

Is Lead (Pb) in Food in the U.S. a Children's Health Problem?

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Executive Summary

This report presents and analyzes data on lead (Pb) concentrations in the U.S. diet.

We also comment on a recent report by the Environmental Defense Fund (EDF, June 15, 2017), “Lead in food: A hidden health threat”, which asserts that “Food is a meaningful — and surprising — source of young children’s exposure to lead.”

The primary data that we (and EDF) analyze are those gathered and reported by the U.S. FDA, as part of its ongoing Total Diet Study (TDS: see <https://www.fda.gov/Food/FoodScienceResearch/TotalDietStudy/default.htm>).

We also present and analyze historical data on Pb in food, and Pb in children’s and adults’ blood.

As shown below, Pb concentrations in foods, and in people’s blood, are currently quite small: blood lead levels have declined by some 70-fold over the past 80 years. Several factors account for these significantly diminishing concentrations of Pb in both food and blood — none of which is acknowledged by EDF.

Instead, EDF suggests that food manufacturers are allowing Pb to contaminate the foods that they market, and that these manufacturers, the U.S. FDA, and parents of young children must take actions aimed to prevent this.

As explained below, few of EDF’s claims survive scrutiny. In fact, in the U.S. today, food is neither a “meaningful” nor a “surprising” source of lead. EDF provides no evidence — not even a single case-report — indicating that children in the U.S. are or have been harmed by lead in their food.

Nothing in EDF’s report, nor in the data upon which they rely, indicates that the U.S. diet is exposing children, infants, or fetuses to dangerous amounts of lead. For this and other reasons, EDF fails to demonstrate that the actions they advocate would actually improve children’s health.

Introduction

A recent report by the Environmental Defense Fund (EDF, June 15, 2017), “Lead in food: A hidden health threat” asserts that “Food is a meaningful — and surprising — source of young children’s exposure to lead.”

In fact, detecting lead (Pb) in food is not surprising but is instead entirely expected¹; and the concentrations of Pb in the U.S. diet are too small to be properly characterized as “meaningful.”

In what follows, we examine concentrations of Pb in foods. The data indicate that (i) Pb concentrations in foods in decades past were much larger than they have been in recent years, and (ii) Pb concentrations in many foods continue to decline. We discuss the reasons for these trends.

We then assess various claims and flaws in the EDF (2017) report.

We end with conclusions, acknowledgements, and references to the literature.

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Lead (Pb) and its presence in foods

Lead (Pb) is a ubiquitous metal that occurs (i) naturally and (ii) because of contamination from (primarily) past uses of Pb and Pb-based compounds in diverse products — such as gasoline, pesticides, pigments and paints, solder for food cans, drinking water distribution pipes, and other materials.

The developing brains of fetuses and young children can be harmed by cumulative exposures to moderate and even relatively low exposures Pb: based on current information, no completely safe level of Pb exposure has been established.²

Because of this lack of an identifiable, completely safe level of exposure, most modern Pb regulations are designed to limit Pb concentrations in air, water, soil, food, and other consumer products to “as low as reasonably achievable.”

Many regulations in the U.S. and elsewhere, since the 1970’s, have banned or severely restricted the use of Pb and Pb-based chemicals as gasoline additives, in paints and

¹ It appears that EDF only recently learned that food contains trace concentrations of Pb, after reading a draft document (U.S. EPA, 2017) pertaining to Pb in drinking water. Thus, although EDF may have been genuinely surprised by this, the ubiquity of Pb — and its both natural and manmade presence in air, dust, soil, food, and essentially all media — has been known for centuries. The ancient Egyptians, Greeks, Romans, Chinese and others all made extensive, intentional use of Pb — which, like all metals, once released to the environment, persists.

² For several technical reasons, the health effects, if any, of today’s typically quite low-level exposures to Pb are quite difficult to assess. These reasons include not only lead’s ubiquity, but also confounding by the influences of socio-demographic and nutritional factors on measures of children’s IQ (see, for example, Ernhart [1992 and 2006], Kaufman [2001], and Stewart *et al.* [2012]). *High-level* exposures to Pb, in contrast, are clearly toxic, to both children and adults.



pigments, in food cans and other food packaging, and in other products. Due to this and other factors, people's dietary exposures in the U.S. are currently small fractions of what they were only several decades ago.

For example, in the early 1970's, infant formula in the U.S. contained Pb at an average concentration of 100 parts per billion (ppb); by the late 1980's, Pb concentrations in infant formula had decreased to only 10 ppb; and more recently infant formula has been found to contain an average of 3 ppb Pb (Bolger *et al.*, 1991; TDS, 2007 – 2013).

Similar patterns appear for Pb concentrations in infant juices and infant foods (see below, in **Table 1**).

Table 1 Historic and current lead concentrations in infant foods (concentrations in parts per billion; ppb)			
Food product	Early 1970's	Late 1980's	Current: 2007 – 2013
Infant formula	100	10	3
Infant juices	300	11	6
Infant foods	150	13	3
Sources: Bolger <i>et al.</i> (1991) and Total Diet Study, U.S. FDA (2007 – 2013)			

Prior to the 1990's, many food cans were sealed with Pb-based solder: use of such solder was phased out in the U.S. by 1991, and banned in 1996. Food cans are now welded shut, rather than soldered — and this change alone has resulted in substantial reductions of Pb in the U.S. diet.

For example, canned baby foods once contained an *average* of 202 ppb of Pb — based on an analysis of 86 canned baby foods and juices (by Mitchell and Aldous, 1974).

And canned tuna fish (sampled and analyzed in the 1980's) once contained on the order of 750 ppb (for tuna from Pb-soldered cans) and 50 ppb (for tuna from non-Pb-soldered cans).

Today, however, *per* the TDS, canned tuna fish contains only 6 ppb of Pb.

Clearly, the essential elimination of Pb from food-packaging (and other environmental changes) has had a substantial impact on the Pb-content of many foods.

Concentrations of Pb in various raw (and/or minimally processed) foods have also decreased over time — and, as might be expected, vary substantially sample-to-sample.



