Determinants of childhood lead exposure in the post leaded-petrol era; the Tooth Fairy cohort from Newcastle upon Tyne

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ABSTRACT

Lead is an environmental contaminant causing irreversible health effects in children. We used dentine lead levels as a measure of early life lead exposure and explored determinants of lead exposure in children living in Newcastle upon Tyne, a historically industrialised UK city, in a cohort born since legislation was introduced to remove lead from petrol, paint and water pipes. The 'Tooth Fairy study' cohort comprised 69 children aged 5-8 years. We collected upper deciduous incisors from children and questionnaire data from their parents in 2005. We measured lead levels in pre and post-natal enamel and dentine using Laser Ablation Inductively Coupled Plasma Mass Spectrometry, and assessed associations between dentine lead levels and residential, dietary, lifestyle and socio-economic characteristics. Dentine lead levels were low (mean 0.26µg/g, range 0.06-0.77), however we observed considerable variability in dentine lead levels within and between children suggestive of differing exposure levels and/or exposure sources across this population. Variables earlier documented to be associated with childhood lead levels were not found to be significant determinants of dentine lead levels in this study. Exposure pathways should continue to be investigated to enable targeted interventions and prevention of lead-induced health impacts in vulnerable populations.

Keywords:

Lead, Environmental Exposure, Lead Poisoning, Child health, Dentine.

INTRODUCTION

Lead is a well-established neurotoxicant, with many studies demonstrating adverse impacts of lead exposure on neurological development, intellectual functioning and social and behavioural conduct.[1] Children are particularly vulnerable to these neurological and developmental effects of lead, in part because of their developing physiology, but also because of age-specific behavioural traits, such as hand-to-mouth activity, which increases their likelihood of exposure.

Environmental lead contamination is a consequence of human activities, such as mining and smelting, sewage sludge usage in agriculture and, historically, contamination from vehicle emissions.[1] Lead levels in humans have been found to be associated with various demographic, environmental and socio-economic factors including: age (exposure peaks at around 1-2 years);[2] ethnicity, notably in the USA;[3] socio-economic factors (including social class, income and education);[4] householders' occupations and hobbies[5]; pica (the consumption of non-food substances);[6] garden and allotment soil lead levels[7] and consumption of allotment/home grown vegetables;[8] age of home and home renovations (due to lead water pipes and lead-paint);[9] home location (e.g. urban, industrial); diet, especially bottle fed infants if water is contaminated,[10] and those with iron deficiency;[11] smoking,[12] and; use of lead-contaminated make-up[13] or lead glazed ceramics.[14]

Legislation to reduce environmental emissions to bring human exposure to 'acceptable' levels (i.e. blood lead levels below the USA Centres for Disease Control and Prevention level of concern of 10μg/dL) was introduced across Europe and North America in the 1970s, and addressed major emissions/exposure sources such as leaded petrol, lead-containing household paint, and lead in water. While such strategies were successful in reducing lead emissions,

environmental contamination and human body burdens,[15] it is now recognised that blood lead levels below 10μg/dL can adversely impact health,[16] and lead remains a global public health issue, with lead poisoning responsible for 0.6% of the global burden of disease,[17] a burden amounting to 1.20% of world GDP in 2011.[18] UK studies conducted since the removal of lead from petrol and paint have found differences in children's blood lead levels by social class,[4] but little evidence of other determinants of exposure. There is, therefore, a need to characterise current exposure levels, and identify key routes of exposure.

Blood lead levels are commonly used to measure lead exposure, but the half-life of lead in blood is in the order of weeks,[19] meaning blood lead levels represent only recent exposure. Teeth incorporate trace elements, including lead, during their mineralisation, and primary teeth naturally exfoliate at around 6 years of age, providing a non-invasive alternative matrix within which to measure long term lead exposure.[19] Tooth development begins prenatally ~14-16 weeks after fertilization, and the mineralisation of enamel (the outer layer of the tooth) and dentine (the inner layer of the tooth) follows a regular incremental pattern corresponding to the circadian growth rhythm. The physiological stress of birth results in the formation of a 'neonatal line', an accentuated feature that can be seen in a polished thin section of a tooth, which can be used to distinguish enamel and dentine mineralised before and after birth.[20] This neonatal line has been used to study the intensity of lead exposure during the pre- and postnatal periods of development.[21]

Deciduous teeth from a cohort of children resident in Newcastle upon Tyne, a historically industrial city in north east England, were collected, along with detailed questionnaire data, to assess pre and post-natal lead exposure, and identify key determinants of exposure in children born since the removal of lead from petrol, paint and water pipes.

