratory. Each soil sample was analyzed in triplicate. In between reads, the sample was mixed within the same bag. The results of the three reads were averaged.

2.3. Data analysis

2.3.1 Statistical analysis.—Descriptive analyses, including summary statistics and graphics, were conducted on egg lead concentration values across the sample and within each household. Egg and coop soil lead concentrations were averaged across the samples provided by each household to calculate a mean household egg lead concentration variable and a mean household soil concentration variable. Egg samples that were below the limit of detection, were replaced with 0.0007 (the detection limit of 0.0014ug/g divided by 2). Soil sample concentrations below the limit of detection of our XRF device (10–15 ug/g) were replaced with 10 ug/g.

Pearson's correlation analysis for pairwise correlations was conducted to assess correlation between household mean soil and egg lead concentrations and between eggs within a given household. Soil and egg lead concentrations were log transformed prior to correlation analysis to maintain normality assumptions. All analyses were conducted in Stata 13.1 (College Station, TX).

2.3.2 IEUBK model application.—We used the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) to model changes to BLL associated with backyard egg consumption.[41] The USEPA developed the Integrated Exposure and Uptake Biokinetic (IEUBK) model to approximate quasi-steady-state blood lead concentrations associated with lead exposures occurring at least once every seven days for a duration of three or more months for children under age 7.[42] The IEUBK model can assess changes in predicted BLL from exposures to lead in soil/dust, diet, air, maternal, and alternate sources.[43] All exposure scenarios were assessed in the IEUBK model (IEUBKwin32 Lead Model Version 1.1 Build 11) using varied exposure parameter inputs.

To assess changes in predicted blood lead level (BLL) from backyard egg consumption for children younger than 7 years of age, the alternate source data input of the IEUBK model was used. This input requires exposure to be entered as a daily lead intake (ug/day). Daily lead intake from backyard egg consumption were calculated for children under age three and for children age three to younger than seven years using a modification of the average daily dose equation[44].

Daily lead intake from backyard egg consumption for four plausible scenarios were calculated for children under age 3 and for children age 3 to less than 7 using the following equation:

$$\text{Alternate lead intake (ug/day)} = \frac{EPC * IR * EF}{AT}$$

where:

EPC= exposure point concentration (ugPb/g of egg)

IR= ingestion rate (eggs/week * 49.6 g/egg)

EF= exposure frequency (weeks)

AT= averaging time (days)

The IEUBK requires an absorption fraction as an input if an alternate source is entered. An absorption fraction of lead from eggs does not exist in the literature; however, the absorption fraction for diet has been estimated to be 40–50% for children[13]. The more conservative estimate of 50% was used in this assessment. All other parameters in the IEUBK model remained at the default values, and the default maternal BLL was updated per the USEPA June 2017 guidance.[45]

In 2017, the default maternal BLL was reduced from 1 ug/dL to 0.6 ug/dL to reflect the most recent years of NHANES lead surveillance data (2009–2014). This updated estimate reflects a trend of maternal BLL decreasing over time in U.S. women aged 17–45.[45]

Four plausible exposure scenarios for the consumption of backyard chicken eggs were considered in each model for two age groups: toddler (age <3 years) and young child (age 3–7 years). The scenarios range from the most likely exposure (Scenario #1) to the highest exposure (Scenario #4) using data from our surveys of household members. Parameters for each scenario are presented in Table 2. Scenario #1 reflects children consuming backyard chicken eggs with mean lead contamination at the mean consumption rate. Scenario #2 represents children consuming backyard chicken eggs with mean lead contamination at the 95th percentile consumption rate. Scenario #3 represents children consuming backyard chicken eggs contaminated with lead at the 95th percentile contamination level at the mean consumption rate. Scenario #4 represents children consuming backyard chicken eggs contaminated with lead at the 95th percentile contamination level at the 95th percentile consumption rate.

When the IEUBK model is run with all the parameters set to the default values, without inclusion of backyard eggs, the "baseline scenario" is reflective of a diet for a child in the US estimated from an analysis of the FDA Total Diet Study 1995–2003, which is assumed to be inclusive of a diet that includes store-bought eggs.[46] To capture the incremental change in blood lead concentrations from consumption backyard chicken eggs, we calculated the differences between the baseline scenario and our four constructed scenarios. We used egg consumption rates from our sample to calculate the daily dose parameter entered into the IEUBK. Lead exposure attributable to BYC egg consumption was quantified as the change in blood lead due to backyard egg consumption. This rate of egg production was assumed in our models to extend for 37 weeks of the year. In the New England region, backyard chickens typically lay eggs nine months per year (March to November) with production decreases associated with reduced daylight during the fall and winter months.