Consider the data collected in a large survey of raw foods, performed in the early 1980's (by Wolnik *et al.*, 1983a, 1983b, 1985). This set of studies analyzed 288 samples of wheat, for example, which had been taken directly from major, U.S. wheat producing areas, and from field locations chosen to be *least* affected by industry, traffic, or other sources of pollution. Despite this last set of precautions and conditions, Pb concentrations in wheat were found to be (i) relatively large and (ii) widely varying — ranging from less than 1 ppb to 716 ppb — and distributed log-normally, with an average of about 37 ppb, and a median of about 17 ppb.

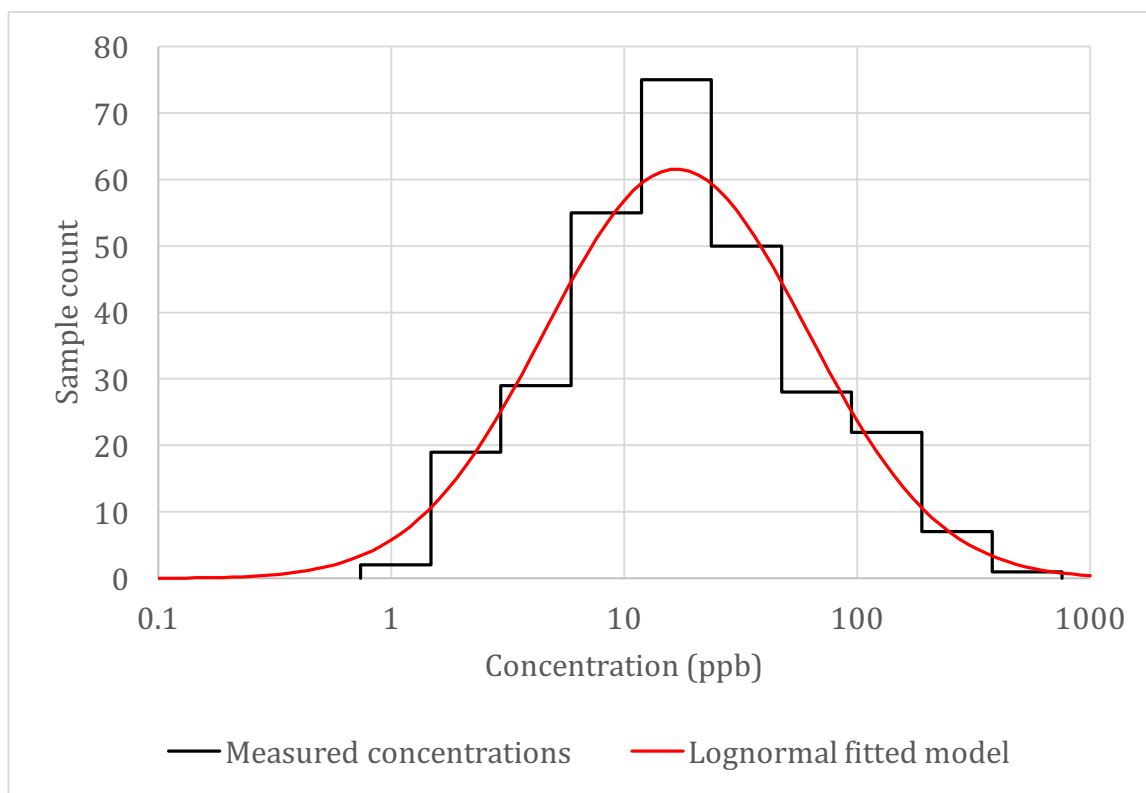


Figure 1 Distribution of lead concentrations in 288 samples of wheat in the U.S. in the early 1980's.

Data on raw foods in the 1990's and more recently also show (i) sample-to-sample variation and (ii) lower and still declining Pb-levels. These trends, in honey and in spinach, are depicted below (see **Figure 2** and **Figure 3**).³

³ The "Exponential model" shown on the graphs below effectively averages the measurements over time, taking account of variability. U.S. FDA's TDS values are composites of 3 samples, one from each of 3 cities, with different cities chosen each year. The four quarterly composites are taken from different regions of the U.S., with the regions chosen in random order each year.



