

Health of Infants in an Imperial Roman Skeletal Sample: Perspective from Dental Microstructure

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ABSTRACT This study examines general health in the first year of life of a population of 127 subadults from the Imperial Roman necropolis of Isola Sacra (2nd–3rd century ACE). Health status was determined by analyzing 274 deciduous teeth from these children for Wilson bands (also known as accentuated striae), microscopic defects caused by a disruption to normal enamel development arising from some generalized external stressor. While macroscopic enamel defects, or hypoplasias, have long been used as proxies of general population health, we believe that this is the first population-wide study of microscopic defects in deciduous teeth. We used micro-

structural markers of enamel to attach very precise chronologies to Wilson band formation that allowed us to calculate maximum prevalence (MAP) and smoothed maximum prevalence (SMAP) distributions to portray what we believe to be a realistic risk profile for a past population of children. There appear to be two periods of high prevalence, the first beginning around age 2 months and continuing through month 5, and the second higher period beginning around month 6 and continuing through month 9. These results are discussed in light of historical records of Roman childhood rearing practices. *Am J Phys Anthropol* 130:179–189, 2006. © 2005 Wiley-Liss, Inc.

The mineralized enamel of tooth crowns preserves the history of an individual's early life and associated physiological events. Defects of enamel structure or enamel hypoplasias appear on the crown surface (in a continuum from the macroscopic to microscopic level) as pitted, furrowed, or plane form defects (Hillson and Bond, 1997). Internally, where they can only be seen microscopically, enamel defects are expressed as prominent or "accentuated" brown striae of Retzius, also called Wilson bands (WB). Enamel defects are the result of developmental disturbances affecting the production of enamel matrix (Boyde, 1989; FitzGerald and Rose, 2000; FitzGerald and Saunders, 2005). An extensive literature records the existence of (mainly macroscopic surface) defects in living children and archaeological samples of human remains. Most research concludes that defects are epidemiological markers of nonspecific stress that can be used to judge the health status of past and present populations.

The nonspecificity of defect formation may be viewed as either problematic or beneficial. While perhaps problematic for clinical studies of causation, the fact that the majority of defects are produced by environmental disruptions (e.g., fevers, infections, dietary deficiencies) led anthropological researchers to use their prevalence in various populations as a generalized indicator of adaptive responsiveness. In addition, the episodic nature of the formation of defects means that there is a possibility of establishing their timing and duration within the crown and thereby associating chronologies of defect formation with specific events in an individual's life history (Skinner and Anderson, 1991; Lukacs and Hemphill, 1991; Li et al., 1995; Lukacs et al., 2001), or with temporal periods of increased health risk (e.g., weanling diar-

rhea) (Goodman et al., 1984; Hutchinson and Larsen, 1988, 1990; Moggi-Cecchi et al., 1994; Ensor and Irish, 1995; Bermúdez de Castro and Perez, 1995; Taji et al., 2000; but see Saunders and Barrans, 1999; King et al., 2002). The common procedure is to establish timing of defect formation using positional measurements from the cement-enamel junction of the crown to the cervical and occlusal margins of each defect, converted into ages of formation using standardized tables (Goodman and Rose, 1990) or the duration of growth disruption as a proportion of crown height.

There are a number of problems with these techniques for estimating the timing of defects. An underlying assumption is that enamel growth occurs in a linear fashion, but this was shown not to be the case in permanent teeth (Beynon et al., 1991; Beynon and Reid, 1987; FitzGerald, 1995; Hillson and Bond, 1997; Reid and Dean, 2000; but see Goodman and Song, 1999). Enamel rates vary through the crown in deciduous teeth too, although not to the same degree as in permanent teeth

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(FitzGerald and Saunders, 2005; Shellis, 1984). There are also concerns associated with the appropriateness of applying modern crown completion standards to archaeological populations (FitzGerald and Saunders, in press).

In this study, we look at enamel defects (WB) as they appear microscopically in the enamel mantle of deciduous teeth. We determine precise ages of formation and duration of defects based on methods of evaluating crown development events from incremental growth markers. These use information endogenous to the tooth (FitzGerald and Rose, 2000; FitzGerald and Saunders, 2005), and do not have to rely on any exogenously derived standards.

Deciduous teeth begin developing early in fetal life, at approximately 13–16 weeks postfertilization. Crown formation of the second maxillary deciduous molars, the last to develop, is complete at about 11 months after birth (Lunt and Law, 1974). It is crucial to study this period of life because the high risk of death among the very young is a long-recognized demographic marker of a population's accommodation to its living conditions. High death rates in early life (especially the first year of life or infancy) are identified as common for all but the most industrialized countries (Wrigley, 1969; Knoedel, 1983; Lancaster, 1990). In addition, a mother's health and nutrition during pregnancy are known to be significant factors affecting the growth and survival of the developing fetus, while the period from birth to about age 3 years is the most crucial for proper growth of the child (Beaton, 1992). If death is avoided, adverse factors can still produce significant growth retardation or alteration in the first few years because growth rates are highest during this period. While skeletal samples are mortality samples, thus representing biased windows onto any of the interactions the living population might have had with its environment (Wood et al., 1992; Milner et al., 2000; Saunders and Hoppa, 2003), dental defects, by their very presence, represent episodic morbidity events that were survived, and which can be examined as serial markers of physiological stress.

In addition, all deciduous teeth develop perinatally, so that they all possess neonatal lines. This means that the ages of developmental events can be accurately estimated based on a single tooth in contrast to the permanent dentition, in which only M1 shows a neonatal line. As a result, large-scale population assessments can be carried out more easily and more exactly in the deciduous than in the permanent dentition.

