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Rural Rickets: Vitamin D Deficiency in a Post-Medieval Farming Community from the Netherlands

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ABSTRACT Rickets is caused by vitamin D deficiency as a result of limited exposure to sunlight and inadequate diet. In the 19th century, rickets was endemic in most northern European cities. In post-Medieval Netherlands, rickets is documented in low frequencies in a few urban samples, but has not been studied in contemporaneous rural populations. Beemster is a rural farming community in the Netherlands that was established in the 17th century upon drained land, with the Middenbeemster cemetery in use until 1866 AD. Ninety-five individuals from the ages of 32 weeks *in utero* to 15 years were examined for rickets in order to understand factors that can cause vitamin D deficiency in rural, non-industrialized populations. To identify rickets in the Beemster sample, ten features were scored, with bending deformities of the lower limb and one other feature, or at least three non-bending features, having to be present in order for diagnosis. Nine individuals (9.5%) had evidence of rickets—a high prevalence, especially for a rural community where ample sunlight was available. The two and three year old Beemster infants were most heavily affected with an age-specific prevalence of 30.4%. Two three-month-old infants also had rickets. Some of the affected may have developed rickets secondarily, as a result of a different illness, but cultural practices including prolonged swaddling, occlusive clothing, and keeping the young indoors, are suggested to have contributed to this high rickets prevalence. Dietary variables including poor weaning foods and common episodes of malnutrition may have also contributed to vitamin D deficiency. This study demonstrates the value of careful analysis of pathological conditions in subadults and highlights that rickets was not only a disease of cities, but affected populations that would appear to have been at low risk, because of maladaptive cultural practices.

Key words: paleopathology; vitamin D; infancy; cultural practices; Western Europe

Introduction

Rickets is a metabolic bone disease with multiple causes. The most common cause is a vitamin D deficiency. Vitamin D is needed for mineralization of newly formed bone matrix, osteoid. When mineralization is impaired due to a prolonged vitamin D deficiency, the bones become soft and will bend due to weight bearing and muscular tension (Ortner, 2003; Brickley and Ives, 2008; Waldron, 2009). The manifestation of vitamin D deficiency in subadults is referred to as rickets, and in adults, osteomalacia. The most important source of vitamin D is dermal synthesis under the influence of UV-B in sunlight. Climate and latitude affect the amount of UV-B and thus the amount of vitamin D that can be produced in the skin. Another way of acquiring this vitamin is by consuming fatty fish (e.g. salmon and tuna) and some other foods (e.g. egg yolk), but the amount in food is low (Holick, 2003). Today, our foods are fortified with vitamin D, and the availability of supplements makes rickets very rare in developed nations (Brickley and Ives, 2008). Neither fortified foods nor supplements were available in the past so limited exposure to sunlight and inadequate diet led to vitamin D deficiency and rickets in many populations.

Beemster is located in the province of North Holland, The Netherlands (Figure 1). The Netherlands have a latitude of 52°N, which almost entirely impedes dermal production of vitamin D from November to March (Holick, 2003). However, sufficient exposure to sunlight in the spring and summer months would provide stores for an entire year and replenish a deficiency (Holick, 2003). Therefore, climatic factors alone cannot adequately explain the occurrence of rickets in Dutch populations, and sociocultural variables must be considered.

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Figure 1. Map of the Netherlands divided into provinces (after http://nl.wikipedia.org/wiki/provincien_van_nederland).

Previous studies of post-Medieval urban collections in the Netherlands found crude rickets prevalences that varied from zero (Baetsen, 2001) to 4.4% (Maat *et al.*, 2002). Studies of rural Dutch communities from this time period are lacking. Comparatively few studies have reported evidence for rickets as caused by non-urban specific causes (i.e. Molla *et al.*, 2000; van der Meer *et al.*, 2006; Mays 2007; Hatun *et al.*, 2011; Palkovich, 2012). This is in part due to the excavation of more urban sites, but is also the result of the preconception that rickets was a disease of the cities, resulting from pollution, crowding, and too much time indoors. As this paper demonstrates, other cultural variables not tied to an urban versus rural juxtaposition, including child care practices, clothing, and diet, can play a major role in rickets prevalence. As well, less research has been done on rickets in past Dutch populations compared to past British populations. More studies of rickets in past peoples from mainland Europe are needed to better understand geographic similarities and differences in etiology.