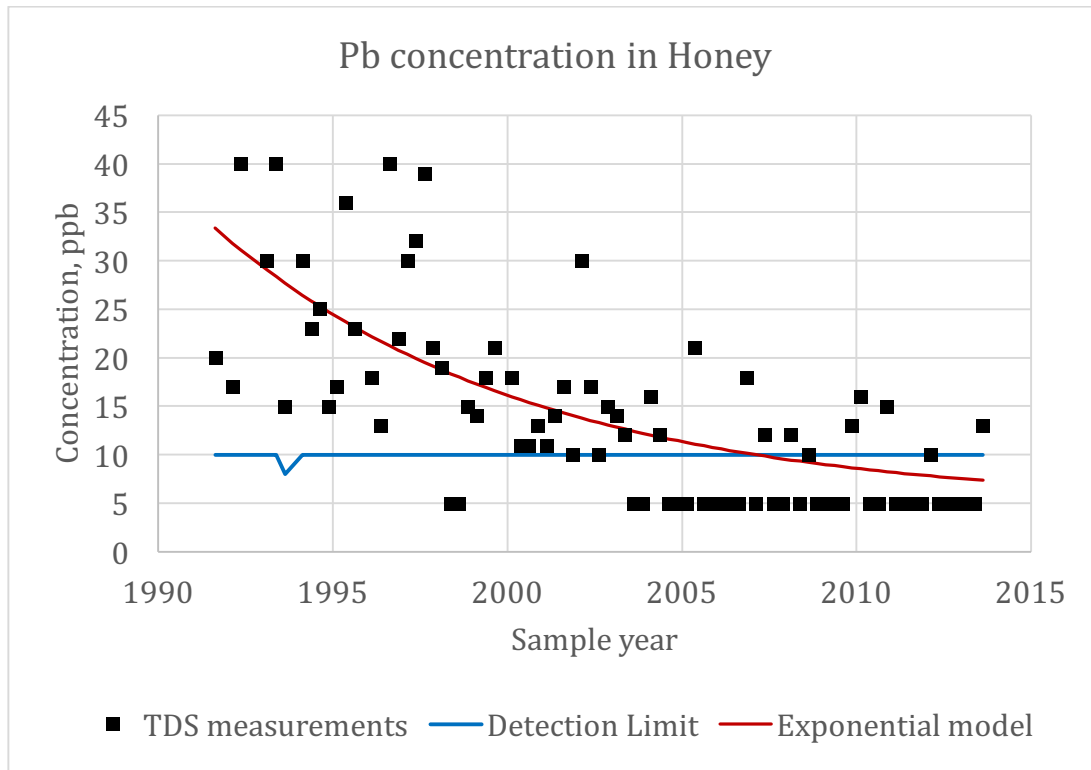


Figure 2 Lead concentrations in honey, 1991–2013. Non-detects plotted at $\frac{1}{2}$ the detection limit.

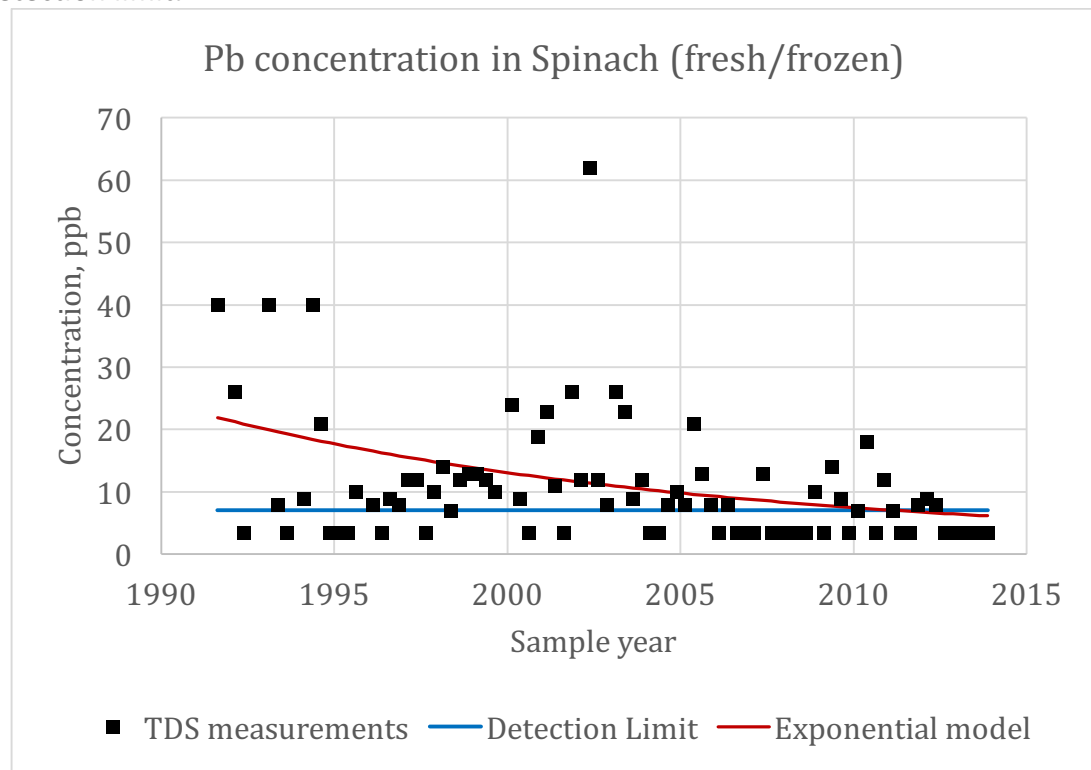


Figure 3 Lead concentrations in spinach, 1991–2013. Non-detects plotted at $\frac{1}{2}$ the detection limit.



The decreases in Pb content of honey and spinach and other raw foods are likely due to decreasing emissions of Pb into air, resulting in decreased deposition of Pb onto leaves, top soil, and other media. Of course, local environmental levels of Pb will vary, so that incorporation of Pb into any food crop will also vary.

Consider, for example, Pb in sweet potatoes. Sweet potatoes will always contain some Pb, both because root tubers incorporate small amounts of Pb from the soil in which they are grown, and because trace amounts of soil often adhere to potato skins.

Soil Pb, in turn, will always be due to some combination of (i) naturally present Pb, (ii) Pb present from prior decades of Pb deposition following combustion of leaded gasoline and other sources, and (iii) Pb present due to deposition from current emission-sources. As noted above, this third set of sources is typically decreasing, even today, but will never vanish, and impacts from the first and second sources will change only if and as soils with different Pb-contents are mixed or otherwise shifted.

As shown below (see **Figure 4**), FDA's TDS data indicate that Pb levels in baby food (BF) sweet potatoes have declined since the 1990's, from about 24 ppb to about 12 ppb, but are still variable, as one would expect.⁴

⁴ We focus on baby foods here, and elsewhere in this report, because infants are, as noted earlier, among the groups most susceptible to the adverse effects of Pb.