3. Results

3.1 Participants and demographics.

Fifty-one households participated in the study, with 50 households completed the questionnaire and 48 households provided egg samples, ranging from 1–12 eggs (total of 201 eggs collected). Forty-eight households provided coop soil samples. Summary statistics regarding backyard chicken egg consumption and lead concentrations in eggs and coop soil

are presented in Table 1. Mean household size was 3.8 persons. Approximately 40% (n=19) of households had children younger than seven years old in the home, of which the majority (n=17) had children younger than 3 years (89% of households with children and 35% of total households had children younger than 3 years). Households kept chickens on their properties from two months to nine years. Households owned a mean of six chickens at the time of the study, and chickens collectively produced on average 26 eggs per week per household (range 8–68). In our modeling, we assumed this rate of production continued over a 37 week laying season given the seasonal factors noted above.

3.2 Egg consumption.

Ninety-four percent (n=47) of households reported eating eggs produced by chickens in their backyards (Table 1). Toddlers (6 months-3 years) consumed approximately 2 eggs per week (mean=2.0; sd: 1.5, range 0–5). Children ages 3–7 years ate approximately 2 eggs/week (mean=2.1; sd: 1.5; range 0–5). The one household with an infant (<6 months) reported no egg consumption by this child. Children older than 7 years ate approximately four eggs/ week (mean=3.8; sd: 2.4, range 0–8; data not included in our models).

3.3 Egg and soil lead analysis.

Mean household egg lead concentration was 0.10 ug/g (SD: 0.18), with values ranging from <LOD to 1.1 ug/g (Table 1). Median lead concentrations were 0.022 ug/g (IQR: 0.01, 0.12). Mean and median household soil lead concentration were 238.4 ug/g (SD: 277.5) and 147.7 ug/g (IQR: 51.8, 282.7) respectfully. Raw data on household level demographics and lead concentrations are provided in the available supplemental file.

3.4 Correlation analysis.

Mean egg lead concentration was strongly positively correlated with mean lead concentration in coop soil on the household level (r=0.64; p=0.000). Positive correlation was observed between lead concentrations of pairs of eggs within the same household, although with varying strength (min r=0.25, max r=0.97 across households).

3.5 IEUBK Model Results

Table 3 provides IEUBK model predictions of BLLs and incremental changes in BLL due to backyard chicken egg consumption for two modeled scenarios (Scenarios 1 and 4). In Scenarios 1–3, the IEUBK model predicted an increase in BLL ranging from 0.1 to 0.8 ug/dL. Greatest incremental change in BLL was observed for infants from 6 months to1 year. In Scenario 4, changes in BLL predicted from the IUEBK were higher, ranging from 0.9 ug/dL for children 6–7 years old to 1.5 ug/dL for infants from 6 months-1 year. For children ages 1–6, change in BLL ranged from 1.0 to 1.1 ug/dL under the maximum exposure scenario (Table 3). An increase in the relative contribution of backyard eggs to BLL was observed among 3–4 year olds in Scenarios 2–4. Figure 1 depicts predicted changes in BLL for children <7 under the four scenarios by age group.

4. Discussion

We observed detectable lead concentrations in backyard chicken eggs collected in the Greater Boston area. Our IEUBK modeled scenarios predicted increases in children's BLL associated with backyard egg consumption ranging from 0.1to 1.5 ug/dL. Our maximum consumption scenario indicates that consuming backyard chicken eggs may increase BLL by as much as 50% for infants <1 year. In our maximum exposure scenario, all children younger than 6 years had increases in BLL resultant from backyard egg consumption 1 ug/dL. Our findings indicate that chicken eggs may be a source of lead exposure children whose families raise backyard chickens and consume their eggs.

There is currently considerable interest in establishing the dose-response relationship between particular exposures, including food, water, soil and dust, and blood lead levels in children as a tool for public health interventions.[47] In a recent analysis by Zartarian et al., food intake was a major contributor to blood lead for children below the 70th percentile for BLL, contributing approximately 0.6 ug/dL to overall BLLs in this age group. Our models indicate that for children who consume backyard eggs, these eggs may account for a significant proportion of overall BLL from food. For example, in our mean backyard egg exposure scenario (Scenario 1), backyard eggs would account for half to two-thirds of BLL from food sources in children younger than 6, and more than 80% of BLL from food sources in children 6–7 years old. Our predicted lead exposures from backyard egg consumption highlight the need to more fully characterize children's exposure to lead from their diet.

The highest exposure scenario (Scenario 4), fortunately, did not exist in our study, as we did not observe any household with this combination of exposure assumptions; however, our sample is relatively small, and it is not unreasonable to assume that this scenario might reflect the exposure of children in other households. Twelve percent of households in our study provided eggs with lead concentrations at or exceeding the highest egg lead contamination level included in our model (0.324 ug/g). Similarly, study participants reported toddlers and young children in their households eating as many as five eggs/week, a level higher than our maximum consumption estimate for our model. As a result, a scenario in which backyard chicken eggs pose an important contribution to overall BLL appears plausible.

