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Environmental and social destitution in a medieval Orkney island community may demonstrate the role of acute Vitamin A deficiency in the occurrence of epigenetic anomalies

Theya MOLLESON

Abstract

In the eighth century AD a small settlement developed around Newark Bay on the east side of Mainland, Orkney. It expanded in the eleventh century, possibly by autochthonous growth as the settlement prospered before suffering a marked decline in the late twelfth century and reverting to the small community of the earliest centuries. Christians, they buried their dead in the low cliff overlooking the shore. The skeletons of over 200 adults and children have been recovered in rescue excavations. The evidence for growth and decline of the settlement in the 11th to 12th centuries is supported by radiocarbon dates on nearly half of the adults (32), of which a third are dated to the 11th – 12th centuries. The decline in the population appears to coincide with the onset of the Little Ice Age, which began earlier in the North Atlantic than it did further south in England and which was a time of increasingly frequent and severe famines. Before this deterioration in the climate the rapid expansion of the population would have made additional demands on the local resources and in times of prolonged famine would have increased the risks of malnutrition. There must also have been socially induced poverty to account for the very high level of mortality among those infants who were not breast fed and for the high rate of epigenetic anomalies attributable to vitamin A deficiency among the infants and adolescents. The farmers in Norse times were often severely taxed. The failure to breastfeed their babies and the evidence for vitamin A deficiency in the etiology of the anomalies suggests that the women at least were subsisting on cereals and secondary protein sources lacking in essential nutrients to meet their energy needs. There are other signs of social poverty in addition to the high level of infant mortality and evidence for deficiency. Evidence of lepromatous leprosy, which is essentially a disease of poverty and malnutrition; and of non-accidental injury of at least one infant, which is also a response to poverty and over-crowding suggest desperate people at the end of their tether.

Keywords: Vitamin A deficiency, Foetus, Epigenetic traits, Orkney island.

Résumé

*Dans le courant du huitième siècle après J.-C., une petite colonie s'est développée autour de Newark Bay, sur la côte est de l'île de Mainland (Orcades). Elle s'est étendue au onzième siècle, probablement suite à un essor démographique local lié à sa prospérité, avant de subir un déclin marqué à la fin du douzième siècle et de revenir à la petite communauté des premiers siècles. Ils étaient chrétiens et ont enterré leurs morts dans la falaise basse surplombant la côte. Les squelettes de plus de 200 adultes et enfants ont été découverts lors de fouilles de sauvetage. La croissance et le déclin de la colonie au cours des XI*ème *et XII*ème *siècles sont étayés par des datations au radiocarbone réalisées sur près de la moitié des adultes (32), dont le tiers date des XI*ème *et XII*ème *siècles. Le déclin de la population semble coïncider avec le début du petit âge glaciaire, qui a débuté plus tôt dans l'Atlantique Nord qu'au sud de l'Angleterre, et qui fut une période de famines de plus en plus fréquentes et sévères. Avant cette détérioration du climat, l'expansion rapide de la population pourrait avoir entraîné des demandes supplémentaires en ressources locales et, en temps de famine prolongée, avoir augmenté les risques de malnutrition. La présence concomitante de pauvreté induite socialement permet d'expliquer le niveau très élevé de mortalité parmi les nourrissons qui n'ont pas été allaités au sein et la fréquence élevée d'anomalies épigénétiques attribuables aux carences en vitamine A chez les nourrissons et les adolescents. Les agriculteurs vivant à l'époque Viking étaient souvent redevables de lourds tributs. L'absence d'allaitement de certains bébés et les anomalies dues à des carences en vitamine A suggèrent que les femmes au moins avaient une subsistance basée sur les céréales et des sources de protéines secondaires trop pauvres en nutriments essentiels pour pouvoir répondre à leurs besoins énergétiques. En plus du niveau élevé de mortalité infantile et des carences, il existe d'autres signes de misère sociale : la lèpre lépromateuse qui est essentiellement une maladie liée à la pauvreté et à la malnutrition, et au moins un enfant porteur de blessures non-accidentelles. Cette maltraitance, qui est aussi une conséquence de la pauvreté et de la surpopulation, révèle des gens désespérés ayant épuisé leurs ressources.*