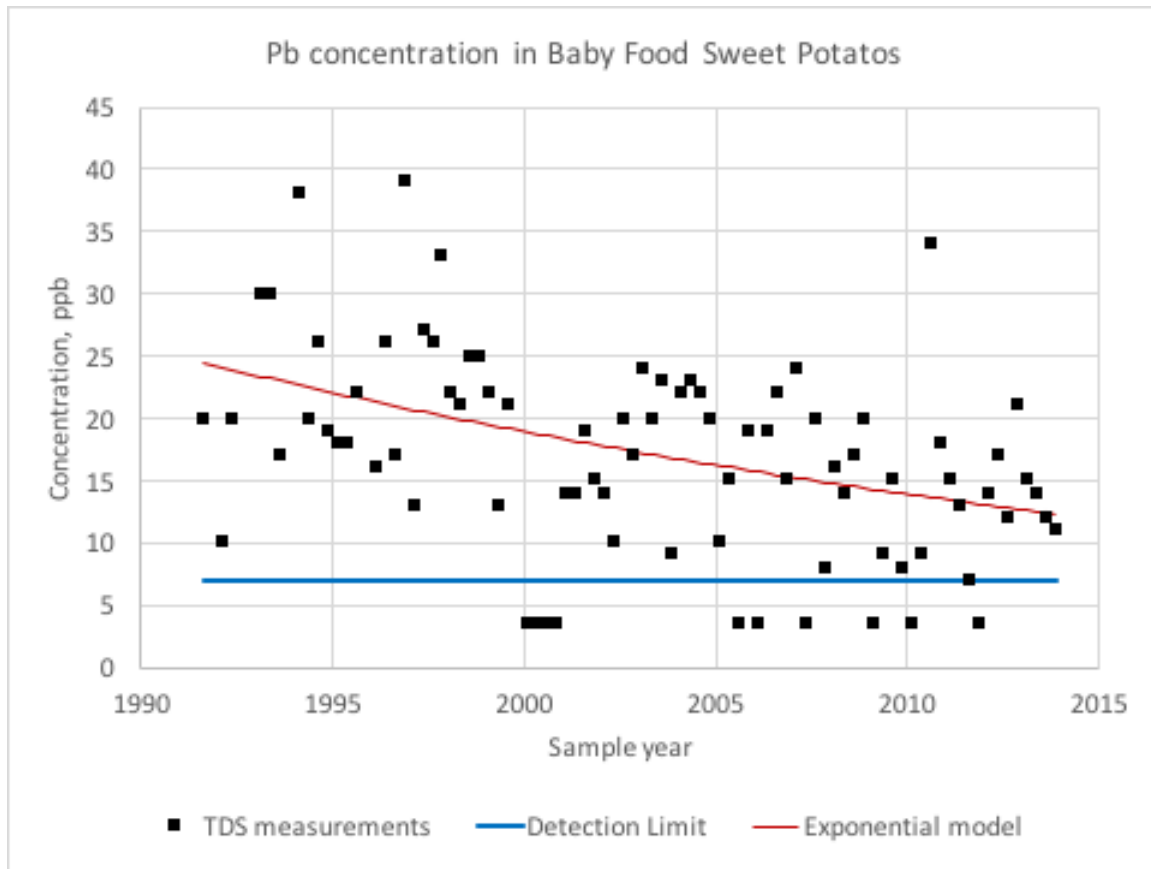


Figure 4 Lead concentrations in sweet potatoes sold as baby food. Non-detects are plotted at $\frac{1}{2}$ the detection limit.

Chocolate products provide another example of how environmental Pb may be incorporated into foods, even at today's low levels. Studying cocoa beans, shells, and cocoa and chocolate products, Rankin *et al.* (2005) showed that the beans had very low lead content (as confirmed by Yanus *et al.*, 2014), but that the shells could easily absorb environmental lead (*e.g.* from exhaust from leaded gasoline used in the production area at the time).

Nonetheless, as shown in **Figure 5**, the lead contamination of final products (such as candy bars) is steadily decreasing.



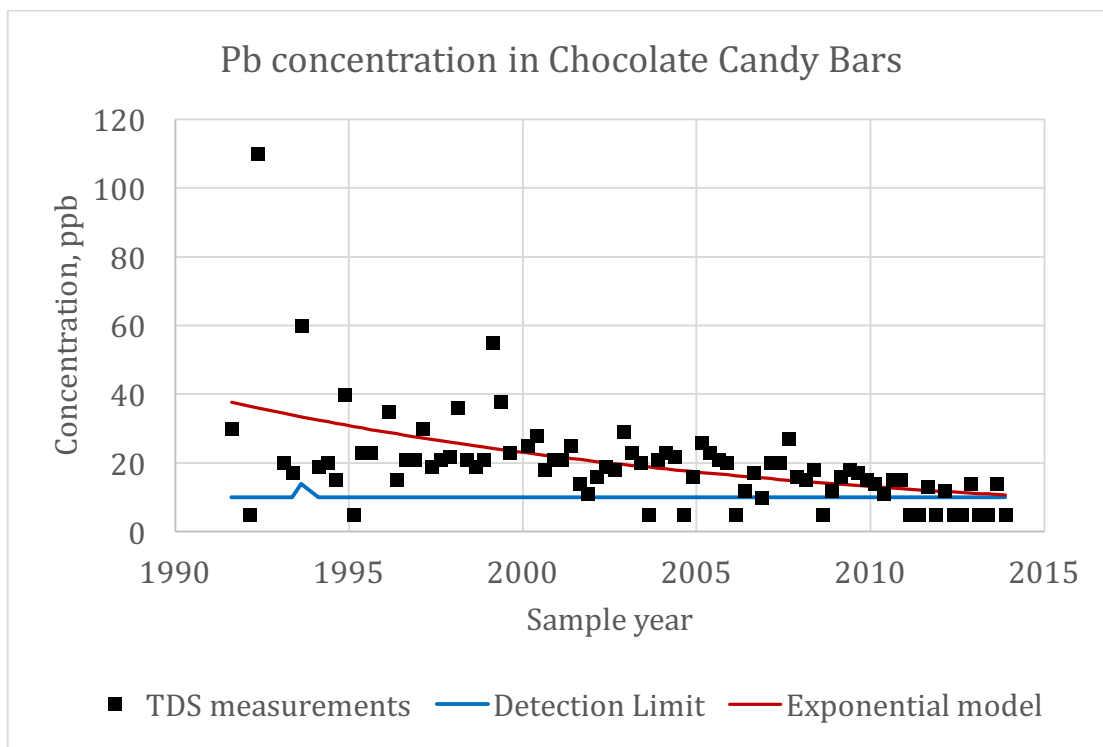


Figure 5 Lead concentrations in chocolate candy bars, 1991–2013. Non-detects are plotted as ½ the detection limit.