While there are several previous histological examinations of individuals with macroscopically observable hypoplasias on their deciduous teeth (Fearne et al., 1994; Ranggard et al., 1994, 1995), the focus of these studies was on individual cases, and observations generally did not examine associated abnormal striae of Retzius. We are unaware of any population-based studies of Wilson bands in deciduous teeth; generally, macroscopic surface prevalence studies in deciduous teeth are not abundant (Sweeney and Guzman, 1966; Sweeney et al., 1969, 1971; Enwonwu, 1973; Infante, 1974; Holm and Arvidsson, 1974; Seow et al., 1984; Nation et al., 1987; Ishida et al., 1990; Weeks et al., 1993; Li et al., 1995, 1996; Kanchanakamol et al., 1996; Sheiham, 2003), although some non-prevalence studies in archaeological samples exist (Sciulli, 1977; Cook and Bulkestra, 1979; Corruccini et al., 1985; Blakey and Armelagos, 1985; Yamamoto, 1989; Lovell and Whyte, 1999; Lukacs, 1999).

The purpose of the present study is to document and evaluate the prevalence of Wilson bands by age in the

first year of life in the deciduous dentition of a large sample of skeletons from an Imperial Roman necropolis. Located on the western coast of Italy, approximately 23 km west of Rome, the necropolis of Isola Sacra served as the cemetery for the inhabitants of *Portus Romae* from the 1st–3rd centuries AD. *Portus Romae* was an important trading center and site of extensive warehouses holding grain to feed the population of Rome. The populace of *Portus* included middle-class administrators, traders, and merchants (Mannucci and Verduchi, 1996; Macchiarelli and Bondioli, 2000), but inscriptional evidence does not refer to a local aristocracy, unlike other Roman towns from the Imperial period (Garnsey, 1999). Individuals in this study come from in-ground burials or burial structures between monumental tombs (Baldassarre, 1990; Macchiarelli and Bondioli, 2000).

The total number of individuals in the skeletal sample is estimated to be around 2,000 (Sperduti, 1995; cited in Rossi et al., 1999; Macchiarelli and Bondioli, 2000). Many of these are commingled remains from excavations in the earlier part of the 20th century but more than 800 skeletons were individually catalogued and analyzed. Of these, 334 were identified as infants, children, or adolescents. Infants, or those estimated to be less than age 1 year, represent approximately 20% of the subadult sample.

MATERIALS

In this study, we examined 274 teeth from 127 subadults, i.e., the majority of the cemetery sample from Isola Sacra with teeth that could be sectioned.

The estimated dental age at death of individuals in the sample, determined using conventional dental and skeletal standards, ranged from birth to about 13 years, with a mean age of 4.2 years.

METHODS

Microstructural analysis

Microstructural analysis involved preparing slides for examination under a microscope, observing the enamel microstructures, and deriving the chronology of tooth development, thus allowing ages of formation to be assigned to Wilson bands. Although no studies utilizing this methodology on deciduous teeth have been published, some using enamel defects in permanent teeth have (e.g., Hillson, 1992; Hillson et al., 1999). However, none of these studies specifically determined the ages of Wilson bands.

The criterion used to identify Wilson bands was of necessity a very broad and minimalist one. As some of us have argued elsewhere (FitzGerald and Saunders, 2005), Wilson bands are similar in etiology and appearance to regular brown striae of Retzius. This means that the atypicality of prism structure and degree of accentuation of stria, which are commonly used by other workers, are not valid discriminators. In this study, Wilson bands were only recognized as such if the stria was visible for at least 75% of its length from the EDJ to the crown surface in the imbricational area of teeth, or in the cuspal area, if the stria was visible for at least 75% of the total distance from the buccal to labial sides of the enamel dentine junction (EDJ) around the dentine horn (under the cusp tip, striae do not crop out at the surface, but form what Hillson (1996) called "caps," which in two dimensions on a microscope slide appear as elongated

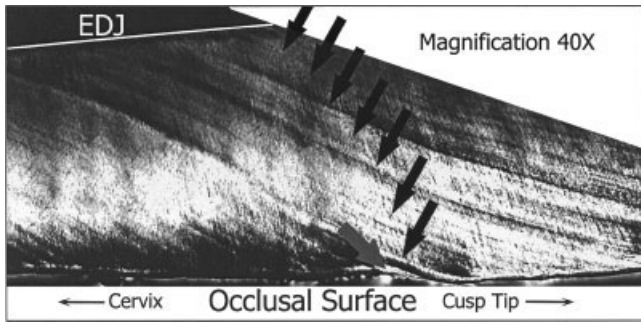


Fig. 1. Photomicrograph of portion of buccal imbricational enamel at $\times 40$ magnification. Seven black arrows indicate accentuated striae identified as Wilson bands because they conform to minimal definition of visibility for 75% of distance between EDJ (at top) and occlusal surface (at bottom). Note their association with obvious hypoplastic deformation (gray arrow). Note also several accentuated striae not classified as Wilson bands because they fail test of discernibility.

arcs stretching from one side of the dentine horn to the other). Figure 1 identifies Wilson bands on a tooth with many striae (note that this is a permanent tooth from a subadult at Isola Sacra, chosen because it demonstrates the problems of identification so well). It is also important to note that Wilson bands were recorded wherever they could be discerned in a crown. Although Hillson and Bond (1997) suggested that an enamel defect should only be confirmed if it can be recorded in all growing teeth in a dentition, we find this to be too exclusive a definition. In archaeological samples (where taphonomic damage is common and complete dentitions are not), it is not possible to lay down strict criteria, such as confining observations only to the bucco-labial side of a tooth or recording Wilson bands only if they can be matched in at least two teeth in a dentition. Wilson bands in our study were recognized on an individual crown basis and without regard to tooth region or location.

