Skeletal Manifestations of Rickets in Infants and Young Children in a Historic Population From England

S. Mays,¹* M. Brickley,² and R. Ives²

¹Ancient Monuments Laboratory, English Heritage Centre for Archaeology, Eastney, Portsmouth PO4 9LD, UK
²Institute of Archaeology and Antiquity, University of Birmingham, Birmingham B15 2TT, UK

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ABSTRACT Gross and radiographic changes characteristic of inadequate bone mineralization due to rickets are described in 21 immature skeletons from a 19th-century urban population from Birmingham, England. The aims of the study are as follows: to evaluate and if possible augment existing dry-bone criteria for the recognition of rickets in immature skeletal remains; to investigate the value of radiography for the paleopathological diagnosis of rickets; and to compare and contrast the expression of rickets in this group with that previously documented for a rural agrarian population from Wharram Percy, England. Some gross skeletal signs of rickets which were not previously well-documented in paleopathological studies are noted. The worth of radiography for evaluating structural changes to both cortical and trabecular bone in the disease is demonstrated, and features useful for the interpretation of vitamin D deficiency are discussed. The pattern of skeletal elements affected and the severity of changes differs in the Birmingham group from that seen in the comparative rural population. It is emphasized that a variety of factors may influence the expression of rickets in paleopathological material, including rate of skeletal growth, age cohort affected, and intensity of vitamin D deficiency. Nevertheless, careful analysis, not only of the frequency of rickets but also of the degree of severity of lesions and the patterning with respect to skeletal elements affected, may enable more nuanced understanding of the biocultural context of the disease in earlier populations. Am J Phys Anthropol 129:362–374, 2006. ©2005 Wiley-Liss, Inc.

Rickets is a disease of infancy and childhood caused by a deficiency of effective vitamin D. Vitamin D plays a crucial role in the metabolism of calcium and phosphorus. When vitamin D is deficient, there is insufficient mineralization of newly formed bone in the growing skeleton and inadequate maintenance of previously formed bone tissue (Mankin, 1974a). The skeletal effects of rickets are porosity, and deformity of the inadequately mineralized bone under mechanical forces.

Vitamin D is naturally present only in minor amounts in most foods; the great majority is synthesized by the action of ultraviolet light on chemical precursors in the skin. Rickets may be caused by a variety of factors affecting vitamin D metabolism, including absorptive disorders of the gut, liver disease, and renal disorders (Mankin, 1974a,b, 1990; Resnick and Niwayama, 1988, p. 2089–2126); but the most important causes relate to inadequate acquisition of vitamin D. In historic populations, inadequate synthesis of vitamin D associated with insufficient exposure of the skin to solar ultraviolet was the cause of the overwhelming majority of cases (Mays, 2003).

The paleopathological study of rickets has a long history. The earliest studies date from the mid-19th century (Burland, 1918). Nevertheless, convincing paleopathological reports of rickets remain fairly few, at least in part because it was, prior to the Industrial Revolution, a genuinely rare disease. Most published work consists of individual case studies (e.g., Power and O’Sullivan, 1992; Formicola, 1995; Blondiau et al., 2002; Pfeiffer and Crowder, 2004) or descriptions of single or some few cases noted during broader physical anthropological or paleopathological studies of skeletal collections (e.g., Gejvall, 1960; Dawes, 1980; Bennike, 1985; Czarnetski et al., 1985; Stirland, 1985; Angel et al., 1987; Molleson and Cox, 1993; Werner et al., 1998).

Recently, however, work began to appear which takes an explicitly biocultural approach to understanding rickets at a population level. For example, Littleton (1998) found changes suggestive of rickets in 10 juvenile skeletons from archaeological sites in Bahrain. She emphasized the importance of cultural factors in the disease, and interpreted her findings as indicating that cultural avoidance of sunlight may have a long history in that region. A review of the paleopathological literature on rickets in England (Mays, 1999) contrasted a high rate in an 18th–19th century London group with low rates for the disease reported for Medieval populations. Climatic change, urbanization, occupational factors, and rising industrial pollution were implicated in the elevated prevalence in the post-Medieval group.

Although rickets is a disease of infancy and early childhood, most paleopathological diagnoses of rickets were made on the skeletons of adults or older juveniles...
from the presence of bending deformities, which in these age groups may be indicative of past episodes of rickets (Mays, 2003). Paleopathological textbooks also traditionally concentrated on the identification of the disease using these aspects (e.g., Steinbock, 1976; Außerheide and Rodríguez-Martin, 1998). Only recently was methodological work aimed at the collation for paleopathologists of the more subtle changes of active infantile disease. Ortner and Mays (1998) described a constellation of gross changes which appeared to be associated with active rickets, using eight Medieval skeletons of infants and young children. They distilled from their observations 10 principal features (Ortner and Mays, 1998, their Table 1), which relate to aspects of bony porosity, flaring of metaphyses, and biomechanical deformation of postcranial elements. Although none of the features they identified are diagnostic on their own, in combination they enable the recognition of rickets in skeletal material, and so might be considered tentative diagnostic criteria for active disease in immature remains.