These changes with time, and sample-to-sample variations, are also apparent in fruit juices.

In 1979 – 1981, apple juice, for example, contained an average Pb concentration of 34 ppb — with individual samples ranging in Pb content from 12 to 164 ppb (Mattick & Moyer, 1983).

Since then, concentrations of Pb in apple juice have decreased significantly, so that the content of Pb in apple juice is now typically not detectable (at a lower limit of detection of 4 parts per billion [TDS, 2007 – 2013]; see **Figure 6** and **Figure 7**).



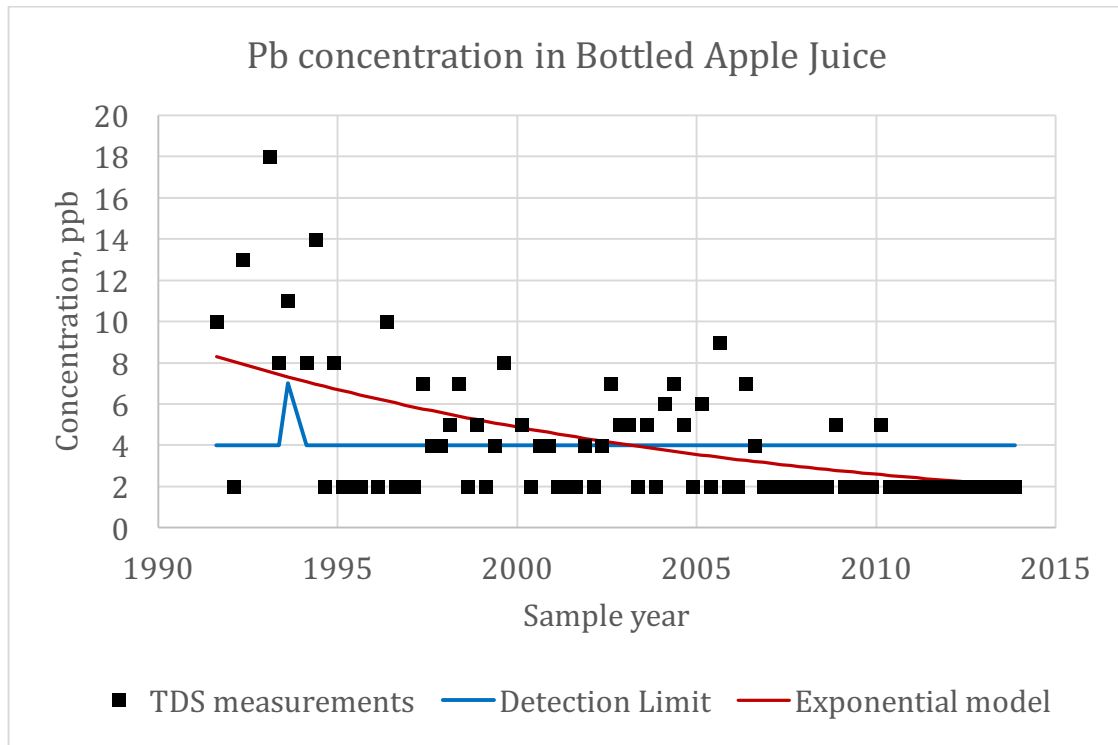


Figure 6 Lead concentration in bottled apple juice, 1991–2013. Non-detects plotted as $\frac{1}{2}$ the detection limit.

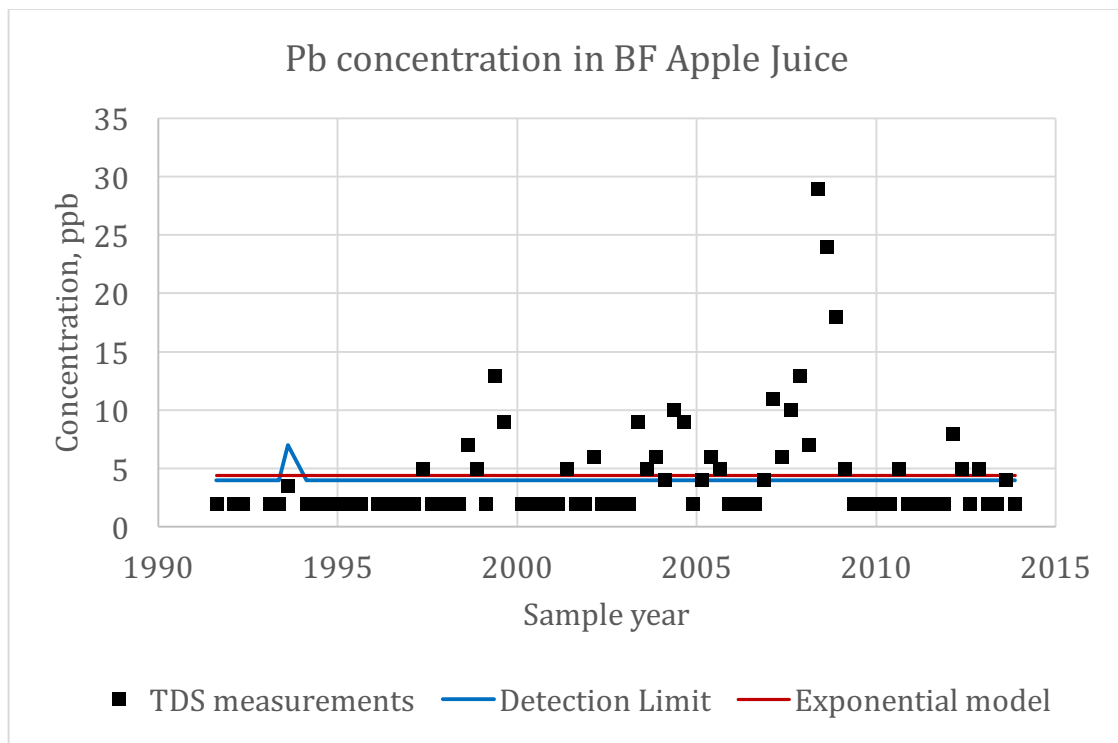


Figure 7 Lead concentrations in apple juice sold as baby food (BF), 1991–2013. Non-detects plotted as $\frac{1}{2}$ the detection limit.