Conventional methods for thin-section preparation were followed (Rossi et al., 1999) to produce one or two longitudinal bucco-lingual sections, approximately 70–150 μm thick, taken from the midsection of each tooth. Many teeth had two or more sections cut from them. Only one slide from each tooth, showing the best discernibility of microstructures and least diagenetic damage, was selected for analysis. All specimens were observed under polarized light, and images were captured with a Polaroid DMC digital video camera attached to the microscope and exported into Adobe Photoshop, which was used to assemble montages of relevant areas of tooth sections from adjacent images. Measurements, counts, and other data were captured from these montage images using SigmaScan Pro software from SPSS, Inc.

FitzGerald and Saunders (2005) gave a full account of the protocol for assigning the age of formation to a Wilson band, so we will only briefly summarize it here. The methodology first required the identification of the neonatal line, which was used to set time calculations to zero (or birth). A photomicrograph that included both Wilson band and neonatal line (Fig. 2) was taken, or a montage of photomicrographs was assembled if the Wilson band and neonatal line were too far apart to appear in the microscope camera's field of view. A prism running between these two structures was traced on the photomicrograph (or montage), and cross striations (the alternat-

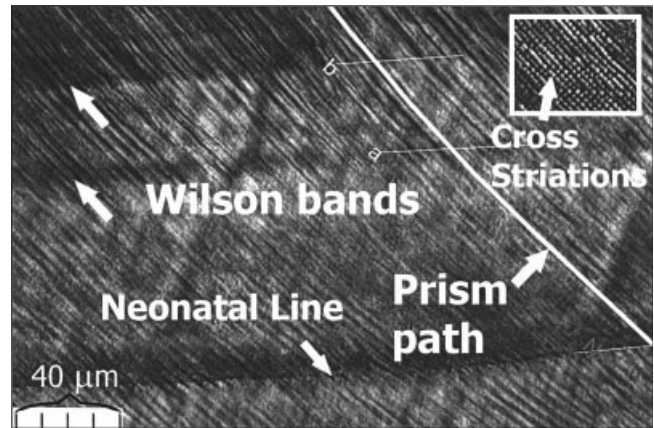


Fig. 2. Cross section of deciduous enamel from specimen from Isola Sacra, showing microstructural features discussed in text: neonatal line, Wilson bands (two in this photomicrograph), prisms (with one apparent prism path traced in white), and because cross striations are not clear in this view, an inset photo from another tooth section with cross striations, consisting of alternating light plus dark bands, clearly visible along its prisms.

ing light and dark bands representing daily appositional enamel growth) were counted along the prism where they were discernible. In areas where they were difficult to see, the average cross-striation repeat interval (i.e., the distance between two adjacent cross striations) was determined by measuring groups of cross striations and then taking the mean of these groups. The length of the traced prism path was also measured where cross striations could not be counted, and the distance was divided by the average cross-striation repeat interval to yield the time in days taken to develop this portion of the prism. By counting and measuring in this way, the chronology of prism formation between birth and Wilson band formation was built up.

Adjustment of crude prevalence data

Prevalence is defined in the following way (Waldron, 1994):

$$\text{Prevalence} = \frac{\text{Number of cases of a condition}}{\text{Total population}} \quad (1)$$

Before prevalence could be calculated in our sample, it was necessary to adjust the raw data in order to arrive at the correct numerator and denominator of the prevalence fraction. Looking first at the numerator, in those children who had contributed two teeth with Wilson bands to the sample (and none had contributed more than this¹), the tooth with the greater number of Wilson bands (of the two teeth) was used to represent the number of stress events for these children, thus ensuring that stress events were not overcounted. These were then added to the total number of Wilson bands for children with one tooth each in the sample, to arrive at the prevalence numerator.

¹Not all 127 individuals (some with more than two teeth in the sample) showed evidence of Wilson bands. No individual who did show Wilson bands contributed more than two teeth to the sample.

TABLE 1. Assumed average age of deciduous crown completion based on 13 teeth from Isola Sacra population

Tooth type (maxillary and mandibular combined)	Number of teeth used to determine crown completion	Age of crown completion (postnatal months)
Central incisor	1	5
Lateral incisor	1	6
Canine	6	12
First molar	4	12
Second molar	1	13

The adjustment of the denominator, i.e., the total population at risk, was more complex. In paleoepidemiological populations, for conditions not resulting directly in death, the dead population may be considered to be a reasonable proxy of the living one when calculating the prevalence statistic (Waldron, 1994, 1996), although this view is not accepted by everyone (Wood et al., 1992; Milner et al., 2000; Saunders and Hoppa, 2003); we will have more to say about this assumption below. In our study, the size of the population at risk changed through the whole period in which teeth were recording Wilson bands because developmental defects are only registered in growing crowns. Since crown formation times vary by tooth type, the upper age limit at which stress will cease to be recognized will therefore also vary by tooth type (e.g., on average, crown formation times for deciduous canines are longer than those for deciduous central incisors). This means that the mix of tooth types in the sample will be an important consideration when arriving at the “total [age-specific] population at risk.” In addition, if a child died before completing deciduous crown growth, the population would also need to be corrected to reflect this. Therefore, two separate adjustments were made to arrive at the denominator:

1. The population was adjusted to take account of premature death. All teeth in this sample came from specimens that had been individually catalogued and analyzed, with age at death estimated by dental formation and skeletal indicators. The age-specific population at risk was therefore adjusted for those children who left the sample through premature death, using the lowest boundary of the estimated age range. This involved listing all teeth in the sample and then counting the number of “lived months” for each specimen during the period of crown growth. The total numbers of lived months then represented the age-specific population in each 1-month interval.
2. The population was also adjusted to take into account differing ages of crown maturity among tooth types. This was more difficult to control than the premature death of individuals, since there are two sources of variability. The first is within-population (individual) variability of crown development times. The only way to completely control for this would be to assess crown formation times for every tooth in the sample. Although this is theoretically possible using microstructural analysis, in archaeological samples it will never be attainable because of the difficulties involved: 1) deciduous teeth have no regular prenatal striae of Retzius, 2) deciduous microstructures are often faint and difficult to discern clearly in both pre- and postnatal enamel, and 3) diagenetic tissue deterio-

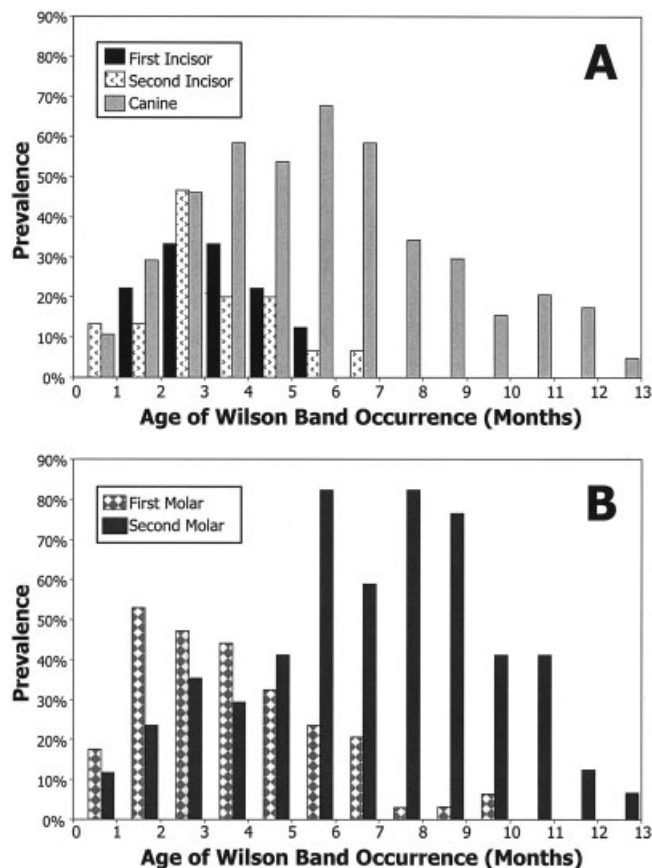


Fig. 3. Wilson band formation by tooth type. A: Anterior deciduous teeth. B: Deciduous molars.

ration occurs over time. All of these make determining a complete record of crown development for every tooth in the sample impossible. A second confounding factor arises from between-population variability of crown formation times. Although the published crown formation standards for deciduous teeth are not highly reliable (Hoppa and FitzGerald, 1999), this is likely less a concern than the application of modern standards from well-nourished Western populations to a population almost two millennia old (FitzGerald and Saunders, 2005). In the absence of appropriate standards, we developed our own (Table 1), based on crown formation times estimated for 13 Isola Sacra deciduous teeth (FitzGerald et al., 1999). Although this is not a large sample, we believe that it provides a better basis for correction than the published alternatives. The adjustment was made by assuming that the crowns of teeth of a particular tooth type all reached maturity at the average age determined for that tooth type. The list of lived months by specimen that had been adjusted for premature death (see above) was now further adjusted to yield a corrected total by month.

Although these adjustments will compensate for many of the problems associated with varying crown formation times and children prematurely exiting the sample, another more important concern still needs to be recognized. Figure 3 illustrates the problem. It shows the prevalence of Wilson bands by month by deciduous tooth

type, given as two graphs to make interpretation clearer. It can be seen that the prevalence distribution of each tooth class has a shape that resembles a bell-shaped normal (or Gaussian) curve, with the highest frequencies in the middle months, bordered by smaller, balanced tails on either side. These “normal” curves do not appear to be linked in any way to a “real” pattern of stress events, since it is possible in any month to have some tooth types recording relatively high frequencies of Wilson bands (for that tooth type), while others in the same month are recording relatively low frequencies (for that tooth type). If this phenomenon truly reflects what is happening during tooth development, the most plausible explanation is that “sensitivity” to stress events varies in some way through the development cycle of the tooth, being lowest early in cuspal development, as well as later toward the cervix, and highest in the middle period of tooth development. Note that this is not necessarily located in the physical middle of the EDJ or halfway down the crown, but instead in the middle of crown development time. With rates of extension, cuspal enamel thickness, and crown geometry varying by tooth class, this means that the midpoint location will also differ by tooth class (e.g., FitzGerald and Rose, 2000; Antoine, 2000; Reid et al., 2000; Dean and Reid, 2001). There is an alternative explanation that may also account for the shape of the prevalence distribution by tooth type, i.e., that it is an artifact of our ability to detect Wilson bands. It may be that Wilson bands are more difficult to discern in buried cuspal enamel, or in the very thin enamel toward the cervix.

Whichever of these explanations is correct, we need to recognize and compensate for this phenomenon. To do so, we calculated the maximum prevalence (MAP) of Wilson bands in each month. This consists of the adjusted prevalence observed for the tooth type with the highest frequency of Wilson bands in any month. Choosing either the “most-sensitive-to-stress” tooth type or the tooth type with most easily discernible Wilson bands will ensure that the maximum number of population-wide stress events is recognized.