The present study investigates rickets in immature skeletal remains from a 19th century cemetery in Birmingham, England. This work has both methodological and biocultural aspects. The methodological aims are twofold. First, it aims to characterize the dry-bone expression of vitamin D deficiency in this group of immature skeletal remains and hence to evaluate and, if possible, extend and augment existing criteria (Ortner and Mays, 1998) for recognizing rickets based on gross bony changes. Second, the work aims to evaluate the utility of radiography in the paleopathological study of rickets. On a biocultural level, it aims to compare and contrast the prevalence and expression of rickets in the study sample, drawn from an urban community, with that previously reported for a rural group (Wharram Percy), and hence to evaluate the effects of rural vs. urban lifestyle on the disease.

**MATERIALS AND METHODS**

Eight hundred and fifty-seven burials were excavated from the churchyard of St. Martin’s, Birmingham. The skeletal remains date predominantly from the 19th century. An osteological report on 505 of the skeletons was prepared (Brickley and Buteux, in press). The collection has been reburied.

Several factors render the study assemblage suitable for the current purposes. Gross bone preservation was generally excellent, with minimal postdepositional erosion of bone surfaces. This is important, as many of the dry-bone manifestations of rickets in infants and young children require first-class bone preservation for their recognition (Mays, 2003). In addition, fragmentation of the material was fairly minimal, so that soil ingress into bone interiors was less of a problem than is often the case with archaeological material. This facilitated radiographic study. Of the 164 immature skeletons, 21 showed signs of rickets and are the subject of the current work. This fairly large group showing signs of vitamin D deficiency offers the potential for observing a range of expression of the disease, including both active and healed cases.

Age at death was estimated using dental development (Buikstra and Ubelaker, 1994). No attempt was made to determine sex. Gross indications of deficient bone mineralization were recorded using careful visual examination,
including use of a hand lens when necessary. Some of the more subtle alterations of the subchondral parts of long-bone metaphyses were evaluated using scanning electron microscopy.

Radiographs were taken of all major intact postcranial elements in medio-lateral and anterio-posterior views. Most of the radiographic criteria for the identification of rickets in the clinical context emphasize aspects such as bending deformities, broadening and cupping of metaphyseal subchondral bone, and "fraying" of bone beneath the epiphyseal plate (Thacher et al., 2000; Pettifor, 2003, p. 555–557) which, in the dry specimen, are readily apparent on gross visual inspection. In our radiographic study, we confined ourselves to the examination of alterations of internal bone architecture which are not visible grossly in the undamaged specimen.

We divided the cases into active and healed rickets. Following Ortner and Mays (1998), active cases were identified as those showing porosity of cortical bone in the cranial or postcranial skeleton, and/or porosis/roughening of the bone beneath the epiphyseal growth plates. These abnormal features would have been filled in vivo with unmineralized osteoid. As the individual recovers from the disease, the defects are filled in with bone, obliterating them. Hence, cases showing signs of vitamin D deficiency but lacking both porous cortices and growth-plate abnormalities were classified as healed.

### RESULTS

#### Macroscopic features

The principal macroscopic observations are summarized in Tables 1 and 2.

Among the active cases, porosity of cortical bone was observed more frequently in the postcranial skeleton than in the cranium (Fig. 1): 12 individuals showed changes in postcranial, and two in cranial elements. Abnormal porosity/roughening of the surfaces of the diaphyseal long-bone ends underlying the epiphyseal growth plates was observed in eight active cases (Figs. 2, 3). Changes were seen, in descending order of frequency, at the distal ulna (3 of 3 individuals in which this surface was intact for observation), distal radius (4 of 5), distal tibia (4 of 8), proximal tibia (3 of 8), and distal femur (1 of 6). These are among the sites of most rapid endochondral long-bone growth in the immature skeleton (Scheuer and Black, 2000, p. 19). Bone changes in rickets due to the direct effect of metabolic disturbance (as distinct from those arising from mechanical deformation or adaptation) are generally most frequent, and most pronounced, at those parts showing the most rapid growth (Adams, 1997; Ortner, 2003, p. 393–398). Ortner and Mays (1998, their Fig. 10b–d) provided a three-stage sequence to illustrate grades of severity of porosis/roughening of the bone underlying the epiphyseal growth plates which they observed in their Medieval cases from Wharram Percy. These changes ranged from fine-grained roughening, through coarser roughening with some pitting, to extreme roughness and porosis. At St. Martin’s, instances resembling each of these three stages were seen (Table 3). However, in about half of all long-bone ends in which abnormalities were found, grossly observable changes were restricted to a slight increased roughness of the bone surface, giving it a texture resembling