The aims of this paper are two-fold. First, to present data on the Beemster individuals with rickets in order to draw more attention to the existence of rickets in rural communities, and in countries other than the United Kingdom. Second, to focus on maladaptive sociocultural and dietary variables that may have been interacting to cause vitamin D deficiency in subadults under the age of four.

Materials and methods

The Beemster was established in the 17th century when the former Beemster Lake was drained. The land was parceled out and a large number of farms and manorial estates were built, owned by rich merchants and governors from Amsterdam, and serving as summer-residences (de Jong, 1998). The farming land was meant for agriculture, but due to the high water table and composition of the soil, the land was converted to pastures for cattle breeding (de Jong, 1998). During the 17th and beginning of the 18th centuries, Beemster enjoyed prosperous times by trading its products of wool, butter, cheese, and cattle (de Jong, 1998). However, by mid-18th century, the

Table 1. Overview of affected individuals

Individual	MB11S032V082	MB11S038V026	MB11S046V023	MB11S062V071	MB11S165V242	MB11S189V332	MB11S314V655	MB11S316V641	MB11S343V732	Frequency of feature
Features										
Mean age	3.5 years	2.2 years	2.5 years	3.0 years	2.5 years	3.5 months	3.0 months	3.0 years	3.5 years	
Cranium porosity	A	-	A	P	-	A	P	A	A	28.6%(2/7)
Orbital roof porosity	P	-	A	-	P	P	-	P	A	66.7%(4/6)
Mandibular ramus angulation	A	-	P	-	A	-	-	A	A	20.0%(1/5)
Deformation arms	P	-	A	P	A	-	P	A	A	42.9%(3/7)
Deformation legs	P	P	P	P	P	P	A	P	P	88.9%(8/9)
Flaring of costochondral rib ends	A	P	P	P	-	-	P	A	A	57.1%(4/7)
Cortex of rib ends porous and irregular	A	P	A	P	-	-	P	A	A	42.9%(3/7)
Irregularities of metaphyses of long bones	P	-	P	P	P	P	P	P	P	100%(8/8)
Cortex of metaphyses irregular and porous	A	-	P	P	P	-	P	P	A	71.4%(5/7)
Thickening of long bones	P	-	P	P	P	P	P	A	P	87.5%(7/8)

Overview of affected individuals with each feature scored as either present (P), absent (A), or unobservable (-).

population experienced more frequent and severe hardships. For example, episodes of rodent infestation in the mid-18th and 19th centuries destroyed pastures and crops, and their rummaging weakened the dams which frequently resulted in partial flooding of Beemster (Falger *et al.*, 2012). A rinder pest in 1744 AD killed two-thirds of the cattle and another in 1769 AD killed about half, severely impoverishing the community (Falger *et al.*, 2012). From 1845 to 1847 AD, much of Western Europe, including the Netherlands, experienced potato, rye, and wheat crop failures (Bergman, 1967; Vanhaute *et al.*, 2007). Thus, periods of dietary inadequacy were likely to have affected the Beemster community.

The cemetery of Middenbeemster, located in the center of Beemster, was excavated in the summer of 2011. It was in use from 1617 to 1866 AD, although most individuals date from the 19th century according to archival sources. Approximately 450 individuals were excavated including both sexes and all ages, their preservation being very good. The sample for this research is based on assessment of 450 individuals, from which 95 individuals with adequate completeness and preservation fell into the relevant age range of fetal (youngest at 32 week *in utero*) to 15 years. Fifteen years served as the upper age limit because with epiphyseal fusion of long bones, growth of that area ceases, and osseous changes of rickets would be less visible.