Mots-clés : *carence en vitamine A, fœtus, caractères épigénétiques, îles Orcades.*

1. BACKGROUND HISTORY

When the Norsemen invaded Orkney (Fig. 1), about AD 800, the land was sparsely populated. Y-chromosome analysis shows modern Orcadians to be intermediate between Celtic and Norwegian (Wilson *et al.*, 2001). The observed symmetry between Scandinavian mtDNA and Y-chromosome ancestry in Orkney suggests a family based Scandinavian settlement (Goodacre *et al.*, 2005). Orkney has significant (30 %) Norwegian input where Norwegian invaders settled. There was population increase in the 11th century through Norse influence and rapid population decline at the end of the Viking era (Ashmore, 2003: 36).

About 208 skeletons have been recovered from the coastal cemetery at Newark Bay including newborn (35), infants (51), juveniles (41) and adults (81) of both sexes (Molleson, 2005). A Christian chapel at Newark is dated to the 10th century by coins of Eadred (AD 946-955) and Anlaf Sihtricsson (AD 941-944). It was in use for the duration of the cemetery (Brothwell, 1977). The growth and demise of the cemetery at Newark Bay can be interpreted against what is known of the political and climatic history of the islands of the North Atlantic in the early medieval period.

Radiocarbon dates have been obtained for 32 adults – more than a third of the total number – and nearly half of these (47 %) fall between AD 1050 and AD 1200 (Toolis, 2008: Figure 1). The distribution of dates is therefore representative of the demographic history of a small settlement that grew rapidly in the late 11th century and collapsed dramatically towards the end of the 12th century. The population increase could be accomplished by the growth of one family over four generations. There was population increase in the 11th century through Norse settlement and rapid decline at the end of the Viking era, which was around the time of the onset of the Little Ice Age, which began earlier in the North Atlantic than further south, and ended the great voyaging days of the Vikings; it was also a time of famines (Dansgaard, 1975: 27; Tanner, 2000).

The age at death distribution of the skeletal material, with roughly equal numbers of females (N = 49), males (N = 42) and juveniles (N = 118) in the assemblage, is interpreted as representing the dead of a normal settlement, with however a large proportion (17 %) of neonate and infant burials and an unusually large proportion of adolescents, most of them males (Molleson, 2005). The study of the skeletal remains from the cemetery suggested that nutritional status of the people could be a factor in the collapse of the population. Some of the evidence is presented here.

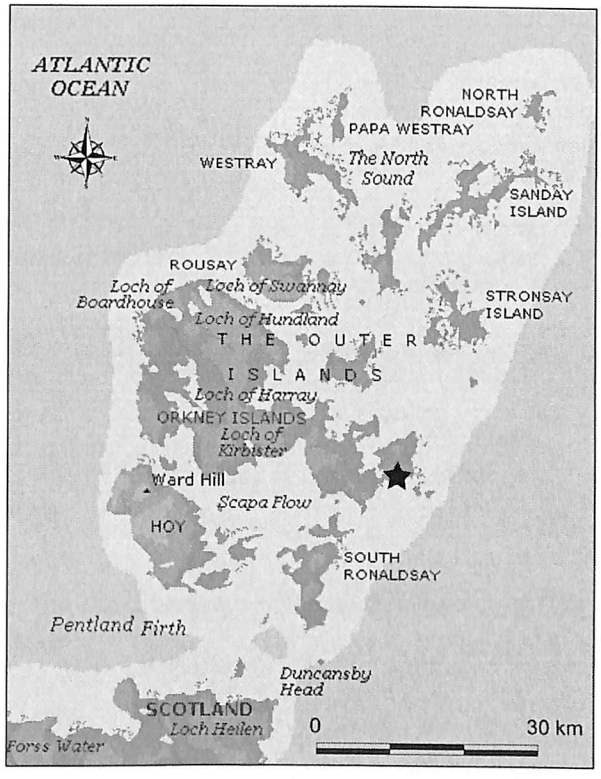


Fig. 1 — Newark Bay is located on the east side of Mainland, Orkney.