![Fig. 1. Frontal bones, burial HB862, showing porosity in glabella region.](image-url)
velvet cloth (Fig. 3). We interpret this as a change more slight than any documented by Ortner and Mays (1998) for the Wharram Percy cases. The appearance of the subchondral surface under scanning electron microscopy seems to support this interpretation. It shows roughening and porosis indicative of deficient mineralization of the growing surface, but to a lesser degree than in the

scanning electron micrograph of the Wharram Percy bone (Fig. 4) used to illustrate the least severe of the grades of Ortner and Mays (1998, their Fig. 10b).

Among the abnormalities observed both in active and in healed cases, flaring of the long-bone metaphyses was the most frequent change (Fig. 5), present in all but one of the 20 cases where observations could be made. In the long bones, metaphyseal flaring was observed (in descending order of frequency) at the distal femur, distal tibia, distal radius, distal ulna, and proximal tibia. In many instances, particularly in the distal forearm bones (Fig. 3) and distal tibia, the subchondral bone was also cupped (concave). Metaphyseal flaring appears to result both from increased width of the epiphyseal growth plate and from spreading of the softened bone-end under mechanical forces; mechanical forces are also responsible for the cupping deformity (Mankin, 1974a). In some instances, thickening of the diaphysis was also present. It is possible that these two phenomena are linked in rickets: as the bone grows longitudinally, metaphysis becomes diaphysis; abnormally wide rachitic metaphyses may hence become abnormally wide diaphyses.

Long-bone bending deformities were present in all the healed and all but two of the active cases. Leg-bone deformity was about three times as frequent as arm-bone deformity. Femur and tibial diaphyses were equally likely to show bending; the fibula was affected rather less often. In the femur, the most frequent pattern was simply an increase in the natural anterior diaphyseal curvature, but in two cases (both healed) there was localized anterior angulation in the proximal part of the diaphysis (Fig. 6). In addition, 6 individuals (3 active cases, 3 healed) showed decreased femur neck angle (coxa vara) and flattening of the bone underlying the femur head (Fig. 5). In the tibia, bending was generally anterior. Another deformity observed in the tibia, in 10 (7 active and 3 healed) of 19 individuals where observations could be made, was an abnormal angulation of the distal end so that the medial side of the epiphysis is raised (Fig. 7).

The ulna was the arm bone which most often showed deformity. This generally consisted of an exaggeration of the posterior curvature of the proximal diaphysis (Fig. 8). Only two individuals showed humeral and one individual showed radial bending.

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**Table 3. Grading of severity of porosis/roughening in bone surfaces underlying endochondral growth plates**

<table>
<thead>
<tr>
<th>Burial</th>
<th>Age at death</th>
<th>Bone surface</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>HB595</td>
<td>3–6 months</td>
<td>Distal tibia</td>
<td>2</td>
</tr>
<tr>
<td>HB8</td>
<td>11 months</td>
<td>Proximal tibia</td>
<td>1</td>
</tr>
<tr>
<td>HB217</td>
<td>1.3 years</td>
<td>Distal ulna</td>
<td>4</td>
</tr>
<tr>
<td>HB38</td>
<td>1.5 years</td>
<td>Distal femur</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proximal tibia</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal radius</td>
<td>1</td>
</tr>
<tr>
<td>HB496</td>
<td>1.5–2 years</td>
<td>Distal ulna</td>
<td>1</td>
</tr>
<tr>
<td>HB402</td>
<td>2 years</td>
<td>Distal tibia</td>
<td>1</td>
</tr>
<tr>
<td>HB108</td>
<td>2–3 years</td>
<td>Distal radius</td>
<td>4</td>
</tr>
<tr>
<td>HB772</td>
<td>2.5–3.5 years</td>
<td>Distal tibia</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal ulna</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Distal radius</td>
<td>4</td>
</tr>
</tbody>
</table>

1 Grade: 1, “velvety” appearance (see text); 2, resembling Ortner and Mays (1998, Fig. 10b); 3, resembling Ortner and Mays (1998, Fig. 10c); 4, resembling Ortner and Mays (1998, Fig. 10d).
In the axial skeleton, four individuals showed rib deformities consisting of an increased acuteness at the angle (Fig. 9), and two showed abnormal lateral curvature of the ilium.

A feature observed only in healed cases was deposition of porous bone on the subperiosteal surface of the concavity in long bones showing a bending deformity. Two individuals displayed this phenomenon, in both the tibiae (Fig. 10) and the femora. The affected bones showed particularly marked bending deformity.