Age was estimated using a combination of several methods: dental measurements of both deciduous and permanent teeth by Liversidge *et al.* (1998), dental development of deciduous teeth by Demirjian *et al.* (1973), dental development of permanent teeth by Moorrees *et al.* (1963), and dental eruption by Ubelaker (1979). For those individuals whose teeth were unobservable, age was estimated based on the stage of bone and epiphyseal fusion by Schaefer *et al.* (2009), long-bone length by Maresh (1970), and clavicle length by Black and Scheuer (1996). Age categories of one year were used, except for the neonates (less than one month of age) who were grouped together with the full-term fetuses (>37 weeks in utero) into a perinate category.

A form was developed scoring ten macroscopic features of rickets as described by Ortner and Mays (1998) and refined by Brickley and Ives (2008). Table 1 shows the scores for all features for each affected individual. The feature 'Growth plate abnormality of long bones' as defined by Ortner and Mays (1998:46), mostly concerns irregularities of the epiphyseal surface and underlying porosity. However, since abnormality of the growth plate in this paper includes cupping and flaring, this feature is renamed to 'irregularities of the metaphyses of long bones'.

Other developmental and pathological processes can result in osseous changes that mimic those of vitamin D deficiency. Therefore, at least three diagnostic features needed to be present, or bending deformities of the long bones and one other feature, for the diagnosis to be rickets. In addition, a distinction was made between healed and active rickets based on the definition of Ortner and Mays (1998): active cases of rickets show porosity of cortical bone in the cranial or postcranial skeleton, and/or growth plate abnormality.

To provide context to the data, Beemster is explicitly compared to four sites from the United Kingdom, the best studied region in Europe. Broadgate and Spitalfields, both located in London (Pinhasi *et al.*, 2006), and St. Martin's in Birmingham (Brickley *et al.*, 2006), are urban sites that date to the same time period as Beemster. Wharram Percy in York (Mays, 2007) occurred earlier during the late-Medieval period but is included because it was a rural community.

Results

Nine individuals have evidence of rickets. Figure 2 presents the distribution of affected individuals. No individuals aged 11, 13, or 15 years (with unfused long bone epiphyses) were encountered. It is not known why so few one-year olds were encountered (n = 2), but because of their underrepresentation, interpretations for this age group are not offered. Table 1 provides an

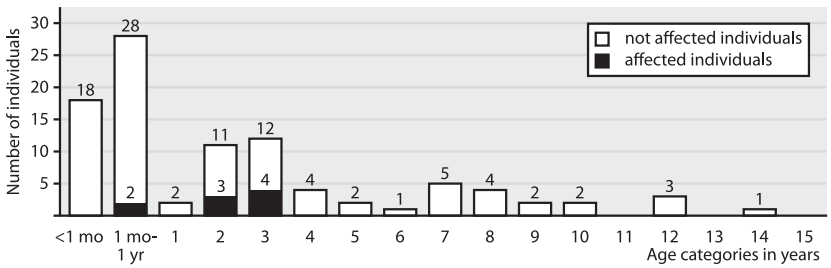

Figure 2. Total number of individuals and the total of individuals affected by rickets. Age is in years, unless stated; 3 denotes 3.00–3.99 and so on.

overview of all affected individuals, their mean age, the scoring of each macroscopic feature as either present (P), absent (A), or unobservable (−), and the frequency of features observed. In Table 2, the distinction is made between active and healed rickets. Five cases were in an active state, and four were in a healed state. All healed cases occur in the three year olds, revealing a trend whereby only older infants moved into the healing process.

Of the affected subadults, individual MB11S062V071 of 3.0 years +/− 12 months of age, suffered most clearly from active rickets. Cranial vault porosity and deformation of both upper and lower limbs are evident. All metaphyseal ends are enlarged and display thickening and deformation of which Figure 3 is an example. The sternal ends of the ribs were enlarged, resulting in a rachitic rosary (Figure 4). Individual MB11S032V082 of 3.5 years +/− 12 months of age also is a clear case of rickets, but in the healing phase, with pronounced bending deformities of the tibiae and fibulae (Figure 5) and upper limb bones, but lacking some other morphological changes such as porous and irregular metaphyseal cortices.