2. ANALYSIS

Study of the skeletons was twofold: by observation of the bone morphology and by stable isotope analysis of rib samples from human and animal remains recovered from the site (Richards *et al.*, 2006).

Observation of bone anomalies or epigenetic variants and bone pathologies were recorded for each of the skeletons recovered during rescue excavations between 1968 and 1972. Although the bones were in general well preserved many of the skeletons were incomplete so that frequencies of anomalies are based on number of observations and do not represent true prevalence levels.

Trophic levels deduced from nitrogen isotope analysis were constructed for 103 individuals including a large number (63) of infants, whose age at death within three months could be assessed from dental development. The average trophic levels of the age cohorts were compared to the average trophic levels of the females representing the mothers (tableau 1).

Age at death	Average δ15N	S.D
Neonate	13.9 (N = 12)	1.7
Perinatal	13.5 (N = 14)	1.9
c. 3 months	12.8 (N = 16)	1.8
c. 6 months	12.3 (N = 12)	1.8
c. 12 months	16.0 (N = 9)	1.2
3-12 years	11.9 (N = 11)	1.2
13-17 years	12.2 (N = 11)	1.6
Females	**12.3 (N = 18)**	**2**

Tabl. 1 — Trophic levels (δ15N) of juveniles from Newark Bay.

3. Results

Trophic levels deduced from the measured levels of stable isotopes δN15 and δC13 for females indicate that the diet generally included both terrestrial and marine animal protein (Richards *et al.*, 2006). The stable isotope values for the newborn babies indicate a trophic level increase above the females of only 1 to 2 ‰ – less than the expected 2 to 3 ‰ – and could reflect a higher mortality among babies born to less well fed mothers since the female cohort is only a surrogate for the mothers (Fig. 2). The average trophic levels of infants who died in each of the following three months were lower than for the neonate group (Fig. 3). These babies cannot have been breastfed. Only those infants who survived about a year have the elevated nitrogen levels expected of breastfed infants.

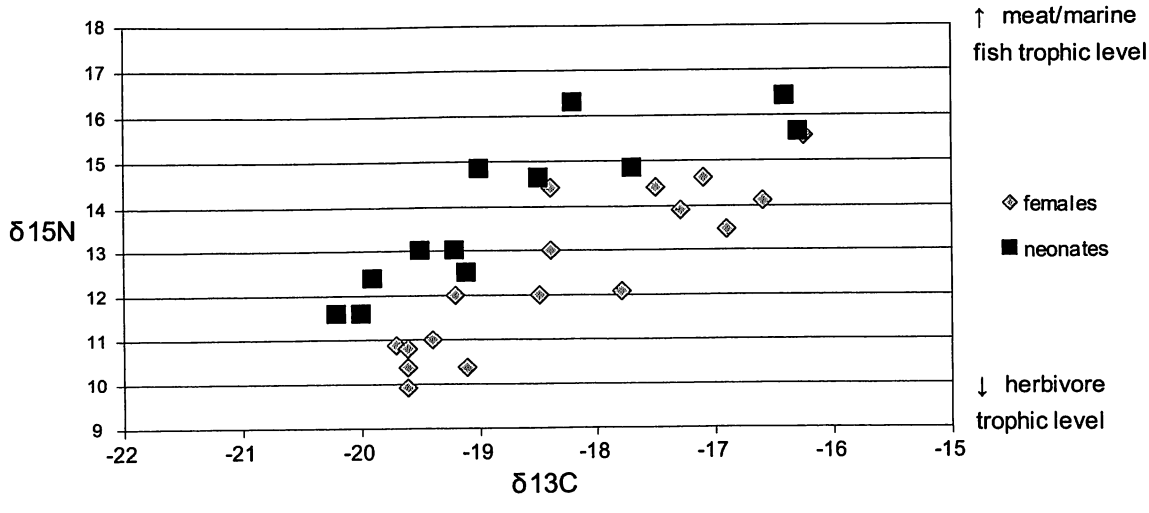


Fig. 2 — Stable isotopes values for neonates and adult females.