METHODS

Study location

Newcastle upon Tyne in north east England was a centre for heavy industry since the turn of the nineteenth century, and the region has a history of extensive lead mining. Although heavy industry in the region decreased drastically in the 20th century, many areas remain impacted by historic environmental lead contamination. The socio-economic range of the population of Newcastle mirrors that of the whole of England, with some of the nation's most and least deprived electoral wards as measured using the area-based Index of Multiple Deprivation (IMD).

Recruitment

Children residing in Newcastle upon Tyne in 2005 aged 5-8 years old who had lost an upper deciduous incisor (naturally exfoliated) were eligible for recruitment into the study. We recruited children via their primary or first schools, with schools randomly selected from each of the five quintiles of school deprivation. Quintiles were derived based on the proportion of children receiving free school meals in each school. Covering letters and self-completion questionnaires (eliciting information on demographic and residential characteristics, dietary behaviour, and socio-economic status (educational achievement, parental occupation and income)) were sent to the parents of eligible children via the school. £10 vouchers were offered to children who had lost a front tooth and would provide that tooth along with the parental consent form and questionnaire. We excluded children with the following characteristics: a) lived outside of the city boundaries for more than one year, b) were to be moving within the next four months, c) had a sibling with history of lead poisoning, d) attended a special needs school. We trained school nurses to collect, store (in sterile solution)

and label the naturally shed milk teeth and collect parental consent forms from participating children.

We assigned area-based measures of deprivation to each child based on the postcode of their main residence and school using the 2007 IMD score of the super output area (a census based unit of geography containing on average 1500 residents) that contained their home/school postcode, linked via ArcView GIS 3.2 (Redlands, California). The IMD score is based on seven domains of deprivation: income, employment, health deprivation and disability, education skills and training, barriers to housing and services, crime and living environment.[22]

Sample preparation and analysis

Teeth were sectioned longitudinally to a thickness of 300µm and polished at the Hard Tissue Laboratory, School of Dental Sciences, Newcastle University. The neonatal line was clearly visible in all sections, allowing identification of pre- and post-natally formed enamel and dentine for sampling.[21, 23, 24]

To investigate tooth lead levels, the individual dental phases (pre and post-natal enamel and dentine) were analysed by laser ablation inductively coupled plasma mass spectrometry (LA-ICP-MS) at the School of Earth and Environment, University of Leeds. Analytical details are provided in full elsewhere.[25] In brief the analysis was performed using a GeoLas 193nm ArF excimer laser coupled to an Agilent 7500c ICP-mass spectrometer. During ablation the individual dental phases were clearly visible allowing the ablation points (4-5/phase) to be precisely located, avoiding micro-fractures in the enamel, the anomalously enriched outer surface layer of enamel[26, 27], areas adjacent to dental caries or excessive enamel wear, and

dentine close to the pulpal cavity. However, the ablation pits (100µm diameter) were randomly positioned and were not aligned to sample the same time interval of enamel or dentine secretion for each tooth. Ion intensities at isotope masses ^{208}Pb and ^{40}Ca were converted into lead/calcium ratios using data for standard reference materials, and absolute Pb concentrations for dentine and enamel calculated by normalising to 26.5 wt.% and 37.4wt% Ca respectively.[21] Routine detection limits (LOD) were typically 0.01–0.02 µgPb/g.

The associations between dentine lead levels and the questionnaire variables were assessed using independent t-tests (for dichotomised variables) and one-way ANOVA (categorised variables). Multivariable linear regression analysis was used to explore potential determinants of lead levels in dentine, and in pre and post-natal enamel, taking a P-value of <0.05 as statistically significant.

Ethics

The 2005 Tooth Fairy study was reviewed and approved by the County Durham and Darlington Local Research Ethics Committee (Reference Number: 05/Q0904/10).