**Radiographic features**

The principal radiographic abnormalities in bone structure are summarized in Tables 4 and 5. In two individuals (burials HB427 and HB862), the poor condition of the remains precluded radiographic study. Among the remaining 19, the most frequently observed change was coarsening and diffuse osteopenia of the trabecular structure in the long-bone metaphyses (Fig. 11). These changes were seen in 10 of 12 active cases where observations could be made and in two healed cases. In one instance, burial HB496, an area of abnormally coarsened trabeculae is separated from the end of the diaphysis by an area of normal trabeculae (Fig. 12).

In four individuals, there was diffuse demineralization of cortical bone and coarsening of its structure, leading to loss of the normal, sharp cortico-medullary distinction. There were fewer instances of this than of changes to trabecular bone, and in no case was demineralization of the cortex observed in the absence of trabecular bone abnormalities. This pattern probably reflects the greater metabolic activity of trabecular bone, which means that it responds more readily to vitamin D deficiency.
In one individual (burial HB772), a distinct type of cortical demineralization, cortical tunneling, was seen. This presented as linear radiolucencies within the cortex, giving a longitudinally striated appearance radiographically (Fig. 13).

Adaptation to altered mechanical forces on bones showing a bending deformity was apparent radiographically in 11 cases. This most usually took the form of thickening of cortical bone on the concave side of the deformity. However, in instances of significant bending at the metaphysis, there was increased mineralization of bone trabeculae on the concave side (Fig. 14). In addition, where a bending deformity was marked, struts of bone were occasionally visible spanning the medullary cavity.

**DISCUSSION**

**Macroscopic features**

In general terms, the macroscopic features of cortical bone porosity, thickening of sternal rib ends and long-bone metaphyses, and distortion of bones in response to mechanical loading resemble those which were observed previously in paleopathological studies of rickets in immature remains (Ortner and Mays, 1998; Littleton, 1998; Blondiaux et al., 2002; Pfeiffer and Crowder, 2004). However, some distinctive features were observed that are not well-documented in the paleopathological literature.

To our knowledge, coxa vara and flattenening of bone beneath the femoral head were not previously described in paleopathological examples of rickets. Although not noted by paleopathologists, coxa vara has long been recognized clinically in cases of rickets, and is ascribed to deformation of the inadequately mineralized bone due to weight-bearing in the upright stance (Hess, 1930, p. 179). The observations at St. Martin’s are consistent with this interpretation: all six cases with these deformities were aged over 2 years, and so would likely have been walking rather than crawling.

Medial tilting of the distal epiphysis of the tibia was a distinctive and frequent deformity. This was, to our knowledge, described paleopathologically in rickets in only one previous instance (Pfeiffer and Crowder, 2004), despite the fact that it has long been known clinically (Hess, 1930, p. 178). Unlike the proximal femur deformities referred to above, medial tilting of the distal tibial epiphysis was seen even in the youngest individuals from St. Martin’s, so it does not seem to be specifically associated with weight-bearing in the upright stance. This tilting of the epiphysis was frequently a result of bending deformity of the distal diaphysis and metaphysis. However, on occasion, epiphyseal tilting occurred in the absence of any marked bending of the distal portion of the bone. In such instances, it seems likely that it was principally a result of asymmetrical muscle pulls on the weakened growth plate (Resnick and Niwayama, 1981, p. 2098).

The presence of porosis/roughening of the bone beneath the epiphyseal growth plates and of porosis of cortical diaphyseal bone indicates deficient mineralization of bone newly deposited as a result of endochondral and appositional growth, respectively, and enables active cases of rickets to be identified. In both healed and active cases in the current material, the metaphyseal flaring and diaphyseal bending deformities were similar in character and frequency in cases classified as healed and active. This suggests that, during infancy and early childhood, there was
little general tendency toward removal or reduction of deformities by bone remodeling following recovery from rickets. Rather than removing or reducing deformity, the action of bone remodeling seems to have been to strengthen the abnormally curved bone. Consistent with this, the only gross feature confined to healed cases was the deposition of porous new bone on the subperiosteal surface on the concave side of abnormally curved long bones. This feature, which has only been noted cursorily in paleopathological cases of rickets (e.g., Blondiaux et al., 2002), appears to represent a biomechanical adaptation to altered stresses on a deformed bone, reinforcing as it does the side of the curvature where compressive forces are greatest.

**Radiographic features**

The general osteopenia and the thinning and coarsening of bone trabeculae in the St. Martin's cases resemble the pattern seen clinically in rickets. The diffuse osteopenia reflects the inadequate mineralization of osteoid, and the coarsening of cancellous bone structure is due to the complete demineralization of finer trabeculae (Feist, 1970; Silverman and Kuhn, 1993, p. 1747–1751; Renton, 1998). Sequential radiographs of living subjects showed that, during the healing process, the coarse trabecular pattern is gradually replaced by normal, fine trabeculae (Pettifor and Daniels, 1997). The fact that abnormal trabecular bone is more often observed in active than in healed cases at St. Martin's is consistent with this.