The crude rickets prevalence of the Beemster subadults is 9.5%. Age-specific rickets prevalences provide more information. Rickets prevalence in the two year olds is 27.3% and in the three year olds 33.3%. Finally, two young infants around three months of age also had rickets, for an age-specific prevalence of 7.1% in the under one-year group. Table 3 shows the age-based comparison of Beemster to three of the British collections (Broadgate, Spitalfields, and Wharram Percy). The comparison of Beemster to the fourth British collection of St. Martin's in Birmingham (Brickley *et al.*, 2006) is shown in Table 4 due to differently comprised age categories.

The frequency of each macroscopic feature of rickets is noted to provide a better understanding of its diagnostic value (Table 1). Deformation of the leg bones was observed commonly, in all cases but a single three-month-old infant. All individuals with observable metaphyses had irregularities, and many had thickening. The features that occurred least are those on the cranium: porosity and mandibular ramus angulation.

Table 2. Rickets phase

Individual	Mean age	Phase
S032V082	3.5 years	healed
S038V026	2.2 years	active
S046V023	2.5 years	active
S062V071	3.0 years	active
S165V242	2.5 years	active
S189V332	3.5 months	healed
S314V655	3.0 months	active
S316V641	3.0 years	healed
S343V732	3.5 years	healed

Figure 3. Tibiae and fibulae of individual MB11S062V071 showing enlargement and bending deformities (photograph B. Veselka 2012).

Discussion

Differential diagnosis

Confounding factors in the diagnosis of rickets are, as Brickley and Ives (2008:105) point out, that many of the features are not pathognomonic. For example, orbital roof porosity is a feature that occurs in a number of pathological conditions and is therefore considered to be a non-specific stress marker. Thus, its high frequency in this sample could be because of rickets,

Figure 4. Rachitic rosary of rib 1 to rib 7 of individual MB11S062V071 (photograph B. Veselka 2012).

Figure 5. Tibiae and fibulae of individual MB11S032V082 show marked bending deformities (photograph B. Veselka 2012).

but other conditions may be involved. Scurvy, which is caused by vitamin C deficiency, prevents osteoid from being secreted and causes features such as cranial vault porosity and swelling of the costochondral rib ends (Brickley and Ives, 2008:103–105). While it is possible scurvy could be contributing to the macromorphological changes seen in the Beemster subadults, it rarely results in bowing deformities (Ortner, 2003; Brickley and Ives, 2008; Waldron, 2009) so is unlikely to be the primary cause of all the Beemster cases, as they all have bowing of the upper or lower limb bones. Congenital syphilis can cause bending deformities, but has other pathognomonic markers that would be easily noted when assessing the entire skeleton (Waldron, 2009) and can be discounted as a cause. Other pathological causes of bending deformities in the lower limbs include Blout's disease and congenital defects (Waldron, 2009). Blout's disease is a rare, acquired, and progressive growth disorder of the proximal tibial epiphyses and metaphyses, producing a sharp lateral bend that is usually asymmetric (Cheema *et al.*, 2003). The deformities of Blout's disease (tibia vara) differ from the ones reported by Brickley *et al.* (2010) where tibial deformities were observed in the proximal third of the shaft. Bending deformities of the Beemster tibiae occur medially in the proximal third of the shaft and are symmetric, thus ruling out Blout's disease.

Congenital bowing of the tibia is usually convex with the bending oriented posteriorly and medially. This does not match the bowing of the Beemster subadults. As well, congenital bowing is rare (Brickley *et al.*, 2010) and will not cause the other osseous changes included in our diagnostic method, whereby a minimum of three non-bending features, or bending deformities and one other feature, had to be present, thus ensuring the exclusion of congenital bowing.