RESULTS

Sixty nine respondents met the eligibility criteria, provided a tooth, and completed the questionnaire (36 female, 33 male; aged 5 to 8 years old). Children were recruited from eighteen schools located within the Newcastle city boundaries.

Lead levels in pre- and post-natal enamel (mean±SD 0.13±0.10 and 0.11±0.16µg/g respectively) were lower than in dentine (mean±SD 0.26±0.16µg/g, $p<0.01$), and tended to be lower in each, although not every child, as shown in Figure 1. All children had detectable levels of lead in dentine (range 0.06-0.77µg/g), 68/69 had detectable levels in pre-natal enamel (range <LOD-0.49µg/g), but only 41/69 children had detectable lead levels in post-natal enamel (range <LOD-0.77µg/g). Lead levels in pre-natal enamel were moderately correlated with levels in post-natal enamel (Pearson correlation coefficient (R)=0.42, p=<0.01) and dentine (R=0.40, p=<0.01), but post-natal enamel and dentine lead levels were not significantly correlated (R=0.22, p=0.67).

Figure 1 here

Trace element concentrations in enamel are affected by the position of the sampling point across a tooth section and require careful interpretation in order to accurately reflect chronological exposure.[28, 29] Although dentine close to the pulp chamber has higher levels of lead than other regions, Shepherd et al. (2012) presented evidence that the sampling position within dentine is not a confounding factor in interpreting trace element concentrations. In addition, dentine incorporates higher levels of lead, and is likely to represent a longer period of exposure than enamel.[30, 31] We decided to focus on dentine lead levels as a marker of long-

term lead exposure in this study, although comparisons with pre and post-natal enamel lead levels are made where appropriate.

Dentine lead levels by the various demographic, socio-economic, residential, dietary, behavioural and other participant characteristics are shown in table 1. None of the socioeconomic measures (including highest educational level of the main wage earner, household monthly income, and the area-based deprivation scores of the home and school address) were significantly associated with dentine lead levels. Although more than two thirds of the children lived in homes built before 1960 when lead pipes and lead paint could be a potential source of exposure, and more than 80% lived in homes that had undergone renovations, neither of these characteristics significantly influenced dentine lead levels.

Table 1 here

Two thirds of children reported living in homes with hard floors in the living spaces. Within this subset of the cohort dentine lead levels (but not pre or post-natal enamel lead levels, data not shown) were significantly higher in children living in homes with hard floors which were cleaned using only a dust pan and brush or broom (i.e. dry cleaned) compared to children living in homes with hard floors which were cleaned using vacuum/wet cleaned with mop (0.67 versus 0.22μg/g, $p<0.01$), however, only two children were in this former group.

Almost a quarter of children lived in homes where fruit and vegetables were grown in the garden or an allotment, and, of these, most (76.5%) ate the fruits and vegetables grown there. Consumption of home grown vegetables did not influence dentine lead levels, however, children from households that did not grow fruit or vegetables had significantly higher

dentine lead levels (0.28 versus 0.19μg/g, p=0.05). Children who reported drinking less than 0.28L (1/2 pint) of tap water per day had higher dentine lead levels than those who drank more (0.34 versus 0.23μg/g, p=0.02). These associations were not seen for pre or post-natal enamel lead levels (data not shown).

With regards to hand-to-mouth behaviour that might increase lead exposure, six children had ever sucked their thumbs; six children chewed/sucked painted objects, put small toys/other objects into their mouth; and one child ate soil/dirt. None of these individual behaviours significantly influenced dentine lead levels, nor did 'any pica-like activity' influence dentine lead levels when assessed together.

Food was not commonly stored in pottery/ceramics with leaded glaze, or lead crystal/pewter (one positive response each). None of the parents knowingly stored food in lead soldered cans.

Parents were asked about specific occupations and activities that might result in exposure (including electronics, car repair, floor sanding, car battery service/repair/manufacture), and in total one third of the parents participated in one or more of these activities; none were associated with children's dentine lead levels.