The observation in one case (burial HB496), that abnormally coarse trabeculae were separated from the bone end by a band of normal trabeculae, seems to indicate episodic disease, with a phase of recovery following one of disease. However, the vitamin D deficiency seems to have returned in this individual shortly before death, as the distal end of the bone beneath the epiphyseal plate showed a "velvety" texture indicative of deficient mineralization of the growing surface.

We interpret the cortical tunneling exhibited in burial HB772 as reflecting secondary hyperparathyroidism, a conclusion supported by observations made on bone microstructure using scanning electron microscopy (Mays et al., unpublished findings). In rickets there is hypocalcemia, and this stimulates the parathyroid glands to secrete parathyroid hormone, which liberates calcium from the skeleton in an attempt to maintain calcium homeostasis. The initial skeletal result of hyperparathyroidism is diffuse osteopenia, but other more specific radiological features may subsequently appear. One of these is increased intracortical resorption which takes the form of cortical "tunneling," i.e., resorption of compacta within Haversian canals (Resnick and Niwayama, 1988, p. 2225). Where changes are advanced and individual resorptive foci have coalesced into larger areas of bone resorption, they become visible radiographically as longitudinal linear radioluencies. Children with rickets resulting from different causes show differing propensities toward displaying bone changes due to secondary hyperparathyroidism. Vitamin D deficiency rickets may, if prolonged, give rise to radiographic signs of secondary hyperparathyroidism, but such changes are uncommon (Swischuk and Hayden, 1979; Adams, 1997, p. 621; Pettifor, 2003). Of the conditions which may lead to rickets, renal osteodystrophy, is that which most often gives rise to radiographic evidence of hyperparathyroidism (Swischuk and Hayden, 1979). However, we felt that this was an unlikely diagnosis for burial HB772, as it lacks
the other skeletal changes associated with renal osteo-
On the contrary, although burial HB772 showed severe
active disease (Fig. 2, Table 3), the bone changes are not
different in type from those seen in other cases, and
there is no reason to suspect that this is anything other
than vitamin D deficiency rickets.

Comparison with Wharram Percy

Wharram Percy is a deserted Medieval village. The
burials excavated from the churchyard are of ordinary
peasants who would have lived at Wharram Percy or in
other villages and farmsteads in this rural, agrarian parish.
By contrast, Birmingham in the 19th century was a
rapidly expanding urban industrial center. By comparing
skeletal rickets at these two sites where living conditions
were very different, we may gain an insight into the fac-
tors which may affect the expression of the disease in
early communities. The data on Wharram Percy cases is
taken from Ortner and Mays (1998).

The frequency of rickets is greater among the St.
Martin’s juveniles than at Wharram Percy (21/164 =
13% vs. 8/327 = 2%; chi-square = 21.0, P < 0.01).
There are also healed examples among the juveniles in
the St. Martin’s assemblage, as well as healed cases of
rickets and examples of osteomalacia among the adults
(Brickley and Buteux, in press). These are lacking at
Wharram Percy. A further difference between the two
assemblages is the age distribution of active cases of
rickets. The ages of St. Martin’s active cases covered a
broader age span, and in general were rather older
than those at Wharram Percy (the age range of the
Wharram Percy cases was 3 months to 1.5 years; that

<table>
<thead>
<tr>
<th>Burial</th>
<th>HB863</th>
<th>HB100</th>
<th>HB453</th>
<th>HB311</th>
<th>HB303</th>
<th>HB411</th>
<th>HB596</th>
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<tr>
<td>Age at death</td>
<td>6–12 mo</td>
<td>2</td>
<td>2</td>
<td>2–3</td>
<td>2.5–3</td>
<td>2.5–3</td>
<td>3–4</td>
</tr>
<tr>
<td>Trabecular coarsening/thinning</td>
<td>A</td>
<td>P</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>P</td>
</tr>
<tr>
<td>Loss of cortico-medullary distinction</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Cortical tunneling</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Biomechanical alterations</td>
<td>A</td>
<td>P</td>
<td>P</td>
<td>P</td>
<td>A</td>
<td>P</td>
<td>P</td>
</tr>
</tbody>
</table>