Maximum egg lead concentrations identified in this study are higher than those reported in similar studies by nearly two-fold (Table 4), and are also higher than egg lead concentrations reported in other studies in India and Europe.[38–40,48,49] Of note, our maximum egg lead concentration was more than six times the maximum level obtained in a study in New York City of eggs harvested from community gardens (0.167 ug/g).[32] Our study participants produced eggs in their backyards, in close proximity to older-stock housing, while the New York City eggs were produced in a community garden environment, at perhaps greater distance from buildings. This observation again may indicate the importance of soil lead contamination from housing stock. Our findings are also notably higher than the egg lead concentrations established in the US Food and Drug Administration's analysis of a small number of market eggs from 2006–2013 (mean lead concentration 0.0004 ug/g; maximum 0.014 ug/g).[50] Industrial hens are typically raised in confined settings without access to

forage or soil, which may further indicate urban environmental pollution as a potential source for the elevated levels observed in our study of backyard producers.

Our findings indicate that backyard soil lead contamination is strongly correlated with egg lead concentration. This observation concurs with those of prior studies in New York, Belgium and Australia that also identified positive correlation between backyard soil lead and egg lead.[32,34,51] Soil remediation to reduce lead contamination in urban gardens is a well-established intervention to reduce lead exposure from homegrown produce.[26] Our results suggest that such efforts should also include remediation for chicken coops with higher soil lead concentrations. Since the toxicokinetics of lead in chickens are not well characterized, it is unknown whether remediation efforts after introduction of a flock would reduce egg lead concentrations. As a result, soil remediation efforts are likely most effective in advance of flock introduction. However, a study of market eggs in India indicated that egg lead concentrations exceeded soil lead concentrations, suggesting that additional contributing factors beyond soil lead contamination may be involved in egg lead concentration.[48] A second study in India identified strong correlation between lead in chicken feed and market eggs, indicating that feed contamination and feeding patterns are possible sources of contamination as well.[49] While soil is likely an important lead source in the chicken coop environment, other lead exposure pathways are worthy of investigation.

Overall, our study and comparable research suggests important regional differences in lead contamination in backyard chicken eggs. This may be due to differences in soil lead contamination, other environmental lead contamination, chicken feeding practices, lead bioavailability, or other regional differences. These are important areas for future research so as to target interventions in regions mostly likely affected. We highlight a study in Belgium observed elevated egg lead levels in conjunction with lower soil lead levels.[35] This finding differs from ours, and from the New York study, both of which identified correlated egg and soil lead concentrations. As well as potential differences in chicken feeding or behavioral practices that affect lead exposure, this finding may indicate important differences in regional bioavailability of lead in soil. Soil composition, which varies regionally, is known to affect lead bioavailability, as is pollution source.[52–55] In Boston and New York City, primary sources of urban lead pollution include degrading building materials and renovations and historical gasoline emissions, while lead contamination in Belgium results primarily from industrial air pollution. The distinct sources of lead pollution, in addition to regional differences in soil composition, may account for the regional differences in soil and egg lead correlations.

We also observed significant variation in lead concentration in the eggs analyzed in our study. This is possibly because our sample size was larger than comparable studies and/or there is important within-flock variation in lead concentration. While our correlation analysis indicated positive within-household correlation between eggs collected from the same household, the strength of these correlations varied. This finding indicates that egg lead concentrations may also be sensitive to micro-environmental differences within the backyard environment (such as higher soil contamination in some areas of the property compared to others), to poultry genetics (e.g. species-level differences in absorption) and to poultry behavioral factors (pecking the ground, frequency of wandering outside the coop, or

preferred dust-bathing locations). These drivers of variability in egg lead concentration within the household are worthy of future analysis.

Spliethoff and colleagues used the IEUBK model in conjunction with egg lead data to evaluate dietary contribution of backyard eggs to children's BLLs from consumption of eggs from New York City community gardens. They observed predicted increases in BLLs based on their data of 0.0–2.4 ug/dL, depending on egg lead levels and consumption rates. Their most typical scenario (egg lead <100 ug/g and consumption of 1 egg/day) resulted in a predicted BLL increase of 0.0–0.3 ug/dL. These BLL estimates differed from ours possibly due to increased egg consumption reported by our study participants, who consumed all or most of the eggs from their flock, unlike members of a shared community coop, who may receive fewer eggs and supplement with commercial eggs.

A growing literature documents the negative cognitive effects associated with BLLs >10ug/dL in children and low-level exposure.[19–21,56] In 2012, the US Centers for Disease Control and Prevention lowered its reference level in children to 5ug/dL from 10ug/dL, indicating acknowledgement of harm caused by low levels of exposure.[57] However, neurological impairment at BLL below 5ug/dL have been documented in studies of children in the literature. Jusko and colleagues established reductions in IQ at lifetime average BLL as low as 2.1 ug/dL in non-linear models.[58] Lanphear noted a reduction of 3.9 points associated with an increase in lifetime average BLL from 2.3–10ug/dL.[19] These findings support the evidence that even small increases in BLLs predicted in our study may contribute to deleterious neurological outcomes for children. An important next step of this research is to identify BLLs in children who consume eggs to identify the targeted health impact on this group of children.