Multivariable analyses

Multivariable linear regression was used to explore the associations between potential determinants of lead exposure and dentine lead levels. Using stepwise entry of variables (probability of F to enter ≤0.05, probability of F to remove ≥0.10), the only variable entered into the model was the binary variable 'grows fruit or vegetables in garden' (Table 2). When

the regression was re-run with pre- and post-natal enamel lead levels as the dependent variable, other variables were found to be significant predictors of these alternative measures of lead exposure (Table 2).

Table 2 here

Figure 2 shows a box plot of the distribution of dentine lead levels from the 4-6 ablation pits in dentine by child. The majority of children had mean dentine lead levels <0.5μg/g (an arbitrary cut off). Seven children (marked A-G) had dentine lead levels >0.5μg/g, and of these, two (B and C) lived with a household member involved with electronics, two (A and C) lived with a household member who worked in car repairs, and another child (D) lived with parents who stored food in pottery or ceramic with lead glaze. Four of these children (B, D, F and G) had a statistically greater spread of sampled dentine lead levels than other children (i.e. >2x the population mean SD of 0.38), and a further three children (H, I, and J) had mean dentine lead levels less than 0.5μg/g but again showed a greater statistical spread.

Figure 2 here

DISCUSSION

The Tooth Fairy study was conducted in 2005 in Newcastle upon Tyne, a city and region with a long history of lead smelting and mining, and therefore a wide range of possible environmental lead sources. We used dentine lead levels as a measure of long-term early life exposure, along with detailed home, lifestyle and socio-economic data to explore determinants of lead exposure in a cohort of children born since the removal of lead from petrol, paint and water. The mean dentine lead levels in this cohort were low, and no convincing associations with any of the anticipated predictor variables were found. Despite the low levels of exposure overall, the within and between child variability in dentine lead levels was suggestive of differing exposure levels and/or exposure sources across this population.

We found mean dentine lead levels to be higher than mean pre- and post-natal enamel lead levels, in keeping with several previous studies,[21, 24, 32] however others had found higher levels in enamel.[33] In some children we observed higher lead levels in enamel than dentine, which could be due to our inadvertent sampling of 'lead enriched' surface enamel; *in vivo* acid biopsies of the enamel surface reveal that the first few microns of enamel have anomalously high lead values.[26, 27]

The overall mean dentine lead level in this study in 2005 (0.26±0.16µg/g) was considerably lower than whole tooth lead levels from a UK study carried out more than twenty years earlier.[34] In this earlier study, Smith et al. analysed 2,564 whole teeth from 1,917 children attending infant schools in London. They reported lead levels in upper central incisors (30.1% of the teeth they collected) of 5.7±2.9µg/g and 5.6±2.9µg/g for right and left incisors respectively.[34] The difference between lead levels in this current and the earlier UK study

could be due to major methodological differences (e.g. the use of whole tooth versus dentine lead levels). They could also reflect the real decline in UK environmental lead contamination and concomitant drop in human exposure over the last few decades.[35] The mean dentine lead levels from this study were also lower than those reported for primary teeth of 48 children from non-polluted areas in South Africa (2.23±1.32µg/g),[32] and primary school children in Taipei (n=753, tooth lead levels 4.4±3.5µg/g) and Boston (n=2,331, tooth lead levels 3.3±2.5µg/g).[36]

Several predictor variables were found to be associated with dentine lead levels, including growing fruit or vegetables in the garden/allotment, daily tap water consumption, and methods of cleaning hard floors in the home. Although these associations were statistically significant in univariate analyses, they were not supported by similar associations with pre- or post-natal enamel lead levels, nor did the relationships hold in multivariable analysis. With >30 variables tested, one or two findings significant at the 5% level would be expected purely by chance, and the above associations may well reflect spurious, chance findings. What is perhaps more surprising than these few potentially spurious associations, is the lack of significant relationships, or even a trend suggestive of an association, with the various socioeconomic measures, variables which have been shown in earlier studies in the UK[15, 37] and elsewhere[38] to be predictive of lead exposure.