1 Age: mo, months; otherwise, age in years; P, condition present; A, condition absent.

Fig. 11. a: Anterio-posterior radiograph, femur and tibia, burial HB108. There is thinning and coarsening of metaphyseal trabecular bone. b: Anterio-posterior radiograph of normal juvenile femur and tibia for comparison.
at St. Martin’s was 3 months to 4.5 years). A Mann-Whitney test indicates that the difference in age distributions is statistically significant ($U = 14.5, P < 0.01$). This is not simply a sampling artifact, as the age-at-death distributions of the total under-5-year cohorts are similar at the two sites (data not shown). The inhabitants of Medieval Wharram Percy would have led an outdoor lifestyle. Ortner and Mays (1998) suggested that the Wharram Percy infants who showed rickets did so because they were otherwise sickly and so were kept indoors in dark, smoky houses, and so did not experience the direct exposure to sunlight which was likely the norm for this population. By contrast, in the crowded cities of the 19th century, sunlight often failed to penetrate the narrow alleys which ran between tene-

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Fig. 12. Anterio-posterior radiograph, distal radius and ulna, burial HB496. In both bones, area of normal trabeculae (1) is interposed between bone end and region of abnormally coarse trabeculae (2).

Fig. 13. Anterio-posterior radiograph, radius burial HB772 (left), together with normal comparative specimen (right). Diseased element shows longitudinally oriented linear radiolucencies within cortex, consistent with demineralization by “tunneling” resorption. There is also loss of normal cortico-medullary distinction.

Fig. 14. a: Anterio-posterior radiograph, tibia burial HB402. Note increased mineralization of trabecular bone on concave side of abnormal curve in this element. b: Anterio-posterior radiograph of normal juvenile tibia for comparison.
ments, and air pollution by industrial processes attenuated solar ultraviolet, so that even if it did penetrate to ground level, it was of little potency (Loomis, 1970). These factors were mainly responsible for the great rise of rickets in the 19th century (Loomis, 1970), and are likely a factor in the high rate seen at St. Martin’s. Rickets in the St. Martin’s population was probably not an exceptional condition experienced only by infants who were otherwise severely sick, but was a common ailment which frequently affected children whose health was not otherwise seriously compromised. This would be consistent with the presence of healed and recurring disease in the St. Martin’s group, showing that many children with rickets did not have other, fatal diseases but survived into later childhood or adult life.

Clinical observations indicate that rickets seldom appears before about 4 months of age or in those older than 4 years (Ortner, 2003, p. 393). The ages of those with active disease at St. Martin’s span the full range at which the disease might be expected to occur, whereas the cases at Wharram Percy fell exclusively into the lower end of the age range. The reasons for this difference are unclear, but the observation that active cases of rickets are present at St. Martin’s, even at ages where children start to become more mobile and adventurous in exploring their environment, may be consistent with the view that the general environment and living conditions were marginal for adequate vitamin D synthesis.

In many respects, the macroscopic changes seen in the active rickets cases at St. Martin’s resemble those documented for Wharram Percy, but there were also important differences, both in terms of the pattern of elements affected and in the severity of bony changes. Mandibular ramus deformity, which was observed at Wharram Percy, was not evident among the St. Martin’s cases. Ortner and Mays (1998) interpreted mandibular ramus deformity as due to muscle action during chewing. There is evidence for a general reduction in the toughness and coarseness of diets in urban Britain during the post-Medieval period (Mays, 1998, p. 80–81). If this included weaning as well as adult diets, then it might explain the lack of mandibular ramus deformity at St. Martin’s: with a softer diet, mechanical forces generated during chewing may have been insufficient to cause significant deformation of this element.

The coxa vara and proximal femur flattening deformities which, it was suggested, are associated with weight-bearing in the upright stance were not seen at all at Wharram Percy. This probably reflects the likelihood that the Wharram Percy cases had (given their young ages) not yet started to walk but were still crawling infants. Turning to diaphyseal bending deformities, at St. Martin’s, leg-bone deformity was about three times as frequent as arm-bone deformity. At Wharram Percy, the situation was reversed, with cases of arm-bone deformity outnumbering those of leg-bone bending by 4 to 1. This too may reflect the generally older ages of the St. Martin’s cases, meaning that many had started to walk. However, some instances of leg-bone deformity at St. Martin’s were from very young infants. Indeed, all four cases of rickets aged under 1 year showed leg-bone deformity: in three cases, the tibia alone showed bending, and in one case, both the femur and tibia. Early 20th century observations (Park, 1932) indicated that infantile rickets may tend to sit for long periods on the floor with their legs crossed, and this may lead to bowing of the tibia and femur. Perhaps this is the explanation for the lower-limb deformities among the infants from St. Martin’s. The lack of such changes among the Wharram Percy infants is puzzling but presumably relates to differences in postural habits. If the supposition that the Wharram Percy infants were severely sick is correct, then perhaps they lay recumbent rather than sat up.