4.1 Limitations and generalizability.

The primary limitation of our study was small sample size, in terms of overall number of households and number of households with young children. While our household demographics reflect general trends in backyard poultry ownership, our participants were volunteers, and may be more likely to be aware or concerned of lead in their homes. Given these limitations, generalizations of our findings should be made with caution. Additionally, several assumptions were made in the IEUBK model to predict childhood exposure to lead from backyard chicken egg consumption in children. Using the model parameters, we assumed children were eating backyard chicken eggs over the duration of the years modeled (ages 0.5–7) rather than assessing annual exposure (e.g. exposed at age 2–3 but not at other ages). These assumptions may overestimate exposure. We addressed uncertainties in consumption and contamination by using multiple scenarios in our analysis, however some uncertainly may remain in our estimates. In our analysis, we assumed children eat the whole egg, which may not be accurate in all cases. Lead may partition into egg yolks at higher concentrations than egg whites, and as a result, exposure may be lower if only egg whites are being eaten.[25] Consumption data by age group from a larger sample would improve exposure estimates. In terms of study design, the study was cross-sectional and we did not have BLL for children in participant households, and relied on models for our estimates of BLL. Additionally, the Greater Boston area is known for elevated environmental lead

contamination, so our findings may reflect higher levels of soil lead contamination than may be seen in other areas.

Scenarios 1 and 4 were constructed to estimate children's BLL outcomes at a central tendency (the mean) and at a reasonable maximum lead exposure values (the 95th percentile). However, actual exposures are more nuanced. Of note, it is possible that baseline BLLs and egg lead exposure are correlated. Children exposed to elevated egg lead levels may also be exposed to higher levels of other sources of environmental lead exposure in and around their home. In the context of high lead exposure from other sources (e.g. high soil or water contamination), backyard egg consumption may be a less significant contributor to a child's overall BLL compared to a child with low levels of baseline lead exposure. Future studies should take these factors into account, ideally in the context of probabilistic modeling that quantitatively accounts for correlations.

5. Conclusions

Lead contamination of backyard chicken eggs was nearly universal in our study of backyard chicken owners in the Boston area. Egg lead concentration was highly correlated with backyard soil lead levels from the coop, indicating environmental contamination. Consumption of backyard eggs contributed to increases in predicted BLLs for children younger than 7 years ranging from 0.1–1.5 ug/dL. We observed the greatest predicted relative contribution of backyard eggs to BLL among young children <1 year old. In light of the growing popularity of backyard chicken ownership, this understudied pathway of lead exposure warrants further scrutiny.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

REFERENCES

1. Behravesh CB, Brinson D, Hopkins BA, Gomez TM. Backyard Poultry Flocks and Salmonellosis: A Recurring, Yet Preventable Public Health Challenge. Clin Infect Dis 2014;58:1432–8. 10.1093/cid/ciu067. [PubMed: 24501387]

2. USCDC. Keeping Backyard Poultry. 2017 URL: https://www.cdc.gov/features/salmonellapoultry/index.html (Accessed 28 February 2018).

3. Stinson S, Mete A. Popular Backyard Flock program reduces biosecurity risks of amateur production. Calif Agric 2013;67:203–9. 10.3733/ca.v067n04p203.

4. Tobin MR, Goldshear JL, Price LB, Graham JP, Leibler JH. A Framework to Reduce Infectious Disease Risk from Urban Poultry in the United States. Public Health Rep 2015;130:380–91. 10.1177/003335491513000417. [PubMed: 26346104]

5. USDA. Poultry 2010. Urban Chicken Ownership in Four U.S. Cities USDA–APHIS–VS, CEAH. Fort Collins, CO #661.0413; 2012.

6. Elkhoraibi C, Blatchford RA, Pitesky ME, Mench JA. Backyard chickens in the United States: A survey of flock owners. Poult Sci 2014;93:2920–31. 10.3382/ps.2014-04154. [PubMed: 25193256]

7. Loharikar A, Briere E, Schwensohn C, Weninger S, Wagendorf J, Scheftel J, et al. Four multistate outbreaks of human Salmonella infections associated with live poultry contact, United States, 2009. Zoonoses Public Health 2012;59:347–54. 10.1111/j.1863-2378.2012.01461.x. [PubMed: 22372941]

8. Hermans D, Pasmans F, Messens W, Martel A, Van Immerseel F, Rasschaert G, et al. Poultry as a Host for the Zoonotic Pathogen Campylobacter jejuni. Vector-Borne Zoonotic Dis 2011;12:89–98. 10.1089/vbz.2011.0676. [PubMed: 22133236]

9. Behravesh CB, Brinson D, Hopkins BA, Gomez TM. Backyard poultry flocks and salmonellosis: a recurring, yet preventable public health challenge. Clin Infect Dis Off Publ Infect Dis Soc Am 2014;58:1432–8. 10.1093/cid/ciu067.

10. US CDC. Multistate Outbreaks of Human Salmonella Infections Linked to Live Poultry in Backyard Flocks, 2017 (Final Update). 2017.

11. Capua I, Marangon S. Control of avian influenza in poultry. Emerg Infect Dis 2006;12:1319–24. [PubMed: 17073078]

12. Leibler JH, Dalton K, Pekosz A, Gray GC, Silbergeld EK. Epizootics in Industrial Livestock Production: Preventable Gaps in Biosecurity and Biocontainment. Zoonoses Public Health 2016 10.1111/zph.12292.