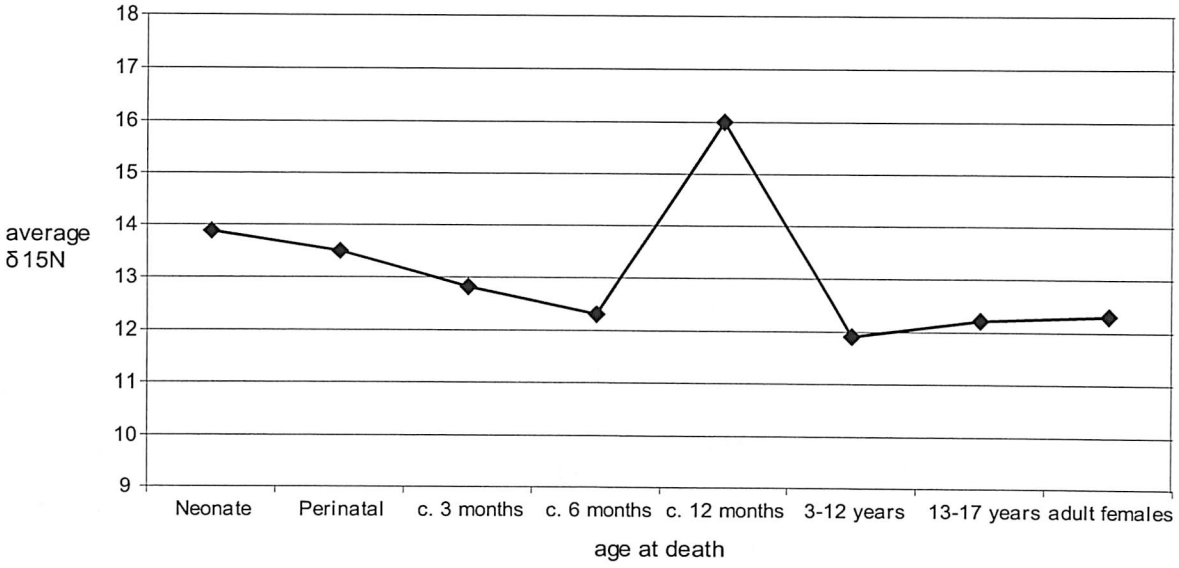


Fig. 3 — Variation in average δ15N with age at death of infants from Newark Bay.

Even these succumbed, presumably when weaned. By the age of three years all surviving children had been weaned. The trophic levels of all the infants analysed are above the levels of sheep or other herbivores indicating that the infants did receive some protein.

More than half of the infants, including some newborn, bore signs of severe bone pathology. Figure 4 shows the thickened cranial bones of a newborn infant. In other infants there are porosities of the orbits, parietals, cranial base, jaws and post cranial bones which are other signs of the failure of bone to remodel (Fig. 5).

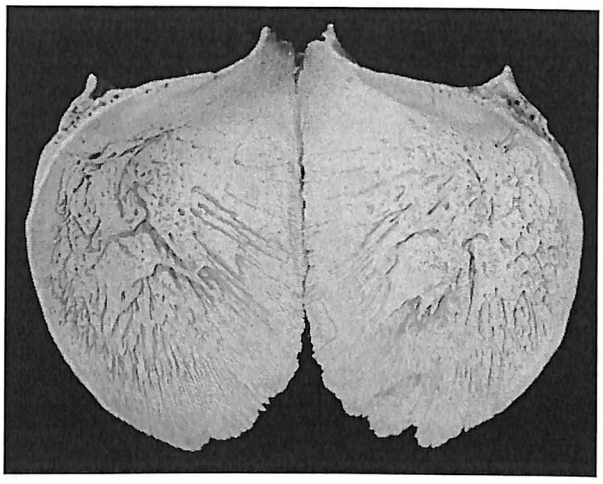
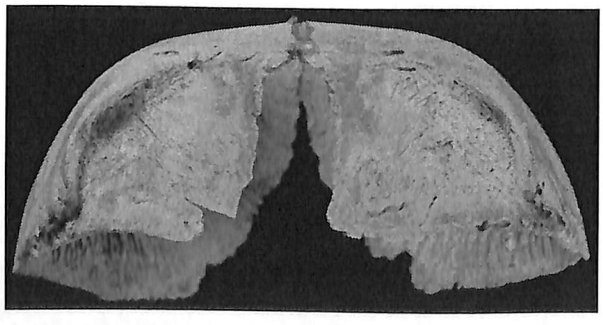
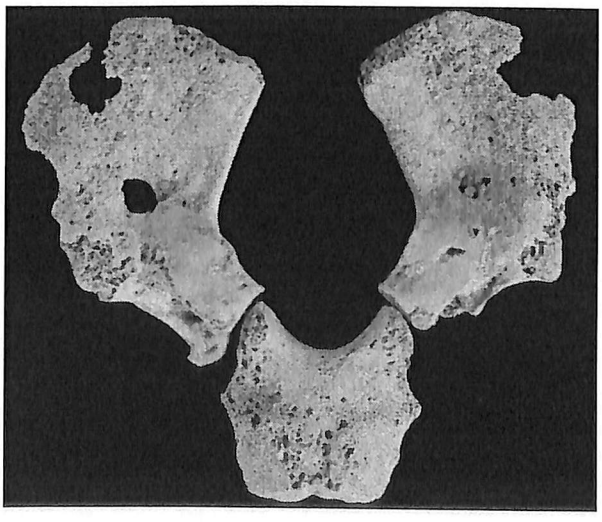