At St. Martin’s, only two individuals showed porotic changes to the skull vault and orbital roofs, whereas at Wharram Percy, all seven cases where observations could be made showed changes at one or both of these locations. In addition, the St. Martin’s cranial lesions (Fig. 1) are less pronounced than those observed at Wharram Percy, lacking large pores and spicular bone formation. As previously noted, subchondral porosis/ roughening at the long-bone ends was also, in general, less severe than in the Wharram Percy cases.

The skeletal changes of rickets which are a result of the direct effect of metabolic disturbance on the growing bone tend to become less pronounced if growth is slowed (Mankin, 1974a). One possible explanation for the generally lesser severity of bony porosity at growing bone ends and elsewhere in the St. Martin’s cases would be that growth was slower in these cases than in those suffering from rickets at Wharram Percy.

Growth is sensitive to nutrition (Eveleth and Tanner, 1990). Some writers asserted that aspects of the expression of rickets, other than the intensity of changes at growing bone surfaces, may differ between individuals according to their nutritional status. One could therefore argue that study of these aspects might provide evidence concerning whether there are likely to have been nutritional differences, and hence differences in growth patterns, between the St. Martin’s children with rickets and those from Wharram Percy.

Jaffe (1972, p. 388) claimed that when nutrition is poor, rachitic bones have thin, porous cortices, but in individuals who are well-nourished, cortices, although still porous, are thicker, due to greater production of osteoid in these cases. Confusingly, as in both instances bones show porotic cortices, he called the former porotic (or atrophic) rickets, and the latter hyperplastic (or hypertrophic) rickets. In active rickets, osteoid will be unmineralized or poorly mineralized, so one might anticipate that cortical hard tissue will show little difference in thickness between malnourished and adequately nourished rachitic individuals, and this was confirmed radiographically in living subjects (Soliman et al., 1996). However, the distinction claimed by Jaffe (1972) might potentially be observed radiographically and in dry bones in healed cases of rickets, where osteoid has mineralized.

A further difference which, it has been claimed (Jaffe, 1972, p. 390; Jain et al., 1985; Silverman and Kuhn, 1993, p. 1749), distinguishes atrophic from hypertrophic rickets is that, in the former, cupping and widening of long-bone metaphyses are often absent because the poor muscular power of malnourished individuals lessens mechanical forces upon the skeleton. However, radiographic observations on living rachitic individuals with and without protein-energy malnutrition (Soliman et al., 1996) indicate that in practice, most show these features regardless of nutritional status.

The foregoing suggests that inferring nutritional status in rachitic individuals from aspects such as diaphyseal thickening and metaphyseal flaring or cupping is not straightforward. In any event, the St. Martin’s and Wharram Percy individuals do not seem to differ greatly in terms of these features, e.g., metaphyseal thickening
was present in long-bones in 12 of 13 active cases where observations could be made at St. Martin's, compared with 5 of 7 cases at Wharram Percy.

A more direct approach to the investigation of whether there was any difference in growth rates of those suffering from rickets at the two sites is to compare bone growth of individuals with rickets at St. Martin’s and Wharram Percy. However, making this comparison is problematic: the fragmentary nature of the remains from Wharram Percy meant that few elements were intact for measurement, and the generally older age range for the Birmingham cases meant that few individuals with rickets at St. Martin’s could be age-matched with counterparts with the disease at Wharram Percy. Nevertheless, some limited comparisons can be made. Humerus length was the measurement which could most often be obtained from the Wharram Percy cases of rickets. Plotting humerus lengths from juveniles from Wharram Percy with and without rickets showed that, although they are not greatly different, those with rickets generally lie toward the lower end of the size range for their ages (Fig. 15). Plotting the humerus lengths from the St. Martin’s cases on the same graph shows that, like the Wharram Percy rickets cases, they lie not far from the overall Wharram Percy growth curve, but tend toward the lower end with respect to length for age. There is therefore little suggestion of any great difference in growth profiles between the rickets cases from the two sites. Although it is difficult to be certain, it is unlikely that the lesser intensity of porotic changes at growing bone surfaces at St. Martin’s was due to slower growth in these cases than at Wharram Percy.

To some extent, the more marked porosis/roughening of the subchondral bone at Wharram Percy may reflect the younger ages at death of those with active rickets at this site. The relationship between long-bone length and age is generally curvilinear, with growth being more rapid at younger ages, particularly in infancy (Humphrey, 1998). Thus, other factors being equal, one might expect to observe more severe changes at the growing bone ends in a younger cohort. The younger ages of the Wharram Percy cases may also explain the more marked and more frequent porotic changes in the cranial vault in these instances. In infants, the neurocranium is an area of especially rapid skeletal growth (Humphrey, 1998), so it is particularly affected by rickets in infancy (Park, 1932).