Hand to mouth behaviour is acknowledged as a potential pathway for the uptake of lead, especially in children living in areas with high soil lead levels. Newcastle upon Tyne remains affected by historic environmental lead contamination. A national survey conducted in 1981/82 analysed lead levels in household dust and garden soils and reported levels of ~600mg/kg and ~300mg/kg respectively from 100 homes in Newcastle.[39, 40] A subsequent

study of soil contamination in Newcastle in 2003 showed soil lead levels from 163 sampling locations ranged from 40 to 4,134mg/kg (mean=350mg/kg), with 27 sites exceeding the contemporaneous UK soil guideline value for residential areas and allotments of 450mg/kg.[41] Despite this continuing soil contamination issue, we did not find particularly high lead levels in components of teeth in this population, nor did we find the anticipated relationships with variables that might reflect exposure from contaminated soil as a source (e.g. dentine lead levels were lower in children from households that grew vegetables/fruits in their gardens). Low bioavailability of lead in soils in this area might explain the presence of high soil lead levels in combination with low tooth lead levels. Anthropogenic lead deposited in soil is likely more bioavailable than lead associated with natural soils.[42] If historic exposure was due to deposits of relatively bioavailable petrol-derived lead aerosols, the removal of this exposure source could have resulted in reduced exposure, despite total soil lead levels remaining high.

Despite the apparent low levels of lead exposure in this population, and lack of credible associations with the predictor variables, we did observe variability in dentine lead levels both within and between the recruited children. Although numbers were not sufficient to permit a statistical assessment, a narrative, contextual review suggests that some of the children with higher dentine lead levels and/or those children showing the greatest intra-tooth variability in dentine lead levels could have been exposed via parental occupations/activities with potential lead exposure, such as electronics and car repairs. These are plausible exposure pathways that warrant further study.

There are several limitations to this study that need to be considered. While this is a relatively large cohort of children with measures of lead in dentine and pre-and post-natal enamel, it is

nonetheless a relatively small sample within which to make meaningful statistical inference. Based on the initial power calculation we had aimed at recruiting at least 80 children from each quintile of deprivation in order to detect (with 90% power at the 5% significance level) a difference of 1.5µg/g in tooth lead between the least and most deprived quintiles. With only 69 respondents meeting the eligibility criteria the study lacked statistical power. It was therefore difficult to ascertain whether the lack of observed statistically significant associations was due to an absence of any true relationships, or a lack of statistical power to detect real associations where they exist. In addition, due to multiple testing, the relationships that we have observed could be due to chance, rather than a real, true result.

We had detailed information on important demographic and residential characteristics, and several relevant measures of socio-economic status, however a key variable that we were not able to include in our analysis was garden soil lead levels and/or indoor dust lead levels. Given the city's industrial past and known soil lead contamination issues, being able to account for contamination 'hot spots' that might confound the relation[43]ships between the other predictor variables and our measures of lead exposure would have been advantageous. Similarly, although we had detailed information on dietary behaviour, we did not collect detailed data on weaning or diet. Differences in the timing of the weaning process [43] the bioavailability of iron in the diet and/or iron deficient diets[11] could have influenced the relationships observed.

The sampling strategy for the Tooth Fairy study was informed by the current thinking at the time. The 4-6 dentine ablation pits per tooth were located randomly in the coronal dentine. Given the chronological development of dentine, the ablation pits represent inconsistently sampled 'snapshots' in time, and while the dentine lead levels tell us something about the

lead exposure experienced by each child during dentine mineralisation, the timeframe sampled potentially varies child by child, making interpretation difficult. Building on pioneering work by Humphrey and colleagues, we now know that a more refined sampling strategy, incorporating detailed dental histology, can enable more meaningful interpretation of exposure,[28, 29, 44, 45] and can, for example, be used to reconstruct the detailed chronology of early life lead exposure in children.[25]

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Table 1 Dentine lead levels by participant characteristics