13. ATSDR. TOXICOLOGICAL PROFILE FOR LEAD. 2007.

14. Lanphear BP, Roghmann KJ. Pathways of Lead Exposure in Urban Children. Environ Res 1997;74:67–73. 10.1006/enrs.1997.3726. [PubMed: 9339217]

15. Lanphear BP, Matte TD, Rogers J, Clickner RP, Dietz B, Bornschein RL, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiologic studies. Environ Res 1998;79:51–68. [PubMed: 9756680]

16. Jacobs DE, Clickner RP, Zhou JY, Viet SM, Marker DA, Rogers JW, et al. The prevalence of lead-based paint hazards in U.S. housing. Environ Health Perspect 2002;110:A599–606. [PubMed: 12361941]

17. Hanna-Attisha M, LaChance J, Sadler RC, Champney Schnepp A. Elevated blood lead levels in children associated with the Flint drinking water crisis: a spatial analysis of risk and public health response. Am J Public Health 2016;106:283–90. [PubMed: 26691115]

18. Silbergeld EK. Preventing lead poisoning in children. Annu Rev Public Health 1997;18:187–210. 10.1146/annurev.publhealth.18.1.187. [PubMed: 9143717]

19. Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ Health Perspect 2005;113:894–9. 10.1289/EHP.7688. [PubMed: 16002379]

20. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. A meta-analysis of modern studies. JAMA 1990;263:673–8. [PubMed: 2136923]

21. Bellinger DC. Very low lead exposures and children's neurodevelopment. Curr Opin Pediatr 2008;20:172–7. [PubMed: 18332714]

22. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain 2003;126:5–19. [PubMed: 12477693]

23. Grandjean P, Landrigan PJ. Neurobehavioural effects of developmental toxicity. Lancet Neurol 2014;13:330–8. [PubMed: 24556010]

24. United States Centers for Disease Control and Prevention. Lead - New Blood Lead Level Information. 2012.

25. Trampel DW, Imerman PM, Carson TL, Kinker JA, Ensley SM. Lead Contamination of Chicken Eggs and Tissues from a Small Farm Flock. J Vet Diagn Invest 2003;15:418–22. 10.1177/104063870301500503. [PubMed: 14535540]

26. Clark HF, Hausladen DM, Brabander DJ. Urban gardens: lead exposure, recontamination mechanisms, and implications for remediation design. Environ Res 2008;107:312–9. 10.1016/j.envres.2008.03.003. [PubMed: 18456252]

27. Latimer JC, Van Halen D, Speer J, Krull S, Weaver P, Pettit J, et al. Soil Lead Testing at a High Spatial Resolution in an Urban Community Garden: A Case Study in Relic Lead in Terre Haute, Indiana. J Environ Health 2016;79:28–35.

28. Mitchell RG, Spliethoff HM, Ribaudo LN, Lopp DM, Shayler HA, Marquez-Bravo LG, et al. Lead (Pb) and other metals in New York City community garden soils: factors influencing contaminant distributions. Environ Pollut Barking Essex 1987 2014;187:162–9. 10.1016/j.envpol.2014.01.007.

29. Scelfo GM, Flegal AR. Lead in calcium supplements. Environ Health Perspect 2000;108:309–13. [PubMed: 10753088]

30. Morgan JE, Morgan AJ. Earthworms as biological monitors of cadmium, copper, lead and zinc in metalliferous soils. Environ Pollut Barking Essex 1987 1988;54:123–38.

31. Darling CTR, Thomas VG. Lead bioaccumulation in earthworms, Lumbricus terrestris, from exposure to lead compounds of differing solubility. Sci Total Environ 2005;346:70–80. 10.1016/j.scitotenv.2004.11.011. [PubMed: 15993683]

32. Spliethoff HM, Mitchell RG, Ribaudo LN, Taylor O, Shayler HA, Greene V, et al. Lead in New York City community garden chicken eggs: influential factors and health implications. Environ Geochem Health 2014;36:633–49. 10.1007/s10653-013-9586-z. [PubMed: 24287691]

33. Bautista AC, Puschner B, Poppenga RH. Lead Exposure from Backyard Chicken Eggs: A Public Health Risk? J Med Toxicol 2014;10:311–5. 10.1007/s13181-014-0409-0. [PubMed: 24943230]

34. Waegeneers N, De Steur H, De Temmerman L, Van Steenwinkel S, Gellynck X, Viaene J. Transfer of soil contaminants to home-produced eggs and preventive measures to reduce contamination. Sci Total Environ 2009;407:4438–46. 10.1016/j.scitotenv.2008.12.041. [PubMed: 19162298]

35. Waegeneers N, Hoenig M, Goeyens L, De Temmerman L. Trace elements in home-produced eggs in Belgium: Levels and spatiotemporal distribution. Sci Total Environ 2009;407:4397–402. 10.1016/j.scitotenv.2008.10.031. [PubMed: 19046597]

36. Grace EJ, MacFarlane GR. Assessment of the bioaccumulation of metals to chicken eggs from residential backyards. Sci Total Environ 2016;563–564:256–60. 10.1016/J.SCITOTENV. 2016.04.128.

