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Relating Tooth and Blood Lead Levels in Children

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Lead concentrations in shed teeth have found increasing utility in research studies of lead exposure and child development. Teeth are useful because they record lead levels and are easily collected. However, in considering internal doses of lead, most of what has been learned about human lead toxicity and kinetics has been expressed in terms of blood lead concentrations. For example, a computerized literature search found "blood lead" as a key word in 1,035 articles cited between January and October 1994. Only 9 articles were found for "tooth lead". Because of the advantages of using teeth to assess lead exposure, the relation between teeth and blood lead levels deserves more attention.

Teeth are anatomically more complex then blood, and lead in a tooth resides in one of several metabolic pools (Jones et al. 1992). For a shed tooth, nearly all of the mass is the crown, the root having been resorbed. Within that crown, nearly all of the mass is dentine. So values obtained from samples of whole shed teeth, of just the crown, or of only the dentine would be approximately equivalent. Although concentrations in the most surficial enamel may be relatively elevated, its mass is very small and its contribution to the whole tooth's lead levels is negligible. However, regarding the circumpulpal dentine, much higher concentrations are encountered (Hanson et al. 1989). So studies which use that zone of the tooth are not directly comparable to those which use the bulk of the crown.

Several studies have already attempted to look at their own blood and tooth data to find the strength and slope of the relationship (Rabinowitz et al. 1989). Recently, with the availability of additional studies, it is possible to reconsider this question across a variety of locations and exposure levels, in hopes of finding more general relationships.

MATERIALS AND METHODS

An extensive review of published literature yielded only 8 studies which report tooth lead and blood lead levels on the same children. Different investigators have used different portions of teeth for their lead measurements. Here, only studies which reported dentine,whole shed teeth, or crown are used here. Values of circumpulal dentine are much higher and not directly comparable. Both cross section and prospective study designs are included.

These studies are shown in Table 1. The locations include Boston during the 1970's (Needleman at al 1979) and the 1980's (Rabinowitz et al. 1989), London (Smith et al. 1983), Germany (Winneke et al. 1983), Scotland (Fulton et al. 1987 and 1989) Italy (Bergomi et al. 1989), Taiwan (Rabinowitz et al. 1991), and Australia (MacMichael et al. 1994). Their mean tooth lead levels range from under 3 to over 12 ug/g.

RESULTS AND DISCUSSION

Overall there is considerable consistency among these different studies, despite different populations, different laboratories and somewhat different portions of tooth used. A straight line fits the data from these eight different studies very well. This best fit line may be further constrained to pass through the origin. This is justifiable because we know that if there were no lead in the blood there would be no lead in the teeth.

The equation for that straight line is: Tooth (ug/g) = 0.49 (SE=0.04) X Blood (ug/dl). The fit is very good: r-sqr is 96.5 percent, F ratio is 195.4, P<.0001, n=8. Even if the line were not constrained to pass through the origin, it still does, yielding a nearly identical line: Tooth = 0.8 (1.5) + 0.44 (0.10) X Blood.

This overall slope as determined across these eight studies is somewhat steeper than the slope we found using only the Boston children, who have a relatively low level of exposure: 0.36 (.02), with an r-sqr of 73 percent for 99 cases using blood collected at 57 months of age.

At exactly what age is the blood lead best preserved in the shed tooth has received some attention (Rabinowitz et al. 1993). In many cases, where the blood lead levels of a child do not change with age, this becomes a mute point. In cases where postnatal blood lead levels do change, it appears that the shed dentine reflects blood lead levels fairly recently, as opposed to many years earlier. For example the blood at age 57 months correlates much better with a shed incisor than does cord blood levels. For nearly all of the studies cited, the blood values were obtained only once.

A recent compendium of lead and IQ studies has tried to use both studies that relied on blood lead and those that relied on tooth lead (Pocock et al. 1994). It was necessary to perform parallel analyses for both types of biomarkers and try to make them

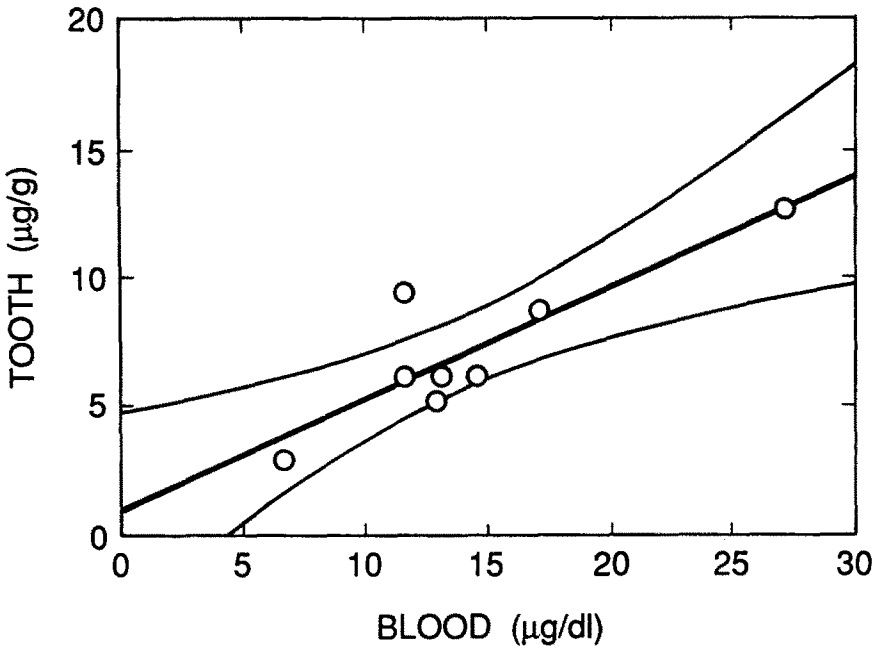

Figure 1. Scatter plots and best linear fits of shed tooth lead and blood lead from eight different studies. The best fitting line passes through the origin with a slope of 0.49.

Table 1. Summary of tooth and blood lead data from several studies of children.

Location	Mean Tooth Lead ug/g	Mean Blood Lead ug/dL
Boston 1970's, USA	12.7	27
Edinburgh, Scotland	9.3	11.5
Port Pirie, Australia	8.6	17
Dusseldorf, Germany	6.2	14.3
School by smelter, Taiwan	6.2	13
London, England	5.1	12.8
Sassuolo, Italy	6.1	11.6
Boston 1980's, USA	2.8	6.5

comparable by insisting that a doubling of dose in one is equivalent to a doubling of dose with the other biomarker. The approach taken here is different, since only studies are considered which report both blood lead and tooth lead values.

The kinetics of lead in adult teeth are very different from those of primary teeth (Steenhout and Pourtois 1981). Lead accumulates in adult, secondary teeth for many years. What has been said here about primary teeth likely do not relate at all to permanent teeth.

Figure 1 shows the mean values of blood and tooth lead concentrations in these eight studies. A straight line passing through the origin gives an excellent fit to these observations. This summary finding serves to demonstrate the usefulness of shed teeth as a biomarker of past lead exposure.

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