Fig. 4 — Frontal bones of newborn NB69.10A.



A



B

Fig. 5 — **A:** Orbits NB69.10A;
B: Cranial base NB69.63.

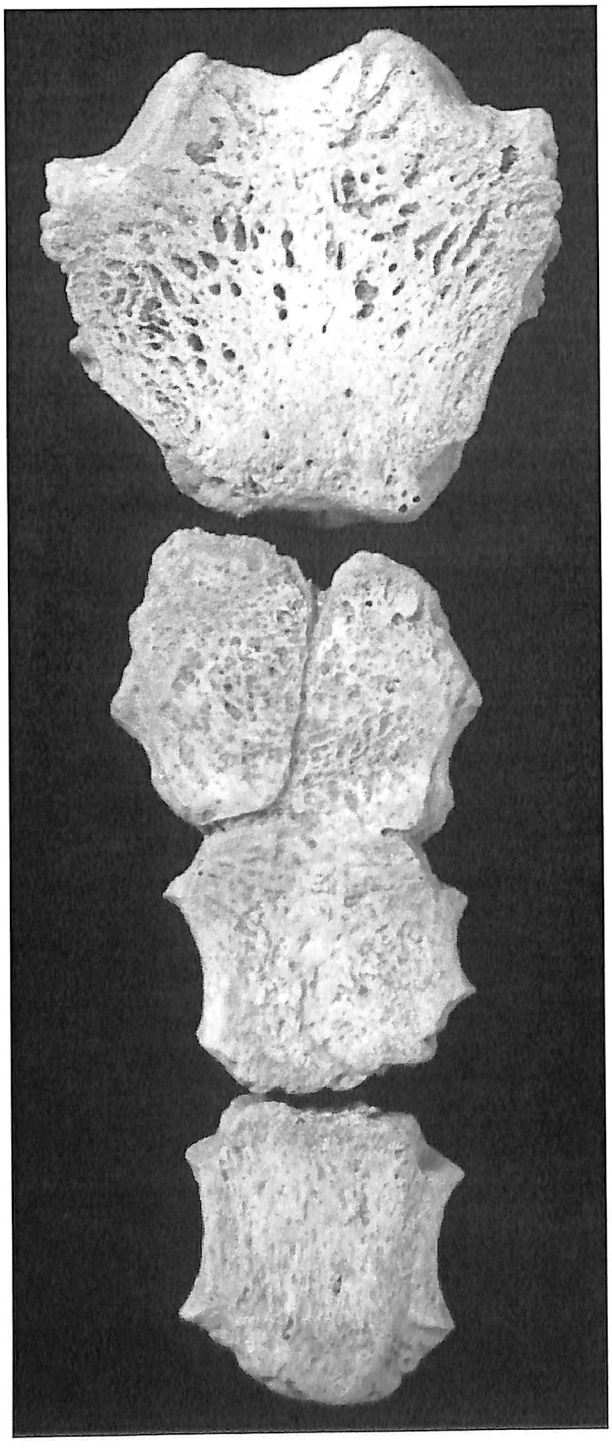
Two thirds of the adolescents presented with congenital anomalies including cleft cervical vertebrae (5 cases), bifid ribs (3 cases), posteriorisation of the lower spine *i. e.* six lumbar vertebrae or sacra with six vertebrae (4 cases) and splitting of sternal vertebrae (2 cases) (Fig. 6).