For the U.S. diet as a whole, we can use data from Adams (1991) and FDA's TDS (1991–2013) to estimate overall, populational intakes, as well as trends over time.

Figure 8, below, shows estimates of total dietary lead intakes for 2-year-old children and 25–30-year-old women — both sensitive population groups (the latter because of the risk to their fetuses if they are pregnant) — obtained from the TDS combined with food consumption surveys (the 1987–1988 National Food Consumption Survey, and the 1994–1998 Continuing Survey of Food Intake by Individuals).⁵ As shown, current Pb ingestion-rates for both groups are now on the order of only 3 micrograms per person per day, and declining still.

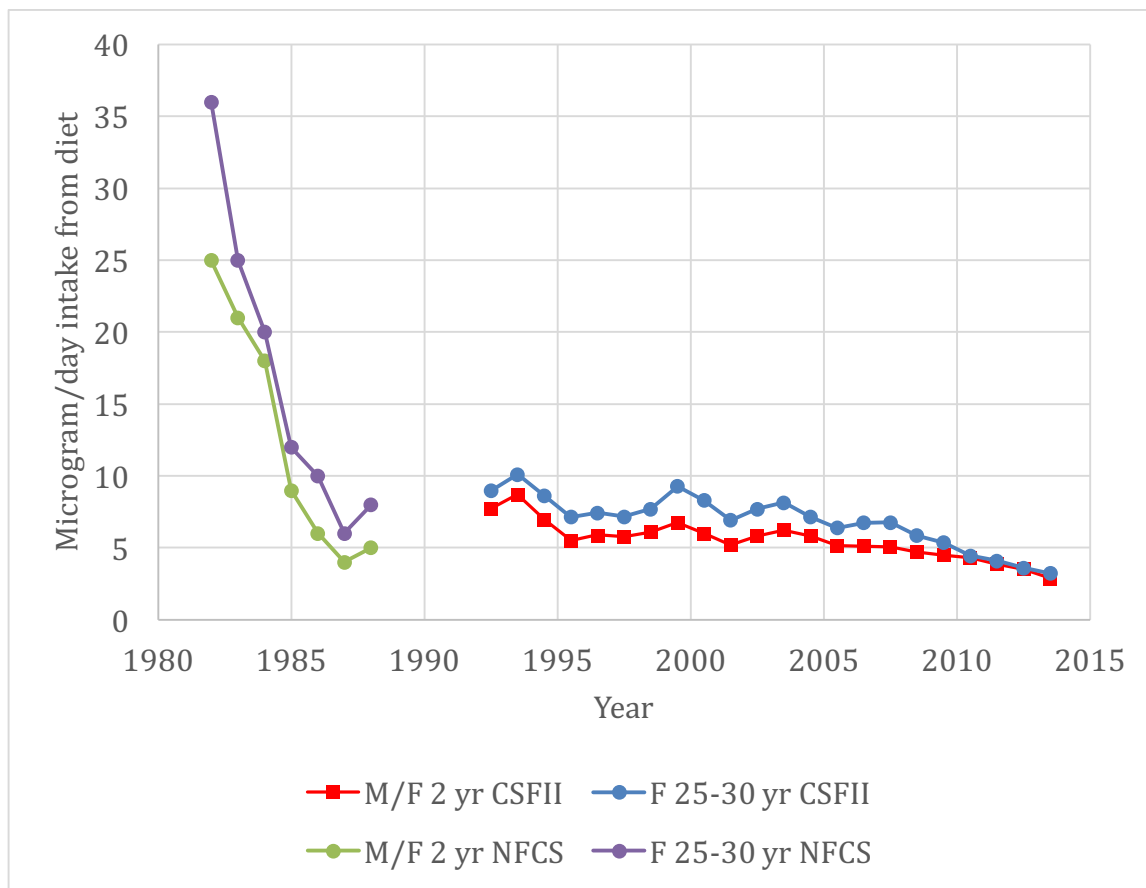


Figure 8 Lead intakes estimated from food consumption surveys (NFCS and CSFII) combined with the TDS for sensitive population groups.

⁵ The discontinuity depicted in estimated levels between the two time periods is likely due to the different food surveys used, combined with the higher detection limits in the earlier TDS and the assumptions about Pb concentrations in foods with no detected Pb. The estimated intakes are quite uncertain in the most recent years, because of the high preponderance of non-detects in the foods most consumed. Of course, as Pb concentrations continue to fall, detecting Pb in foods becomes increasingly difficult.



Despite the currently low level of Pb in the U.S. diet, national and international groups of regulators, farmers, food producers and manufacturers, and others continue to be actively engaged in both researching and attempting to minimize, whenever feasible, the concentrations of Pb (and other contaminants) in our diets. Nationally, these efforts include guidelines and regulations promulgated by U.S. FDA (notably, the Hazard Analysis Critical Control Point [HACCP] set of guidelines that act to minimize food-contamination; FDA [2017]), U.S. EPA, and others; internationally, relevant guidelines and regulations include those designed by Codex Alimentarius Commission (Codex, 2015) and by the Joint Food Agricultural Organization/World Health Organization Expert Committee on Food Additives (JECFA, 2011). Various companies, trade associations, and other groups have also developed relevant guidelines (see for example, the good manufacturing practices [GMPs] designed by the International Maple Syrup Institute, 2015).

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Lead (Pb) in people's blood

Naturally, these declining and now very low levels of Pb in the U.S. diet have led to declining and now very low levels of Pb in our bodies (typically assessed by measurements of Pb concentrations in blood).