Although MAP takes us much closer to envisaging a prevalence distribution, we realize that because of possible sampling bias in our cemetery population as well as other factors including artifacts of analysis (such as forcing continuous events into monthly classes), our MAP distribution was likely unrealistically “spiky.” Therefore, we calculated smoothed maximum prevalence (SMAP), the maximum prevalence distribution drawn as a smoothed curve. This curve was based on a trend line produced using higher-order (sixth) polynomial regression, with the resultant line manually modified to eliminate, for instance, early and late kinks. We believe that SMAP represents a closer approximation of the shape of the “true” prevalence distribution curve for the children in this Imperial Roman population as expressed in their enamel crowns.

RESULTS

We looked for Wilson bands in 274 deciduous teeth from 127 subadults. Fifty individuals, or almost 40%, were identified as having one or more Wilson bands in at least one of their teeth. A summary of some key raw results is shown in Table 2, summarizing the calculations of Wilson band frequencies by individual, by stress event, and by tooth.

TABLE 2. Summary of key results

Basis of calculation	Parameter	Result
By individual	Number in sample	127
	Number with WB	50
	Percentage affected with at least one WB	39.4%
By stress event (net number of Wilson Bands) ¹	Number of WB	447
	Number of teeth affected by WB	64
	Average number of WB per affected tooth	7.0
By tooth	Total number of teeth examined	274
	Number of teeth affected by WB	64
	% teeth affected with at least one WB	23.4%

¹ Net number of WB (Wilson bands) for a child with two teeth in sample reflects number of stress events suffered by individual (i.e., adjusted for coeval events in two teeth; see text for full explanation).

Table 3 shows the total number of teeth analyzed, apportioned between those affected by at least one Wilson band and those showing no Wilson bands, and additionally broken down by tooth type and jaw. It can be seen that canines were by far the most common tooth type in the sample, representing about 47% of the total number of teeth analyzed in the study. This can also be seen in the bar chart of Figure 3, which additionally highlights the fact that Wilson bands were seen in only about 26% of canines. Nonetheless, because canines are such a significant proportion of the total sample, more Wilson bands were found in them than in any other tooth type (about 52%, as shown in Table 3). Wilson bands were also about as likely to be seen in second molars (28%, from Fig. 4) as in canines, although there were far fewer of them in the sample than canines (about 14%, as shown in Table 3). However, there is no statistically significant relationship between the likelihood of seeing a Wilson band and a particular tooth type ($\chi^2 = 3.242, P = 0.518$). As also seen in Table 3, although there are some differences between the likelihood of observing a Wilson band in a particular jaw, none of these differences, either within individual tooth types or overall, are significantly different ($\chi^2 = 0.088, P = 0.767$).

Figure 5 graphs the crude (unadjusted) Wilson band frequency by age, shown in semimonthly classes. The number of Wilson bands contributed by those children with two teeth in the sample was adjusted by taking out coeval Wilson bands, ensuring that no double counting of stress events occurred, and that only the actual number experienced by each child is shown. It can be seen that the number of stress events increases substantially in the first half of month 2 and reaches a maximum in the second half of month 5, trending downward to a low of two events in the first half of month 12.

Figure 6 illustrates prevalence distributions of teeth in the sample (i.e., number of teeth with at least one Wilson band). One set of bars shows the adjusted epidemiological prevalence. This is determined by adjusting the crude prevalence denominator, as discussed in Methods, so that each monthly population reflects the number of forming crowns in that month. Tooth crowns maturing and no longer capable of registering Wilson bands, or children dying before complete crown maturation and no

TABLE 3. Total number of teeth analyzed, broken down between those affected by at least one Wilson band and those with no Wilson bands, by jaw and tooth type

	Maxillary			Mandibular			Total		Grand total
	WB observed		Total	WB observed		Total	WB observed		
	No	Yes		No	Yes		No	Yes	
Central incisor									
Count	9.0	2.0	11.0	7.0	0.0	7.0	16.0	2.0	18.0
% within WB observed	11.5	9.1	11.0	5.3	0.0	4.0	7.6	3.2	6.6
% within tooth type	81.8	18.2	100.0	100.0	0.0	100.0	88.9	11.1	100.0
% of total	9.0	2.0	11.0	4.0	0.0	4.0	7.6	3.2	6.6
Lateral incisor									
Count	6.0	2.0	8.0	17.0	3.0	20.0	23.0	5.0	28.0
% within WB observed	7.7	9.1	8.0	12.8	7.3	11.5	10.9	7.9	10.2
% within tooth type	75.0	25.0	100.0	85.0	15.0	100.0	82.1	17.9	100.0
% of total	6.0	2.0	8.0	9.8	1.7	11.5	10.9	7.9	10.2
Canine									
Count	34.0	12.0	46.0	62.0	21.0	83.0	96.0	33.0	129.0
% within WB observed	43.6	54.5	46.0	46.6	51.2	47.7	45.5	52.4	47.1
% within tooth type	73.9	26.1	100.0	74.7	25.3	100.0	74.4	25.6	100.0
% of total	34.0	12.0	46.0	35.6	12.1	47.7	45.5	52.4	47.1
First molar									
Count	18.0	4.0	22.0	30.0	8.0	38.0	48.0	12.0	60.0
% within WB observed	23.1	18.2	22.0	22.6	19.5	21.8	22.7	19.0	21.9
% within tooth type	81.8	18.2	100.0	78.9	21.1	100.0	80.0	20.0	100.0
% of total	18.0	4.0	22.0	17.2	4.6	21.8	22.7	19.0	21.9
Second molar									
Count	11.0	2.0	13.0	17.0	9.0	26.0	28.0	11.0	39.0
% within WB observed	14.1	9.1	13.0	12.8	22.0	14.9	13.3	17.5	14.2
% within tooth type	84.6	15.4	100.0	65.4	34.6	100.0	71.8	28.2	100.0
% of total	11.0	2.0	13.0	9.8	5.2	14.9	13.3	17.5	14.2
Total									
Count	78.0	22.0	100.0	133.0	41.0	174.0	211.0	63.0	274.0
% within WB observed	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
% within tooth type	78.0	22.0	100.0	76.4	23.6	100.0	77.0	23.0	100.0
% of total	78.0	22.0	100.0	76.4	23.6	100.0	100.0	100.0	100.0