37. Van Overmeire I, Pussemier L, Hanot V, De Temmerman L, Hoenig M, Goeyens L. Chemical contamination of free-range eggs from Belgium. Food Addit Contam 2006;23:1109–22. 10.1080/02652030600699320. [PubMed: 17071513]

38. Rubio C, Ojeda I, Gutierrez AJ, Paz S, Gonzalez-Weller D, Hardisson A. Exposure assessment of trace elements in fresh eggs from free-range and home-grown hens analysed by inductively coupled plasma optical emission spectrometry (ICP-OES). J Food Compos Anal 2018;69:45–52.

39. Esposito M, Cavallo S, Chiaravalle E, Miedico O, Pellicano R, Rosato G, et al. Trace elements in free-range hen eggs in the Campania region (Italy) analyzed by inductively coupled plasma mass spectrometry (ICP-MS). Environ Monit Assess 2016;188:326 10.1007/s10661-016-5316-1. [PubMed: 27147239]

40. Uluozlu OD, Tuzen M, Mendil D, Soylak M. Assessment of trace element contents of chicken products from Turkey. J Hazard Mater 2009;163:982–7. 10.1016/j.jhazmat.2008.07.050. [PubMed: 18752893]

41. USEPA. Lead at Superfund Sites: Software and Users' Manuals. 2010 URL: https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals#overview (Accessed 28 February 2018).

42. USEPA. ASSESSING INTERMITTENT OR VARIABLE EXPOSURES AT LEAD SITES. 2003.

43. USEPA. GUIDANCE MANUAL FOR THE IEUBK MODEL FOR LEAD IN CHILDREN. 1994.

44. USEPA. Risk Assessment: Guidance for Superfund Volume 1 Human Health Evaluation Manual (Part A) Interim Final. Washington DC; 1989.

45. USEPA. OLEM Directive 9285.6–56 'Update to the Adult Lead Methodology's Default Baseline Blood Lead Concentration and Geometric Standard Deviation Parameters'. 2017.

46. USEPA. Overview of Changes From IEUBKwin version 1 build 264 to IEUBKwin version 1.1. 2009.

47. Zartarian V, Xue J, Tornero-Velez R, Brown J. Children's Lead Exposure: A Multimedia Modeling Analysis to Guide Public Health Decision-Making. Environ Health Perspect 2017;125:097009–097009. [PubMed: 28934096]

48. Dey S, Dwivedi SK. Toxic metals in hens' eggs in India: A preliminary report. Arch Environ Health Int J 2000;55:365.

49. Khan K, Naeem M. Simultaneous determination of accumulated hazardous metals in hen's eggs by atomic absorption spectroscopy. J Appl Sci 2006;6:198–201.

50. United States Food and Drug Administration. Total Diet Study Elements Results Summary Statistics – Market Baskets 2006 through 2013. College Park, Maryland; 2017.

51. Van Overmeire I, Pussemier L, Hanot V, De Temmerman L, Hoenig M, Goeyens L. Chemical contamination of free-range eggs from Belgium. Food Addit Contam 2006;23:1109–22. 10.1080/02652030600699320. [PubMed: 17071513]

52. Smith E, Kempson IM, Juhasz AL, Weber J, Rofe A, Gancarz D, et al. In vivo-in vitro and XANES spectroscopy assessments of lead bioavailability in contaminated periurban soils. Environ Sci Technol 2011;45:6145–52. 10.1021/es200653k. [PubMed: 21707121]

53. Ming H, He W, Lamb DT, Megharaj M, Naidu R. Bioavailability of lead in contaminated soil depends on the nature of bioreceptor. Ecotoxicol Environ Saf 2012;78:344–50. 10.1016/j.ecoenv.2011.11.045. [PubMed: 22226328]

54. Wijayawardena MAA, Naidu R, Megharaj M, Lamb D, Thavamani P, Kuchel T. Using soil properties to predict in vivo bioavailability of lead in soils. Chemosphere 2015;138:422–8. 10.1016/j.chemosphere.2015.06.073. [PubMed: 26151314]

55. Zia MH, Codling EE, Scheckel KG, Chaney RL. In vitro and in vivo approaches for the measurement of oral bioavailability of lead (Pb) in contaminated soils: a review. Environ Pollut Barking Essex 1987 2011;159:2320–7. 10.1016/j.envpol.2011.04.043.

56. Canfield RL, Henderson CRJ, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. N Engl J Med 2003;348:1517–26. 10.1056/NEJMoa022848. [PubMed: 12700371]

57. US Centers for Disease Control and Prevention. What do parents need to know to protect their children?. n.d. URL: https://www.cdc.gov/nceh/lead/acclpp/blood_lead_levels.htm.