Often some degree of bowing is present in children's lower limbs which could be mistaken for rickets (Brickley *et al.*, 2010). Bleck (1982) researched several forms of bowing in children's lower limbs and found most were due to normal developmental processes which usually resolved in the course of maturation. Brickley

Table 4. Comparison between Beemster and St. Martin's*

	Beemster			St. Martin's*		
Age	N	Na	P(%)	N	Na	P(%)
Infant	47	5	10.6	73	14	19.2
Child	36	4	11.1	52	6	11.5
Total	83	9	10.8	125	20	16.0

N is the total of individuals; Na is the total of affected individuals; P is the prevalence.
*= data from Brickley et al. (2006)
Infant = birth – 3 years; Child = 4 – 12 years

Table 3. Comparison between Dutch and British collections

	Beemster			Broadgate*			Spitalfields*			Wharram Percy**		
Age	N	Na	P(%)	N	Na	P(%)	N	Na	P(%)	N	Na	P(%)
0	34	2	5.9	9	1	11.1	38	6	15.8	32	0	0
1	2	0	0	5	0	0	27	7	25.9	69	6	8.7
2	11	3	27.3	4	1	25.0	12	0	0	30	2	6.7
3	12	4	33.3	3	2	66.7	6	1	16.7	21	0	0
4	4	0	0	5	2	40.0	1	0	0	11	0	0
5	2	0	0	2	1	50.0	5	0	0	21	0	0
6	1	0	0	7	0	0	6	0	0	23	0	0
Total	66	9	13.6	35	7	20.0	95	14	14.7	207	8	3.9

N is the total of individuals; Na is the total of affected individuals; P is the prevalence.
*= data from Pinhasi et al. (2006),
**= data from Mays (2007)
Age in years; 3 denotes 3.00–3.99 and so on.

et al. (2010) compared several of the bending deformities examined by Bleck (1982) to the ones found in their study. One of the typical deformities was an anterior twist of the head and neck of the femur. The deformities of the femora noted by Brickley *et al.* (2010) differed by having an anterior curvature of the proximal third of the femoral shaft below the level of the lesser trochanter. In Beemster, the femora had similar bending deformities as the ones reported by Brickley *et al.* (2010). Thus, the observed bowed femora are likely not due to normal developmental processes.

Overall, the observed morphological changes in the affected Beemster individuals are most consistent with a diagnosis of rickets. Certain features may be more common in one sample than another. For example, Ortner and Mays (1998) found medial angulation of the mandibular ramus to be common, but this feature was encountered only rarely in the Beemster subadults, as in the studies of Pinhasi *et al.* (2006) and Mays *et al.* (2006). In the Beemster sample, the cranial traits were least common, while postcranial changes, especially those of the long bones were most common, particularly bending deformities of the legs and irregularities and porosity of the metaphyseal ends. These osseous changes are diagnostic for rickets (Ortner, 2003; Brickley and Ives 2008; Waldron, 2009). Assessing as many reliably characteristic features as possible will lead to a diagnosis that is clear-cut and accurate.

Etiology

While inadequate sunlight is by far the most common cause of rickets, there are other causes of vitamin D deficiency. There are inherited and acquired forms of rickets due to problems in the synthesis of vitamin D either by the liver or kidneys or alterations in mineral metabolism (Brickley and Ives, 2008), and hypophosphatia, which is a disorder of low blood phosphate levels, that causes rachitic changes (Brickley and Ives, 2008). All of these conditions are rare and therefore unlikely to be the cause of rickets in an archaeological sample.

For the Wharram Percy collection, Ortner and Mays (1998) suggested that the occurrence of rickets in only an active state could be partially explained if it formed secondarily. Historical sources and archival documents from the Beemster note the presence of many infectious diseases and episodes of food shortage which would have caused substantial infant morbidity and mortality (Bergman, 1967; Vanhaute *et al.*, 2007; Falger *et al.*, 2012), so it is possible that some rickets cases developed secondarily. However, rickets prevalence in Beemster is markedly higher than in Wharram Percy (Table 3) which implies that not all cases are likely to be secondary. Moreover, the mix of active and healed cases in the Beemster sample, and wider demographic spread of affected individuals, suggests rickets developed in a primary context as well.