CONCLUSIONS

In the St. Martin’s material, the features tabulated by Ortner and Mays (1998) tend, as in the Wharram Percy cases, to occur together. This supports the notion that they are all a manifestation of a single disease. Ortner and Mays (1998) argued that this was rickets, and the current work gives no cause to doubt this. On the contrary, the co-occurrence of these features, together with further gross alterations characteristic of inadequate mineralization of bone, and with radiographic changes consistent with the clinical picture of rickets, serves to strengthen that conclusion.

We identified several macroscopic features in the St. Martin’s material which are consistent with rickets, and the St. Martin’s juveniles, and fewer but more severe cases of rickets but with less severe bone changes among the St. Martin’s juveniles, and fewer but more severe cases at Wharram Percy, may be consistent with the arguments, made above, that rickets at St. Martin’s arose because the environment was generally marginal for adequate vitamin D synthesis, whereas the rickets cases at Wharram Percy may have resulted from more complete exclusion from sunlight for a few individuals.

Unlike previous methodological work (Ortner and Mays, 1998), the material in the current study permitted collation of radiographic signs of rickets in immature individuals. In many instances, alterations to the internal bone structure are subtle, and comparison with radiographs of bones of normal individuals of similar age at death was often helpful in evaluating changes. Radiographic study helped elucidate the response of bones deformed by defective mineralization to mechanical forces; there was evidence for localized trabecular sclerosis and formation of abnormal trabecular struts spanning the medullary cavity, as well as cortical thickening. As with individual dry-bone changes, each radiographic feature is not on its own diagnostic, but in combination, and coupled with careful study of gross changes, they aid in the recognition of rickets in ancient bones. This may be particularly so in cases where postdepositional erosion of bone surfaces limits the gross observations which can be made. For example, the cortical surface porosity and, especially, the porosis/roughening of bone beneath epiphyseal growth plates, which are important manifestations of active rickets, require first-class preservation of bone surfaces for their identification. In circumstances where these were removed or rendered difficult to identify by postdepositional erosion, signs of rickets in the internal bone structure, identifiable by radiography, assume a greater diagnostic importance. When fragmentation of remains precludes radiography, it is important to examine macroscopically the internal bone structure at postdepositional breaks, as this may permit the gross identification of rachitic features which may be seen radiographically in intact specimens.
graphic abnormalities are probably of less value in diagnosis of healed rickets than of active cases. Upon recovery, trabecular abnormalities due to the direct metabolic effects of vitamin D deficiency are progressively removed, and biomechanical adaptations visible radiographically, such as reinforcement of concave sides of sites of abnormal curvature, would be expected to be similar to adaptations to abnormal bending due to other causes.

Most paleopathological work on rickets has relied on identifying the disease in individual cases, and what population-based work has been done has generally looked at prevalence rates. We contend that a better understanding of vitamin D status and its effects on health may potentially be gained if a more detailed study of lesions, including radiographic and, in some instances, scanning electron microscopic examination, is undertaken. This is illustrated in the present study by the findings of evidence for episodic disease and of secondary hyperparathyroidism in specific individuals by radiography, and by evaluation of subtle pathological changes in growing bone ends by scanning electron microscopy. At the population level, this point is illustrated by the comparison with the rural Medieval group. That rickets was more prevalent in the 19th century population of St. Martin’s than in the Medieval group was not unexpected, and is consistent with documentary and skeletal evidence for the great rise in rickets which occurred with industrialization and urbanization in England (Mays, 2003). However, what was less expected was the generally lesser severity of changes in the rickets cases from the St. Martin’s group and the contrast that in the 19th century group some cases were healed, but in the Medieval population all were active at time of death. A variety of factors may influence the skeletal expression of the disease, including rate of skeletal growth (and hence the nutrition and infectious disease burden of those with rickets), the age cohort affected, and the duration and intensity of vitamin D deficiency. Nevertheless, careful analysis of data may allow the relative importance of these different factors affecting the expression of rickets to be elucidated. In the current work, we made the case that the differences in skeletal findings in cases of rickets from St. Martin’s and Wharram Percy reflected, at least in part, differences in the nature of vitamin D deficiency affecting these populations. At Wharram Percy, it seemed likely that the population was not in general vitamin D-deficient, but that the few cases of rickets that were found were of children who were otherwise sick and hence kept indoors in dark, smoky houses. By contrast, at St. Martin’s, the observations were consistent with the idea that this population was generally of marginal vitamin D status, and that rickets was not an exceptional condition, but an ailment commonly experienced by children who were not otherwise severely sick. This comparison illustrates the more fine-grained understanding of disease experience in past populations which may be gained if careful interpopulation comparisons are made, not only of the frequency of rickets but of the severity and patterning of lesions in the skeleton.

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LITERATURE CITED