58. Jusko TA, Henderson CR, Lanphear BP, Cory-Slechta DA, Parsons PJ, Canfield RL. Blood lead concentrations < 10 microg/dL and child intelligence at 6 years of age. Environ Health Perspect 2008;116:243–8. 10.1289/ehp.10424. [PubMed: 18288325]

59. USDA. United States Standards, Grades, and Weight Classes for Shell Eggs AMS 56. Washington D.C; 2000.

HIGHLIGHTS

- Backyard chicken eggs from most households providing egg samples (98%) contained detectable concentrations of lead.

- In prediction models, backyard egg consumption increased overall blood lead in children <7 years by 0.1–1.5 ug/dL, depending on quantity of egg consumption and egg lead contamination.

- Infants <1 year of age experience the greatest increase in predicted blood lead concentration from backyard egg consumption.

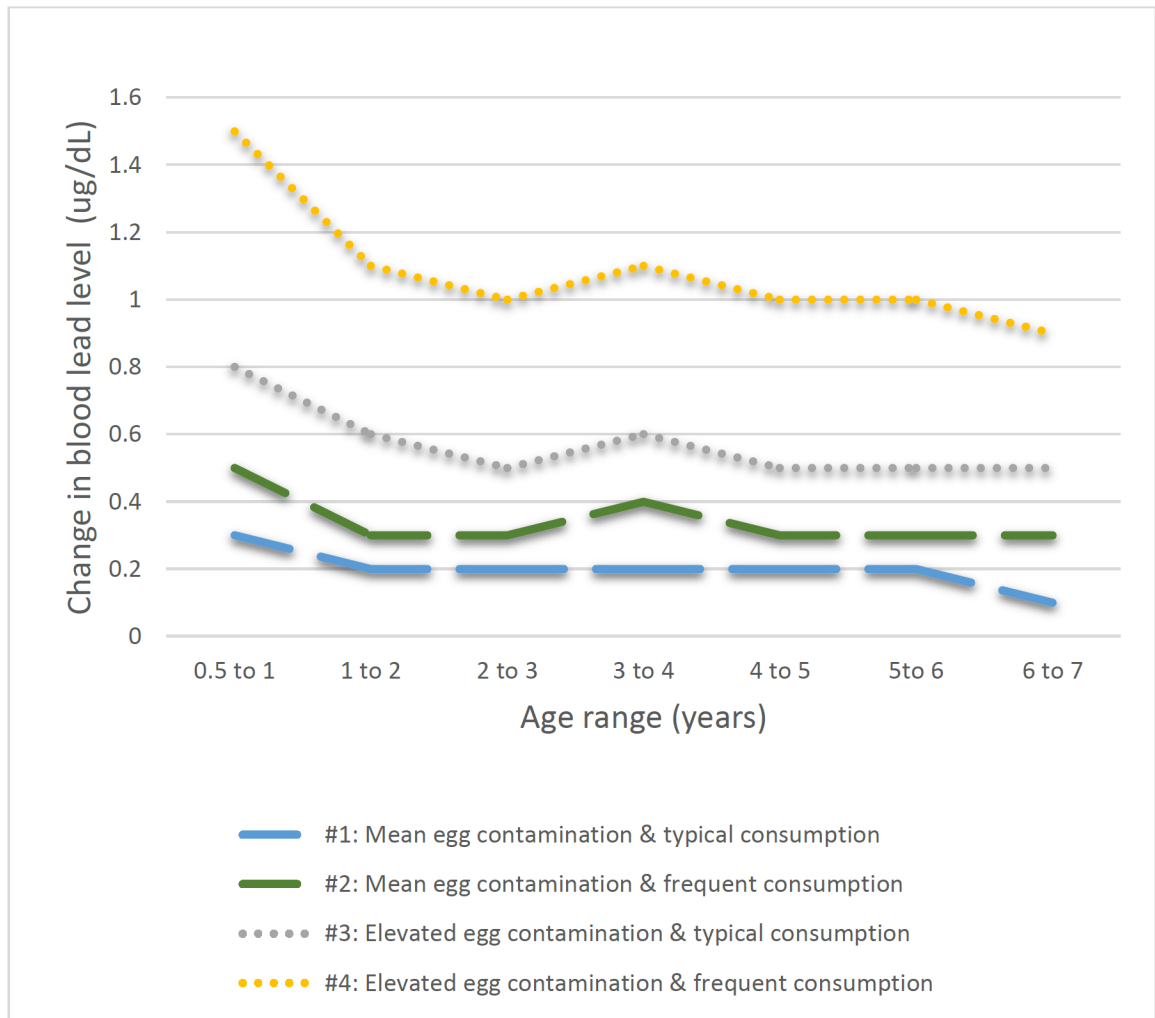- Mean household egg lead concentration was strongly positively correlated with coop soil lead concentrations.

Figure 1.
Predicted change in blood lead level for children < 7 years due to consumption of backyard chicken eggs[a]

[a] Results generated using the EPA's Integrated Uptake Biokenetic Model (IEUBK) for Lead in Children model: https://semspub.epa.gov/work/HQ/175348.pdf

Table 1.