For example, in the mid-1930's, *average* blood Pb concentrations in the general U.S. population were on the order of 55 micrograms of Pb per 100 milliliters of blood (55 $\mu\text{g}/\text{dL}$), with a range extending from 40 to 70 $\mu\text{g}/\text{dL}$: none of the people (who ranged in age from 15 to 72 years old) whose blood was sampled was considered to be lead-poisoned (Tompsett & Anderson, 1935).

Today, blood Pb levels this high would be cause for alarm and often clinical treatment; but in those days, such Pb body-burdens were, clearly, entirely normal and unremarkable.

By 1976–1980, in contrast, the median concentration of Pb in the blood of U.S. children aged 1 to 5 years had fallen to about 15 $\mu\text{g}/\text{dL}$ of blood (NCHS, 1984).

And since then, concentrations have fallen more or less steadily with time, such that the current (measured in 2013–2014) geometric mean concentration of Pb in the blood of U.S. children (aged 1 to 5 years) is only 0.78 $\mu\text{g}/\text{dL}$ (Tsoi *et al.*, 2016).

In other words, over about 80 years (from the 1930's through today), blood Pb levels in the U.S. have decreased by about 70-fold. Given the persistence of Pb in the environment, such a substantial (and continuing) decrease is remarkable.

Similarly, the proportion of young children (aged 1 to 5 years) in the U.S. whose blood Pb concentrations exceed 5 micrograms per deciliter (which is the current “upper reference level” for blood Pb), has fallen from about 10% (as recently as 1999–2000) to only about 0.5% (in 2013–2014; Tsoi *et al.*, 2016).

Of course, 0.5% of children in the U.S. is still a large number (about 100,000); however, as recently as the 1976–1980 survey (NCHS, 1984), *more than 95% of all children and*



adults in the U.S. had blood Pb concentrations that were larger than this “upper reference level” of 5 $\mu\text{g}/\text{dL}$.

These days, children with much higher-than-average blood Pb concentrations are believed to be overexposed to Pb primarily via contaminated dust, soil, and drinking water — and not via their diets (U.S. EPA, 2017). Even for children in the highest (90th to 100th) percentile of exposure, food contributes only a small fraction of their overall intake of lead (U.S. EPA, 2017, Exhibit 55).

Moreover, detailed studies (Budd *et al.*, 2000 and 2004) indicate that modern children’s exposures to lead are approaching those of pre-historic (indeed, pre-metal-working) populations — when practically all lead intake would have been due to ingestion of foods and beverages that had accumulated only naturally occurring Pb from weathered rocks, soils, and dusts.

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The EDF (2017) Report

A recent report by the Environmental Defense Fund (EDF, June 15, 2017), “Lead in food: A hidden health threat”, asserts that “Food is a meaningful — and surprising — source of young children’s exposure to lead.” As indicated above, however, finding lead (Pb) in food is not surprising but is instead entirely expected. And the current concentrations of Pb in the U.S. diet are far too small to be properly characterized as “meaningful.”

Indeed, EDF’s own analysis suggests that lead in food in the U.S. could be responsible for blood lead concentrations in children of about 0.46 micrograms per deciliter ($\mu\text{g}/\text{dL}$) — which is much smaller than CDC’s reference value (currently set at 5 $\mu\text{g}/\text{dL}$), and of no known clinical significance.⁶

Moreover, there is little to nothing surprising in the data that EDF examines. *It is entirely expected* that, as children’s exposures to lead in paint, dust, soil, water, air, and other sources decrease, food should become a *relatively* large contributor to lead exposure, since lead is present everywhere in the environment and in all foods.

EDF claims to have “found that more than 1 million children consume more lead than FDA’s limit” for dietary lead, by which they mean 6 micrograms of lead per child per day. In fact, EDF has found no such thing.

Children’s diets vary from day to day, as do concentrations of lead in different batches of any given food-type. *On any given day*, a child, indeed a million children in the U.S., may ingest more than 6 micrograms of lead via his (their) diet— but on any other given day, those children may ingest, say, only 1 microgram or less of dietary lead. Importantly, lead in children’s blood has a relatively long half-life (on the order of 30

⁶ EDF’s calculation of this very small blood lead concentration due to food is not mentioned in the body of the EDF report (nor in its Press Release; <https://www.edf.org/media/edf-report-finds-lead-1-5-baby-food-samples>), but is instead presented only in an Appendix to the report.



days), so that what matters is children's lead intakes — from all sources — *over one to several months*, rather than during any single day.

Indeed, the EPA simulation data upon which EDF itself relies indicate that U.S. children's long-term population average consumption of lead from food is on the order of only 2 to 3 micrograms of lead per day (and, for infants, only 0.3 to 0.7 micrograms of lead per day) — which ingestion-rates are (i) well within FDA's guideline and, as indicated below, (ii) a small fraction of the amounts of lead ingested by all Americans only decades ago.

EDF seems to not understand that FDA's TDS data, combined with the NHANES⁷ food intake data, cannot be used to evaluate *long-term* average exposures to *individual children* — but instead indicates only the distributions, based on 280 foods regularly sampled, of *daily* intakes among U.S. children, and long-term *population* average exposures.

EDF's analysis of the data from FDA's TDS is misleading in several other respects.

First, EDF combined data from 11 years of FDA's TDS (from 2003 to 2013), but, as detailed above, Pb levels in many foods have been dropping even over this short period of time — so that, all other things being equal, EDF's conclusions may be out of date. Indeed, EPA (in their simulation study that EDF cited) recognized this, and so limited its analysis of FDA's TDS data to the most recently available seven years.

For example, EDF claims (correctly) that fruit juices marketed as baby food, *when grouped together over the past 11 years*, consistently had higher lead detection rates than regular fruit juices. What EDF fails to notice, however, is the *time variation* in these differences, and the reasons for them.