Characteristic		**n**	**Mean dentine lead level (µg/g)**
Demographic characteristics			
Sex	Male	33	0.25
	Female	36	0.26
Age (years)	5	9	0.26
	6	32	0.24
	≥7	28	0.28
Adults (over 16) in residence (n)	1	8	0.31
	≥ 2	61	0.25
Children (≥5 yrs) in residence (n)	1	24	0.22
	2	35	0.27
	≥ 3	9	0.27
Socio-economic characteristics			
Highest education of main wage earner	Secondary	15	0.26
	College	13	0.23
	Higher Education	41	0.26
Household monthly income (£)	417-1,249	15	0.24
	1,250-2,083	21	0.28
	2,084-2,916	13	0.28
	≥2,917	20	0.22
Deprivation (IMD) score, home	≤10	30	0.23
	10.01-20.00	11	0.29
	20.01-30.00	10	0.30
	≥30.01	18	0.24
Deprivation (IMD) score, school	≤10.00	2	0.17
	10.01-20.00	34	0.25
	20.01-30.00	24	0.28
	≥30.01	9	0.23
Residence characteristics and renovations			
Home ever had lead water pipe	Yes	13	0.31
	No	22	0.23
	Not sure	31	0.25
Year house built	≤1930	19	0.26
	1931-1960	30	0.27
	≥1961	19	0.23
Condition of interior paintwork	Good condition	51	0.25
	Flaking/peeling	18	0.26
Renovation	Yes	57	0.26
	No	12	0.23
Old paint sanded/water blasted	Yes	30	0.28
	No	39	0.24
Old paint chemically stripped	Yes	7	0.32
	No	62	0.25
Ceiling/floor/wall demolished	Yes	26	0.26

	No	43	0.25
House cleaning			
Frequency of vacuuming	>Once per Week	44	0.24
	Weekly	19	0.29
	< Weekly	6	0.22
Hard floor in house	Yes	43	0.27
	No	26	0.23
Frequency of cleaning hard floor	>Once per Week	26	0.28
	Weekly	10	0.18
	< Weekly	7	0.34
Methods of cleaning hard floors	Dry cleaning	2	0.67 (p=0.001)
	Vacuum/mop	27	0.22
	Both	14	0.30
Dietary characteristics			
Grow fruit/vegetables in garden	Yes	17	0.19 (p=0.05)
	No	52	0.28
Eat fruit/vegetables grown in garden	Yes	13	0.19
	No	4	0.21
Daily tap water consumption	< 0.28L (1/2 pt)	14	0.34 (p=0.05)
	0.28-0.56L (1/2 - 1 pt	38	0.22
	>0.56L (1 pt)	17	0.26
Child ever bottle fed	Yes	58	0.26
	No	11	0.23
Child bottle fed at age	<1 month	19	0.27
	1-12 months	19	0.28
	≥13 months	20	0.23
	Never	11	0.23
Hand-to-mouth behaviours, health and medicines			
Sucks thumb	Yes, ever	6	0.27
	No, never	63	0.25
Chews/sucks objects/toys	Yes, ever	6	0.30
	No, never	63	0.25
Medical conditions	Yes	9	0.31
	No	60	0.25
Has used traditional remedies	Yes	8	0.26
	No	61	0.25
Smoker in residence	Yes	13	0.27
	No	54	0.25
Parental occupations/activities with potential lead exposure			
Car repair	Yes	10	0.26
	No	59	0.25
Electronics	Yes	11	0.27
	No	58	0.25
Car battery service/repair	Yes	3	0.26
	No	66	0.25
Floor sanding	Yes	5	0.21
	No	64	0.26

Table 2: Multivariable linear regression for predictors of dentine, pre-natal enamel and post-natal enamel lead levels.

Dependent variable	**Predictor variables**[1]	**B (95% CI)**	**p**	**Model R^2**
Dentine lead level	Constant	0.282 (0.238, 0.326)	0.000	0.089
	Grows fruit or vegetables in garden	-0.113 (-0.206, -0.021)	0.017	
Pre-natal enamel lead level	Constant	0.122 (0.047, 0.197)	0.002	0.223
	Paint chemically stripped	0.106 (0.035, 0.176)	0.004	
	IMD score, home	0.025 (0.008, 0.041)	0.004	
	Tap water consumption	-0.032 (-0.063, -0.001)	0.044	
Post-natal enamel lead level	Constant	0.005 (-0.080, 0.090)	0.905	0.153
	Child sucks painted objects	0.154 (0.040, 0.269)	0.040	
	Child ever bottle fed	0.096 (0.006, 0.187)	0.006	