Unlike in urban settings architecture, diminishing sunlight and smoke from industrial factories were not factors responsible for causing rickets in the Beemster subadults. As a rural community, abundant sunlight was readily available in the spring and summer months. With a latitude of 52°N, however, dermal synthesis of vitamin D is almost entirely impeded in winter and early spring (Holick, 2003). During that part of the year, the entire population would be dependent on bodily stores and diet for their required supply of vitamin D. Those with low vitamin D stores and/or an inadequate diet would have been especially vulnerable to developing rickets from November to March. Thus, the main consideration for why an individual developed rickets is cultural factors that led to low bodily stores of vitamin D.

As well, the contribution of dietary sources of vitamin D must be considered, because the foremost vitamin D containing foods can sometimes provide enough to prevent the development of rickets. Fatty fish and cod liver oil have the highest amount of vitamin D, with foods like egg yolk and beef liver having lower amounts (Holick, 2006). Yet, episodes causing food shortages such as crop failures, livestock epidemics, and low fish procurement, common throughout the mid-18th and 19th centuries in Holland (Bergman, 1967; van Poppel *et al.*, 2005; Vanhaute *et al.*, 2007), may have limited access to these foods. Regardless, the dietary contribution of vitamin D is comparatively minimal. Those that developed rickets likely had low preexisting vitamin D stores entering the winter months, possibly exacerbated by a diet with inadequate vitamin D. Infants born in the fall or winter would be reliant on fetal accumulation, breastmilk, and possibly early weaning foods (see below) for vitamin D. By about eight weeks after birth, transplacental stores of vitamin D are expired, and breastmilk is a poor source of vitamin D, especially in mothers with low levels (Henderson, 2005). Thus, rickets in the two Beemster three month olds may have been seasonal.

Most of the two year olds with rickets died while it was in an active phase, while most of the three year olds had evidence of healing (Table 2). This suggests that the vitamin D deficiency began at the age of two years, and that those who survived past this age were able to enter a phase of healing. What would have made two year olds most vulnerable? Poor weaning foods and cultural practices, including long periods of swaddling,

occlusive clothing, and being kept indoors, all could have resulted in low vitamin D levels in two-year-old infants.

Weaning foods were likely quite similar among households, with rather homogenous options including cow or goat milk and paps made of grains such as wheat and rye. Weaning foods made with cow's milk are low in vitamin D, and also low in calcium compared to breast milk (Henderson, 2005). Inadequate calcium (hypocalcaemia) leads to an increase in vitamin D requirements to restore the unbound levels of calcium (Brickley and Ives, 2008), thus raising the risk of developing rickets. Weaning foods containing wheat or rye have a high level of phytic acid which inhibits iron and zinc absorption, reducing calcium levels, thus increasing vitamin D requirements (Coulibaly *et al.*, 2011). While there are several ways to prepare cereal-based foods that will reduce or neutralize phytic acid, such as germination and fermentation, these methods are not commonly used in the preparation of weaning foods (Coulibaly *et al.*, 2011). Clearly, common Beemster weaning foods could have contributed to vitamin D deficiency. In addition, the quality of drinking water in Dutch coastal areas in the 18th and 19th century was poor due to the high water table and gradual salinization (van Poppel *et al.*, 2005). The consumption of polluted water may have been a major cause of gastrointestinal illness, decreasing consumption and absorption of critical nutrients, the number one cause of infant mortality (van Poppel *et al.*, 2005). Thus, it is perhaps not surprising that only the infants in Beemster were affected by rickets, as commonly eaten foods either increased vitamin D requirements or caused illness that impacted food intake and/or absorption.

Prolonged swaddling, which implies swaddling over a period of more than six months, was practiced in some farming communities (de Leeuw, 1992) and would have diminished sunlight exposure. Swaddling was thought to ensure straight growth, keep infants warm, promote sleep, and prevent harm, and would also give caregivers more time to tend to other tasks (Gerard *et al.*, 2002). Although already in 1762 AD, philosopher Rousseau warned against swaddling clothes, family tradition, and community norms would have largely determined its duration (Lipton *et al.*, 1965). Van Poppel *et al.* (2005) suggested that mothers of lower socioeconomic status were more likely to swaddle their infants for a longer period of time. Obviously, rickets in the two three month olds from Beemster may have been caused or exacerbated by swaddling, but even rickets in the older two year olds may have been partially caused by low levels of vitamin D during the first year of life because of prolonged swaddling. The low number of infants of one year of age limits assessment of this possibility.