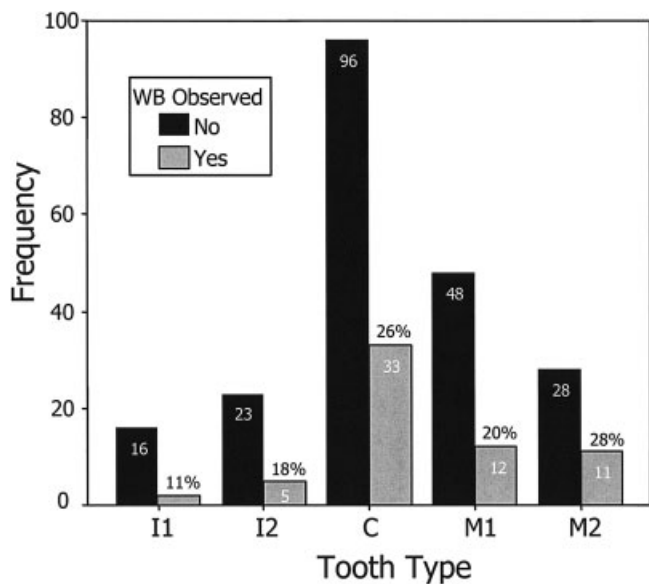


Fig. 4. Number of teeth analyzed in sample by tooth type, split between those where at least one Wilson band was observed and those showing no Wilson bands. Numbers within bars are actual count frequencies; those above bars represent percentages within each tooth type where Wilson bands were seen.

longer contributing teeth to the sample, were removed. These age-specific populations represent only those teeth at risk of recording a morbidity or stress event.

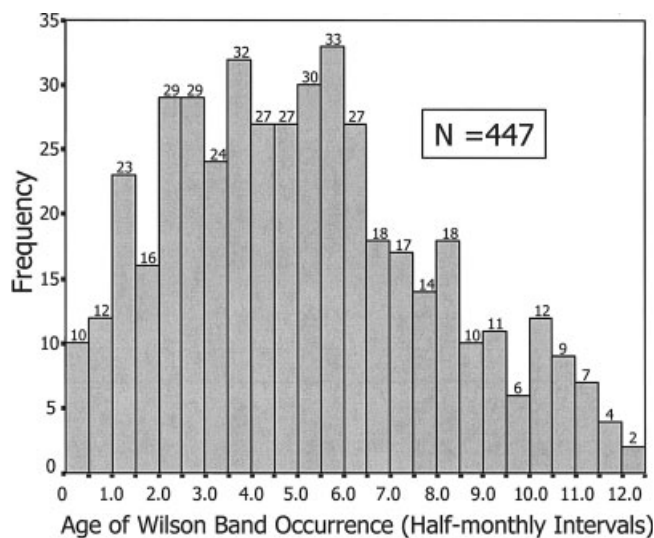


Fig. 5. Crude (unadjusted) count of Wilson bands observed in semimonthly intervals.

The second set of bars in Figure 6 shows the MAP, i.e., the maximum prevalence among all tooth types for each month, representing the greatest likelihood of experiencing stress events in the *Portus Romae* population in any month. It is evident from Figure 6 that MAP is significantly higher in all months than the overall adjusted prevalence.

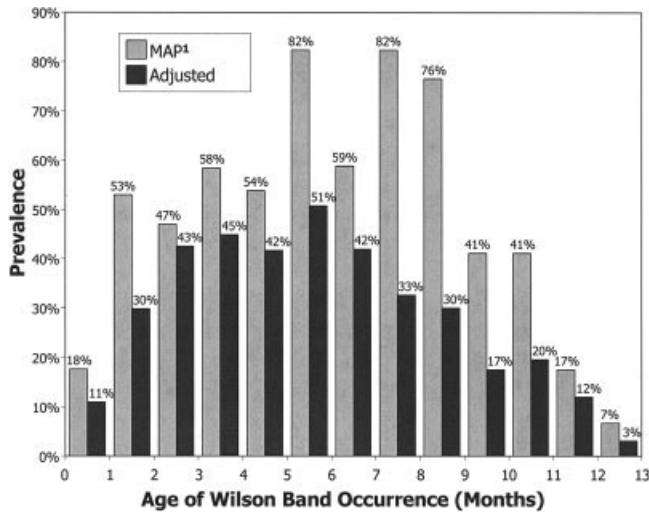


Fig. 6. Prevalences of Wilson bands by tooth: 1) adjusted epidemiological distribution (raw data were corrected to reflect number of teeth with forming crowns in each month); 2) maximum prevalence, abbreviated as MAP, among all tooth types by month (selected from 1), i.e., adjusted raw data; see text for full explanation.