[1] Predictor variables available for inclusion in the model: Sex (M/F); age (categorical); home ever had lead water pipe (no/yes/don't know); tap water consumption (categorical); grows fruit or vegetables in garden (no/yes); period house built (categorical); highest education of the main wage earner (categorical); IMD score of main residence (categorical); IMD score of school (categorical); household monthly income (categorical); make-up of household (adults over 16 in residence (categorical), children ≥5 in residence (categorical)); condition of interior paintwork (categorical); home renovations (paint sanded/water-blasted (no/yes), paint chemically stripped (no/yes), ceiling/floor/wall demolished (no/yes)); frequency of vacuuming (categorical); hard floors in house (no/yes); child ever bottle fed (no/yes); pica-like activity (sucked: thumb (never/ever), painted objects (never/ever), puts small toys in mouth (never/ever)); food storage (in pottery (no/yes/don't know), crystal or pewter (no/yes/don't know), lead soldered cans (no/yes/don't know)); medical conditions (no/yes); use of traditional remedies (no/yes); sibling with elevated blood lead level (no/not sure); activities with potential for lead exposure (car repair (no/yes), lead window making (no/yes), electronics (no/yes), ammunition handing (no/yes), floor sanding (no/yes)); household member smokes (no/yes). Note, where variables are listed as 'categorical', the categories are the same as those shown in table 1.

Figure 1: Mean pre and post-natal enamel and dentine lead levels in children recruited in the Tooth Fairy Study.

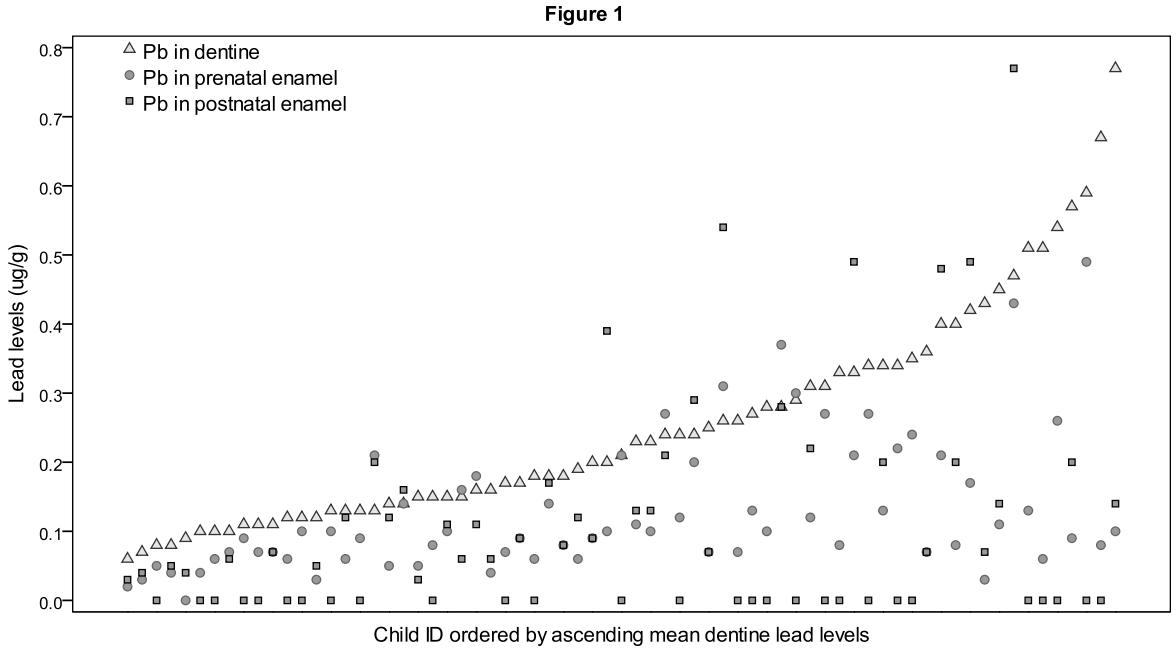

Figure 2: Box plot showing the variability in dentine lead levels by child (The bar represents the median dentine lead levels, the box shows the 25th and 75th centiles, whiskers are 1.5 times the interquartile range, and outliers and extreme outlets are shown by circles and asterisks' respectively).