What factors could be limiting dermal synthesis of vitamin D for infants past the age of swaddling? Children younger than four or five years of age would not have been able to genuinely help their parents on the land. During periods of increased farm work, when mothers were needed to help on the land, infants and children were tended for by their grandmothers or older sisters (Schenkeveld, 2008). Tasked with domestic chores in and around the house, caregivers may have kept the young inside, thus inhibiting sunlight exposure. As such, gender-based labour norms could be influencing the amount of sunlight that individuals received. In post-Medieval Netherlands, the division of labour is thought to have been traditional: women doing work in and around the house, men working mostly in the fields (Haks, 1985). This labour division was put into effect at a young age with children older than five years beginning to perform various jobs: boys typically herded cattle or tended the land and girls typically did housework or childcare (Schenkeveld, 2008). This suggests that girls may have been more at risk of developing rickets. Yet, as rickets is absent in individuals older than four years, by this age, both girls and boys must have had sufficient sunlight exposure, and the risk of rickets seems more related to age than gender.

During the post-Medieval period in the Netherlands, children's clothing may have played an important role in limiting dermal synthesis of vitamin D. In the 19th century, children were considered to be small adults and were dressed as such (de Leeuw, 1992). They would be dressed in many layers of clothing covering as much skin as possible, a practice frequently depicted in contemporaneous art, such as David Artz' 'Mother with children and a lamb' (www.niceartgallery.com). Clearly, occlusive clothing and being kept indoors would inhibit sunlight exposure (Molla *et al.*, 2000; van der Meer *et al.*, 2006; Hatun *et al.*, 2011).

As mentioned, by the age of about five years, children became more independent and were likely required or permitted to spend more time outdoors. This transition roughly coincides with when rickets is no longer observed in the Beemster sample. While subadults aged four to fifteen years have no evidence of rickets, ongoing research has found residual rachitic changes in the older adolescents and adults. Thus, some individuals who contracted rickets, likely during infancy, healed and survived. There may be a lack of older subadults with detectable evidence of healed rickets in part because over time healing can diminish or erase some, though not all, rachitic changes (Brickley *et al.*, 2010).

Finally, it is pertinent to consider if differences in socioeconomic status could have influenced the occurrence of rickets in Beemster. At this time, the socioeconomic statuses of the families of affected individuals are not known, but archival data indicate the majority of households were engaged in cattle farming with statuses ranging from quite good (land owners) to rather poor (transitory labourers) (register of deaths, Beemster). One group may have been especially vulnerable. From 1680 AD into the 19th century, an orphanage existed next to the Middenbeemster church (Falger *et al.*, 2012). Complaints to the municipality about poorly fed children and children being late for work are on record. Child labour was common until 1902 AD when the public education law was put into effect (Schenkeveld, 2008). Orphaned infants could have been especially vulnerable to developing rickets because of an inadequate diet, and it is possible they spent a limited amount of time outside, although it is not well known if they were made to work in indoor or outdoor settings, nor the organization of the facility for outside access. Future research will focus on linking archival data to the skeletal data with a major goal being the assessment of socioeconomic status on health.

Population comparisons

In environments with sufficient sunlight, rickets is more rare in rural than urban communities; however, there are a few examples from archaeological contexts (Mays, 2007; Palkovich, 2012). For example, Palkovich (2012) suggested that malnutrition and family social dynamics resulted in cases of rickets in the ancestral Puebloan community of Arroryo Hondo, New Mexico, where ample sunlight was available on a year-round basis. Mays (2007) found several cases of rickets in the one and two year olds at Wharram Percy and suggested rickets to be a secondary condition. Thus, a variety of sociocultural and dietary factors have been proposed to explain the occurrence of rickets in rural populations from different geographic locations and temporal periods.