Figure 7 shows the adjusted MAP distribution that we call SMAP, i.e., a smoothed maximum prevalence distribution, a closer approximation of the shape of the “true” prevalence distribution curve. Here it can be seen that the risk of experiencing a stress event rises sharply after birth, reaching about 55% by the end of month 2 of life, a level maintained for the next several months. During months 5 and 6, another increase in risk to about 80% occurs, and this peak level is sustained into month 9, when a decline to about 40% takes place. Although a few Wilson bands are observed beyond month 11 (as seen in Fig. 6, MAP declines to 17% and 7% in months 12 and 13, respectively), these last 2 months were left out of Figure 7 since the number of teeth and tooth types remaining in the sample by this time are likely too few to accurately represent this population’s systemic stress-event signals. In fact, even discarding the last 2 months, it is likely that the SMAP curve in months 10 and 11 still underestimates true prevalence, since the Wilson bands observed in these months are located toward the cervix of the only two tooth types, canines and second molars, left in the sample (Fig. 3). This means that the latter 2 months of the SMAP curve are in the higher tails of the Wilson band distribution (see above).

DISCUSSION

Early childhood living conditions in *Portus Romae* as detected in enamel growth

The SMAP curve for the first 11 months illustrated in Figure 7 allows us to explore whether the changing prevalence rates through the period can be related to specific morbidity events for this population. The life of infants and children in Imperial Rome is considered to have been precarious. Earlier historians promulgated the “indifference” hypothesis (the belief that Roman society cared little for the young) because of reports of exposure and abandonment, as well as descriptions of wet nursing

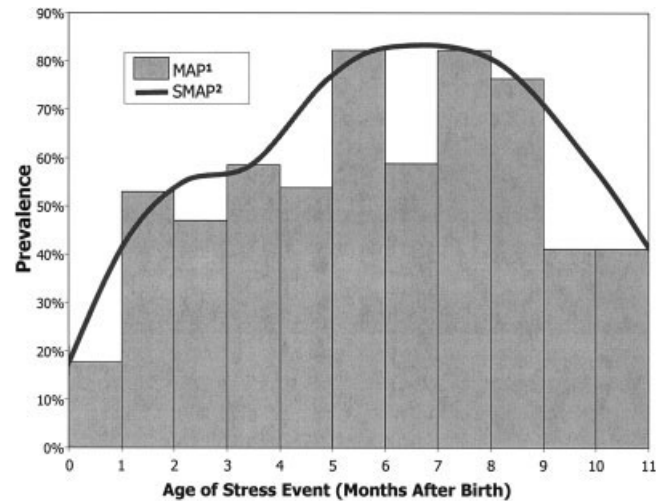


Fig. 7. Maximum prevalence by month and smoothed curve, suggesting shape of “true” prevalence distribution, which we call smooth maximum prevalence (SMAP). See text for more detailed explanation of these distributions.

and swaddling. This interpretation was sufficiently criticized, but there is no doubt that infant mortality rates were high, just as they were in all preindustrial and developing countries until the early 20th century (Garnsey, 1991; Saunders and Barrans, 1999; Rawson, 2003).² High infant mortality rates as well as high post-neonatal mortality are known to be powerful barometers of environmental and social conditions in human societies, particularly of patterns of infant feeding and care.

The newborn infant of Roman antiquity faced a variety of additional challenges to survival that could have contributed to morbidity episodes. Historical sources show that the medical profession of ancient Rome warned against giving colostrum or food of any kind right after birth (Soranus of Ephesus and Temkin, 1991). Tight swaddling of almost the entire body was also recommended for up to 2 or 3 months. Wet nursing too was common, at least among the wealthy Roman classes, although this practice might have actually been helpful in cases where it was felt that the child was not receiving enough milk from its mother.

Garnsey (1991) identified two periods during infancy as particularly vulnerable for infants of the Roman period. The first is said to be around 3 months, when supplementary foods that were nutritionally suspect or unhygienically prepared and administered might first be introduced. Three months makes sense as a common point for such introductions, since an infant developing relatively normally begins to sit up and take a more active interest in his/her surroundings at 3 months (Berk, 2001, 2003). Accounts of milk replacement foods in ancient and Imperial Rome refer to cereals or bread softened with milk, sweet wine, or honey wine.

²On the basis of documentary and epigraphic evidence, Garnsey (1991) calculated the infant mortality rate in ancient Rome to have been 280/1,000, an extremely high value even when compared to recent documented rates in very poor developing countries.

A second period of infant vulnerability to disease often occurs if the supply of breast milk is diminished and inadequate weaning foods are provided. This often happens around 9 months, when there is acceleration in the velocity of growth. But cultural attitudes toward the initiation and progress of the weaning process are crucial to understanding infant health (Katzenberg et al., 1996; Herring et al., 1998). Garnsey (1991) described prescriptions for a weaning timetable in the Roman period. Galen said that only milk should be given until the infant cut its first teeth, at around 7 months. Soranus, in his *Gynecology*, offered detailed instructions, stating that the infant is not ready to receive solid food before 6 months (he also noted that women who give cereal at 2 months are “too hasty”).

If the documented prescriptions for infant feeding were actually incorporated into cultural norms found among the populace of Imperial Rome (and thus *Portus Romae*), then the concurrence between peak age frequencies of Wilson bands in this sample and reconstructed behavioral descriptions seems more than fortuitous. It can be seen in Figure 7 that the prevalence of Wilson bands can be divided into several episodes. After the initial dramatic rise, the curve reaches a maximum at around 2 months that is maintained through month 5, and then a second, higher level is reached around month 6, which continues through month 9. This pattern appears broadly consonant with both general expectations for risk periods during the first year and for descriptions given for the classical Roman period, although morbidity events other than those associated with the weaning process are also involved.