For apple juice for example, splitting the 2003–2013 date range examined by EDF into an earlier and a more recent date-range yields the following results:

Apple juice	TDS Years 2003–2013		TDS Years 2003–2006		TDS Years 2007–2013	
	Detections/ Total # samples	Mean	Detections/ Total	Mean	Detections/ Total	Mean
Baby juice	24/44	4.8 ppb	10/16	3.9 ppb	14/28	5.4 ppb
Regular (non-baby food)	11/44	1.5 ppb	9/16	3.4 ppb	2/28	0.4 ppb
p-value*	0.004		0.5		0.00039	

* Fisher exact test, 1-sided

⁷ National Health and Nutrition Examination Survey, <https://www.cdc.gov/nchs/nhanes/>; the dietary component is the United States Department of Agriculture's "What We Eat In America," <https://www.ars.usda.gov/northeast-area/beltsville-md/beltsville-human-nutrition-research-center/food-surveys-research-group/docs/wweianhanes-overview/>



As shown, the difference noted by EDF (over all 11 years):

- (i) is due entirely to the most recent time-period (shown in the right-most set of columns); and
- (ii) arises because of a substantial *decrease in the detection rate* of Pb in regular apple juice (with only 2 of 28 samples containing detectable concentrations), which in turn affects the *estimated* mean concentration of Pb in that juice — and not because of any apparent increase in Pb concentrations in baby food juice.

Moreover, looking over a longer time span (see above, in **Figure 6** and **Figure 7**), it appears that the entire difference (between regular apple juice and baby food apple juice) is due to three high measurements in 2008 in apple juice marketed as baby food — high measurements that have subsequently not been repeated.

Next, EDF's analysis focuses almost exclusively on *percentages* of foods and beverages in which FDA's TDS detected Pb — but *this is a meaningless statistic*, since what matters is *how much* Pb is in food, and not how frequently Pb can be detected in food.

Of course, as EDF presumably knows but fails to mention, with a sufficiently sensitive method of detection, 100% of all foods will have detectable Pb.

Moreover, as one might expect, Pb is much easier to detect in clear liquids, such as juices, than it is in many solid foods. Indeed, in FDA's TDS, the limit of detection of Pb in juice is 4 ppb, while in various solid foods, the lower limit of detection is typically 5, 7, or 10 ppb, with an appreciable number of foods at 20 ppb. Clearly, if a sample of solid food contains, say, 19 ppb Pb, but the lower limit of detection for Pb is 20 ppb, then that food will appear, by EDF's "method", to be lead-free, even though, in reality, it certainly is not.

Next, EDF reproduces, in its Table 2, some of FDA's data from its "special study of heavy metals in infant and toddler foods." But it leaves out the most important set of statistics, clearly presented by FDA, which are the *average* levels of Pb detected in these foods.

EDF chooses instead to report only the maximum level detected in any sample of each food. But why report that one sample of raisins, for example, was found to contain 151 ppb of Pb, without also reporting, as FDA does, that raisins on average were found to contain only 18 ppb? Or that the worst single sample of teething biscuits contained 131 ppb of Pb, without also telling readers that teething biscuits on average contain only 12 ppb of Pb?

Of course, in any given food, concentrations of lead will vary, sometimes quite widely.

This is why it is the *averages* of samples over time, or the averages over many samples taken at a single time, that provide relevant information on the time-averaged lead intakes, which in turn affect blood lead levels in consumers.

EDF asserts that "FDA and food manufacturers can and must do better to reduce lead in food", without acknowledging that lead-levels in food in the U.S. are (i) at historically



low concentrations, and (ii) appear to be due largely — and perhaps entirely — to factors beyond FDA's or manufacturers' control — such as incorporation of lead from soil, dust, and air into food-crops and food-producing animals.

EDF seems unaware of (or at least makes no reference to) the substantial progress that has been made, by both FDA and the food industry, to eliminate and/or strictly minimize the sources of lead-contamination over which the Agency and/or the industry *do* have control. To the extent that some of this progress has been due to environmental and public health advocacy, from at least the 1970's through today, such an omission seems odd.

Moreover, EDF failed to note that many foods that contain the highest levels of Pb are those that are the least “processed”. These include not only raisins, as noted above, but also honey, shrimp, rice, quinoa, and other “natural” foods that, presumably, contain Pb only, or certainly largely, because of their growing (and in the case of honey, gathering) environments, and not because of food manufacturing or food packaging.

EDF suggests that companies that sell food should “ensure[s] that, especially for baby food, there is less than 1 ppb of lead in the food and juices they sell.” EDF provides (i) no justification for this limit, (ii) no evidence that such a limit would be achievable for foods which, inevitably, reflect the environments in which they are grown or otherwise produced, and (iii) no evidence that U.S. children's health would be improved were all foods to be able somehow to meet this limit. As shown above, it is currently impossible for many natural foods (such as honey and sweet potatoes) to consistently contain less than 1 ppb of Pb. Does EDF really believe that children are being poisoned by the Pb content of these foods?

This is *not* to say that both the FDA and the food industry should not continue to be vigilant with regard to contamination by Pb or any other potentially toxic substance; indeed they are and they should be.

It is instead to say that, although EDF claims to find it “surprising” that Pb can be detected in food, no one familiar with the ubiquity of lead, its historic uses, and its persistence in our topsoil and other media, would or should be so surprised. To *not* find trace levels of lead in food, in any industrial or post-industrial society's food supply, would be the surprise.



Conclusions

Taken together, then, all of the relevant data indicate that foods in the U.S. today are not significant sources of lead.

Decades ago, many foods contained much higher lead levels, due primarily to substantial contamination of either the growing environment (to leaves and topsoil, primarily through deposition from the atmosphere) and/or to lead that migrated into food from lead-based soldered cans and other materials.

The recent EDF report, which alleges that the U.S. food supply today contains dangerous amounts of Pb, fails to provide reliable information, including from several of the governmental sources that it uses for other purposes.

EDF alleges that lead in food is a “hidden health threat,” but the data are not hidden (they have been public all along), and they do not signal a health threat.

The EDF (2017) report fails to demonstrate that the actions it urges would in fact improve children’s health.

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Acknowledgement

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We take responsibility for any errors or omissions.

Please feel free to write to us with any questions, comments, or concerns.
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