4. Discussion

The stable isotopes indicate that protein intake was probably adequate even for the very young. Trophic levels however only record protein source, they do not give the full dietary picture, and it is necessary to consider other reasons for the high infant mortality, the skeletal malformations and the bone pathology.

Generalized bone pathologies of infants have been attributed in the past to Barlow's disease, a condition of uncertain etiology (Dick, 1922; Molleson, 2005). Recent experimental work by See *et al.* (2008) suggests that the consequences of vitamin A deficiency should at least be considered in the diagnosis.

The role of vitamin A in bone development is well documented. Retinoic acid is the major form of vitamin A involved in gene expression and control of cellular differentia-



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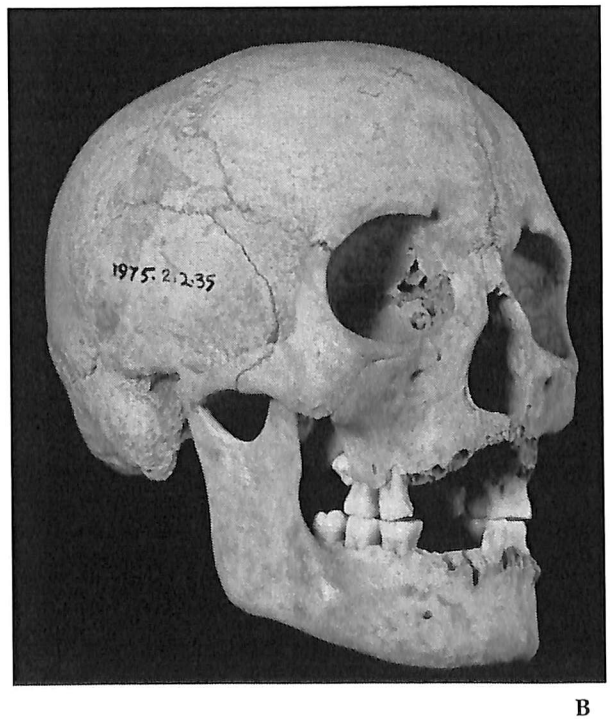

B

Fig. 6 — **A:** Sternum NB69.CC4;
B: Cranium of NB69.CC4 with signs of leprosy.

tion. There are specific binding sites on cellular nuclei, from which retinoic acid interacts with DNA and controls synthesis of proteins and gene expression (Barasi, 1997). In experimental conditions vitamin A deficiency leads to foetal deaths unless the deficiency is rectified (See *et al.*, 2008). Vitamin A deficiency in vulnerable populations might be seasonal and if of short duration the surviving foetus will bear any defects into post natal life. In the Newark Bay cemetery, we see the accumulation of anomalies over many generations.

Where there is vitamin A deficiency bone growth ceases in the cranium, the cranial cavity and spinal canal become too small for the central nervous system leading to paralysis, herniation of the brain into arachnoidal villi. Remodelling of the bone ceases and appositional bone formation continues at different rates in locations which vary with the species. Long bones are shorter and thicker. Replacement of cancellous bone by compact bone ceases; there is excessive deposition of periosteal bone secondary to reduced osteoclastic activity. Skeletal growth eventually ceases (Wolbach, 1947). There is anaemia despite adequate iron status; a poor immune system and no resistance to infection.