Interpopulation comparisons and other considerations

As indicated earlier, so far as we are aware, there were no previously published “large-scale,” population-wide microscopic studies of deciduous teeth with which to compare our results. Although there were some macroscopic studies, we do not feel that such comparisons are apt. It is not only that between-study figures are complicated by differences in reporting approaches and diagnostic criteria; the fundamental problem is that attempts to establish “true” prevalence distributions like our SMAP have not been undertaken by anyone else. Importantly, too, in archaeological populations, using dental microstructures to establish a precise lesion chronology allows us to overcome many of the problems that plagued other surveys.

However, as we said elsewhere (FitzGerald and Saunders, 2005), there are limitations on the epidemiological interpretation of Wilson bands because of the nature of the phenomenon itself. Since Wilson bands and regular striae of Retzius are formed in the same way (although the triggers that cause the disruptions in enamel growth are different), their appearances are very similar. We believe that they can only be distinguished by an arbitrary definition based on length of the striae, a Wilson band being a stria that extends for 75% or more of the distance between the enamel-dentine junction and occlusal surface in imbricational enamel, or for 75% of the distance around the dentine horn in cuspal enamel. Having to rely simply on length means that some lesions are likely not to be identifiable because of the impossibility of structuring a definition that allows the whole continuum of stress events to be recognized, while still excluding regular brown striae of Retzius from the cate-

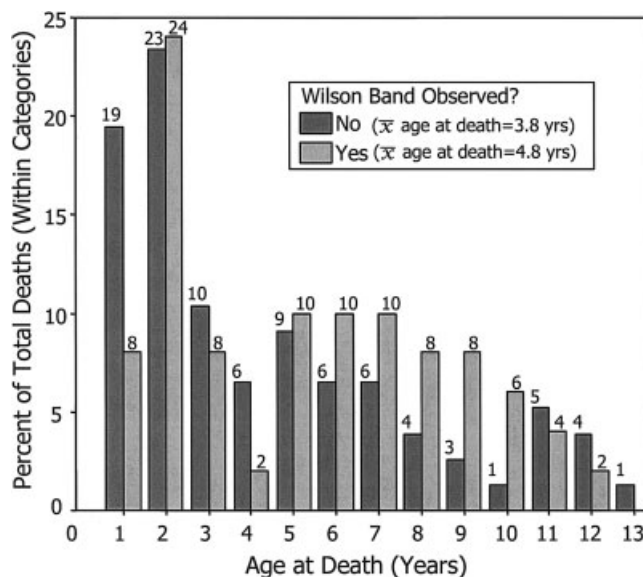


Fig. 8. Age at death distribution, showing percentage by year of total number of deaths for those individuals with Wilson bands observed in their crowns, and for those with no observed Wilson bands. Numbers over bars represent actual percentages of children dying in year in each category.

gory (for a full explanation, see FitzGerald and Saunders, 2005). The result of this is that Wilson band frequencies (and also stress events, for which they are the proxy measure) should be recognized as being understated. Our SMAP curve consequently expresses the minimum risk profile for this population.

Figure 8 shows the percentage of deaths in each year for our sample, broken out between those in whom Wilson bands were observed, and those in whom none were seen. The mean age at death is also shown: those with Wilson bands have a higher average age of death, at 4.8 years, than those without Wilson bands, whose average age at death is 3.8 years. A Mann-Whitney test reveals that the two mean ages are significantly different ($Z = -2.207$, $P = 0.043$). Somewhat at odds with this, Figure 8 also shows that a greater proportion of children unaffected by Wilson bands died in the first year of life than those marked by stress events (19% of those without Wilson bands vs. only 8% of those with Wilson bands), although it should be pointed out that a Kolmogorov-Smirnov test indicates that the shapes of the two cumulative distributions do not significantly differ ($Z = 1.264$, $P = 0.082$), suggesting that this first-year result lacks statistical significance.

The fact that those with no Wilson bands have a lower mean age of death than those with Wilson bands may suggest that these two groups do not share the same “frailty” (Wood et al., 1992; Milner et al., 2000), a term used to describe the risk or propensity, for whatever reason, of dying. Heterogeneous frailty means that not all children have the same age-specific risk of dying. This difference in mortality risk can confound aggregate-level analyses like ours. However, we feel that we have a special case in terms of archaeological studies since: 1) we are able to date our dental lesions very precisely and are confident that their accuracy is measured in days rather than longer periods of time (FitzGerald and Saunders,

2005); 2) dental defects, by definition, are “healed” and not active at time of death; 3) the age of death of our population, consisting as it does of all young children, can be quite accurately established; 4) the shapes of the two distributions of age at death for those with Wilson bands and those without are not significantly different (as above), which may well translate into similar frailty distributions, and by extension no selective mortality; 5) the period of focus of our Wilson band study, the first year of life, is very short, and in fact most children survived it; and 6) importantly, the approach to analysis and the establishment of chronology using dental microstructures effectively yield longitudinal data; we have a complete record through the whole period of crown development.

CONCLUSIONS

This is the first population-wide study of microscopic dental defects in deciduous teeth. We established the chronology of defects utilizing enamel microstructural growth markers, which produce a very high level of accuracy (FitzGerald and Saunders, 2005), allowing timing to be established in very discrete units. This gives us confidence that the resultant population frequency statistics accurately reflect real peaks and troughs of Wilson band occurrence in the population of *Portus Romae*. This in turn allowed us to produce MAP and SMAP distributions that for the first time portray a realistic risk profile for a past population of children. There are two periods of high prevalence, the first beginning around age 2 months and continuing through month 5, and the second higher peak beginning around month 6 and continuing through month 9. Further work extended our information on morbidity events in the permanent tooth of this skeletal sample (Bondioli et al., 2004). We hope that others will follow with similar studies that will permit interpopulation comparisons of this indicator of childhood health.

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