Previous studies of post-Medieval urban collections in the Netherlands found rickets prevalences for subadults that vary from zero (Baetsen, 2001 for the site of Alkmaar; Maat *et al.*, 1998 for the site of Dordrecht), to 4.4% at 's-Hertogenbosh (Maat *et al.*, 2002), compared to the Beemster's prevalence of 9.5%. Maat *et al.* (2002) note that epidemics of cholera (1830–1850 AD) and typhus (1850 AD), as well as other diseases more common in densely populated urban areas, was likely a major contributory factor to the development of rickets. None of the Dutch collections have a rickets frequency or age pattern that is similar to the Beemster, suggesting a different interaction of causes.

Comparison of Beemster to three contemporaneous British urban collections, Broadgate, Spitalfields, and St. Martin's, as well as the late-Medieval rural community of Wharram Percy, is made to enhance our knowledge of the impact of rickets in Western Europe. At Broadgate, Pinhasi *et al.* (2006) examined subadults from the ages of birth to seven years and found age-specific rickets prevalences higher than those of Beemster for subadults from birth to age five (excluding the one year olds), clearly indicating insufficient vitamin D levels in the many of the subadults. At Spitalfields, subadults from birth to age two were most affected, and some at age three, but at levels usually lower than at the neighbouring site of Broadgate (Pinhasi *et al.*, 2006). Pinhasi *et al.* (2006) point out that individuals from Spitalfields were of higher socioeconomic status than those of Broadgate, which resulted in better living conditions and nutrition for more of the Spitalfields subadults. In the other urban site, St. Martin's, almost 20% infants from birth to age three had rickets, as well as some of the four to twelve year olds. As mentioned, at the rural site of Wharram Percy it was one and two year olds who were most affected (Mays, 2007). None of the British sites used in this comparison have a pattern of age-specific prevalence that is the same as the Beemster, where the highest frequencies occurred in the two and three year olds, with no cases occurring past this age and very few prior to this age. This suggests the way the factors responsible for vitamin D inadequacy in the Beemster subadults combined and interacted is different from what has been previously documented and makes clear the value of additional research on rickets in non-British and non-urban populations.

The etiology of rickets is complex because of the multiple practices that can affect sunlight exposure and diet. Unfortunately, there is a paucity of research on daily life in rural Dutch communities which limits our knowledge of cultural practices thus limiting the specificity of our interpretations. In the Beemster, we propose that dietary and cultural practices such as poor weaning foods, prolonged swaddling, occlusive clothing, and a lack of time spent outdoors could have all played a part in causing a high frequency of rickets in the two- and three-year-old subadults. Also possible is the development of rickets secondarily to a different disease. The suggestions regarding causes of rickets are meant to provoke discussion and research about non-urban-based factors and contribute to our understanding of the etiological intricacy of vitamin D in Western Europe.

Conclusion

In the post-Medieval Middenbeemster cemetery, rickets occurred predominately in two and three years olds. As a rural community, Beemster had ample access to sunlight for over half the year; cultural practices and possibly dietary factors were likely causes of most rickets cases. The interplay of factors limiting vitamin D synthesis, including poor weaning foods, prolonged swaddling, occlusive clothing, and a lack of time spent outdoors, is proposed to have been major causes of rickets in the Beemster infants. Future research will assess the prevalence of residual rickets and osteomalacia in adolescents and adults which will enhance our knowledge about the impact of vitamin D deficiency on the Beemster community as a whole and allow us to analyze other groups that may have been susceptible to developing a vitamin D deficiency, particular women, and how this may have affected their offspring. Compared to contemporaneous Dutch urban sites, Beemster had a higher crude rickets prevalence, highlighting the importance of examining not only industrialized, urban communities for rickets. Moreover, this research demonstrates that rickets is 'not only the English disease' (Belton, 1986: 68) as the overall Beemster prevalence, while lower than that of the comparative British urban sites, is very high in the two and three year olds and higher than that of the British rural site.

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