Many of the congenital anomalies of the spine ribs and sternum noted on the bones of adolescents have been induced experimentally in mouse foetuses on severely vitamin A deficient nutrition (See *et al.*, 2008). Usually severe vitamin A deficiency is lethal to the foetus. That the youths of Newark Bay survived to adolescence may indicate that the deficiency was of short duration during early foetal development. Retinol levels that support growth in non-pregnant animals do not in later stages of pregnancy. It has a short half life thus the stage of development when deficiency occurred is critical. Vitamin A plays a critical role in patterning the entire axial skeleton. Late foetal vitamin A deficiency leads to unique defects in the thoracic, sacral and pelvic regions of the developing skeleton (See *et al.*, 2008). It is these anomalies in the axial skeleton that have been noted in the Newark Bay bones.

The mechanism for the appearance of epigenetic traits is becoming clearer (Turner, 2009). At key regulator genes (*e.g.* Hoxb1, HoxbG) histone modifications are heritable *i. e.* passed through mitosis to later embryo stages. Modifying enzymes provide a potential interface through which the environment interacts with the genome. The modification may constitute a code that determines the complex changes in gene expression that drive early development (VerMilyea *et al.*, 2009). This is a long way from what we have seen at Newark Bay but an epigenetic adaptation to environmental stress seems probable. Retinoic acid regulates a set of vertebrate transcription factors (Acin-Perez *et al.*, 2010).

Famine in pregnancy is associated with miscarriages, premature and stillbirths (Stein *et al.*, 1975). While it is understandable that miscarriages may not have been buried in the cemetery, it is puzzling that so few foetal and only two premature deaths have been identified in the Newark Bay assemblage. However, not all the cemetery was excavated and the area where unbaptised infants were buried may not have been exposed. The proportion of babies who died at birth (17 %) is very high for a non-industrial society.

In medieval times the Orkney islands were ruled from Norway and it is something of a mystery how vitamin A deficiency could be prevalent when the Norse kept so many sheep. Stable isotope analyses have shown that there was animal protein in the diet of both men and women (Richards *et al.*, 2006). However, stable isotopes merely indicate protein sources in the diet. Neither quantities of protein nor other essential nutrients of vitamins or trace elements can be inferred.

Sources of vitamin A as retinol in meat liver, eggs, milk, cheese, fish oils and liver should have been available, although vitamin A is depleted in even these sources if the animal feed is deficient. Fish oils were

used for lighting but why no animal fat, eggs or cheese? There is no vitamin A in cod fish meat. Apparently they were not eating much mackerel or herring, or liver (offal) from domestic animals. Eggs of sea birds were seasonal and if stored loose much of their vitamin A content. Plant sources of carotene the vitamin A precursor would not have been readily available given the northerly location of Orkney. Some of the mothers at least must have been chronically deficient although vitamin A stores in the liver should last two or more years. A reliance on stored foods could be responsible for the frequency of abnormalities seen on the bones. Vitamins in particular diminish during storage.

Drummond and Wilbraham (1991: 77-78) drew attention to the restrictions inherent in many agricultural economies in medieval times. When the peasant was unable to get milk and eggs, there was a real danger that the shortage of animal fats meant a deficiency of the associated vitamins A and D. Vitamin A as β-carotene is found in all green vegetables but these foods contain very little fat and if insufficient green vegetables are eaten a proper intake of animal fat becomes essential in order to ensure a sufficiency of the vitamin. The amount of vitamin A in milk or egg yolk is dependent on the diet of the cows or hens. It can disappear if overwintered cattle are fed dry hay. Conditions arising from vitamin A deficiency are more prevalent in early spring and in cereal based diets.

Socially induced poverty is a further factor and there is evidence for this. The Orkneyinga Saga records how heavily Orkney farmers could be taxed by their Norse overlords (Pálsson & Edwards, 1981). The presence of leprosy, which is a disease of poverty, has been confirmed by DNA identification of the pathogen (Taylor *et al.*, 2000); while evidence for non accidental injury of at least one infant is also a symptom of poverty and impoverished quality of life (Molleson, 2005).

5. Conclusions

Cause of the poor infant survival due to malnutrition of mothers although protein intake was probably adequate other nutrients notably vitamin A were lacking. Restricted resources were the greatest limitation to this island community's survival.

Acknowledgements

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Author's address:

Theya MOLLESON
Department of Palaeontology
The Natural History Museum
SW7 5BD LONDON (United Kingdom)
t.molleson@nhm.ac.uk