Select descriptive data from backyard chicken egg study (n=49 households)

Characteristic	Mean (SD; range)
Household size	4(1.7; 1–11)
Number of chickens on property	6 (3.9; 2–21)
Eggs produced per week per household	26 (12.1; 8–68)
Eggs consumed per week by	
Toddlers (3 years)	2 (1.5; 0–4)
Young children (3 7 years)	2 (1.5; 0–5)
Older children (7–18 years)	4 (0–8)
Household mean egg lead concentration (ug/g)[a]	0.10 (0.18; LOD[b] –1.1)
Household mean soil lead concentration (ug/g)	238.4 (277.5; 21.3–1145.3)
	n (% of total)[c]
Children 7 years residing in household	19 (39.6)
Toddlers in household (3 years)	17(35.4)

[a] Values for 201 eggs collected from 49 households.

[b] Limit of detection (LOD) 0.0014ug/g (1.4ppb)

[c] Values may not add to 100% due to missing dat

Table 2.

Calculated daily lead intake from backyard chicken egg consumption and resultant alternate lead intake inputs for IEUBK model[*]

Variable	Scenario #1	#2	#3	#4	Units	Rationale/ Reference
Exposure Point Concentration (EPC)	0.098	0.098	0.324	0.324	ugPb/gEgg	Scenarios #1 & #2: mean egg lead concentration Scenarios #3 & #4: 95th percentile egg lead concentration
Ingestion Rate (IR)						Scenarios #1 & #3: mean consumption rate Scenarios #2 & #4: 95th percentile consumption rate Weight of an egg (50g) estimated from the USDA minimum net weight per dozen medium eggs: 21 oz.[59]
Toddler	2	3.8	2	3.8	eggs/week [*] 50 g/egg	
Young child	2.1	4.2	2.1	4.2		
Exposure Frequency (EF)		37			weeks	Assumes backyard chickens lay eggs 9 months per year (March to November) and a 2 week vacation
Averaging Time (AT)		365			days	IEUBK model is a quasi-steady-state model requiring annual exposure inputs
IEUBK Model Inputs						
Alternate Lead Intake						Daily lead intake attributable to backyard egg consumption
Toddler	0.993	1.888	3.284	6.240	ug/day	
Young child	1.043	2.086	3.449	6.897		
Absorption Fraction (AF) for eggs		49.6%			unitless	No AF for eggs in literature; default AF value for diet in IEUBK

Note: All other parameters in the IEUBK model remained at the Version 1.1 Build 11 IEUBK default values except the default maternal BLL (standard model input), which was updated per the USEPA June 2017 guidance to 0.6 ug/dL.[45]

[*] Lead intake attributable to backyard egg consumption $(\text{ug/day}) = \dfrac{EPC * IR * EF}{AT}$

Table 3.

Predicted geometric mean blood lead levels (GM BLL) and changes in GM BLL due to backyard egg consumption from IEUBK models (µg/dL)

Ages	No backyard eggs (baseline)	Mean exposure (Scenario 1)	Maximum exposure (Scenario 4)	Change in BLL from baseline to mean exposure scenario	Change in BLL from baseline to max exposure scenario
0.5 to 1	3.0	3.3	4.5	0.3	1.5
1 to 2	3.5	3.7	4.6	0.2	1.1
2 to 3	3.2	3.4	4.2	0.2	1.0
3 to 4	3.0	3.2	4.1	0.2	1.1
4 to 5	2.5	2.7	3.5	0.2	1.0
5 to 6	2.1	2.3	3.1	0.2	1.0*
6 to 7	1.9	2	2.8	0.1	0.9

The rightmost columns reflect the incremental change in geometric mean blood lead levels (GM BLLs) in children associated with egg consumption and egg contamination levels associated with three models: the default (no backyard egg consumption), the mean exposure scenario (mean egg consumption levels and mean egg lead concentrations derived from our study), and the a reasonable maximum exposure scenario (95th percentile egg consumption and egg lead concentrations).

Table 4.

Egg lead concentrations in this study and past similar studies

Egg lead concentration (ug/g)		n (eggs)	Mean	Minimum	Maximum
CLUC study (this study – household measures; total egg)		201	0.103	LOD (<0.0014ug/g)	1.061
USFDA Total Diet Study (2006–2013) – boiled eggs[50]		32	0.0004	0	0.014
New York City – urban community gardens		58	0.017	<0.01	0.167
California – backyard case study[33]		21	0.17	<0.05	0.97
Iowa – small farm case study[34, a]		5			
	yolk		0.262	<0.02	0.4
	albumen		<0.01	<0.01	<0.01
Australia – primarily urban backyard[25]		26	0.09	0.02	0.28
Belgium – backyard[36]		40	0.116	0.003	0.471
Belgium – backyard[35, b]		22	0.069	0.019	0.240

[a] Some statistics not reported in original manuscript: mean and median concentrations were calculated by replacing non-detects with detection limit 0.05

[b] Eggs samples are a composite of 10–15 eggs per household; only fall results presented in this table (fall and spring were reported separately in original manuscript)