**[SUPPLEMENTARY MATERIAL]**

**Nutritional deficiency and ecological stress in the Middle to Final Western Jomon**

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**Section 1: materials and methods of skeletal analysis**

**Materials and preservation**

*Ōta*

Thirty-two individuals over the age of 15 (defined here as adults) from Ōta were included in the palaeopathological analysis. While two neonates (the only subadults) were also present in the assemblage, their remains were too fragmented for inclusion in scurvy diagnosis. Therefore, subadult scurvy could not be identified at Ōta. The Ōta site, relatively dated by pottery chronologies to 5000–4000 BP, is a Jōmon shell midden site (Kiyono 1969; Shiomi *et al*. 1971). As a Middle Jōmon site, Ōta dates to a time period of climatic stability. Ōta is a shell midden, similar to sites where pit dwellings are found, and exhibited tools for both foraging and ritual purposes (Kiyono 1969; Temple 2018). As was the case with other Middle Jōmon sites, Ōta was likely a place of longstanding occupation, and a sedentary settlement (Kiyono 1969). Palaeodietary reconstruction of the Ōta diet using stable carbon and nitrogen isotopes indicates that food resources were predominantly terrestrial with between 6–13% reliance on marine resources (Kusaka *et al*. 2010, 2015). Marine resources were then likely an important protein source (Kusaka *et al*. 2010, 2015). Temple (2007) reported a linear enamel hypoplasia prevalence per individual of 63.2% and carious lesion rate of 3.8% at Ōta, suggesting high levels of systemic stress in Ōta individuals. However, their diet does not appear to consist of highly cariogenic foods such as roots, tubers and processed nuts (Temple 2018). Temple (2007) also reported a low prevalence of cribra orbitalia (15.7%) for Ōta.

The one adolescent from the Ōta assemblage (15–19 years) was fragmented, whereas over 50% of the post-adolescence adults were at least partially complete (after Buikstra & Ubelaker 1994). Three individuals had some skeletal elements which were charcoal black in colour. Most individuals exhibited grade 2 to 3 surface erosion (after McKinley 2004), with two individuals exhibiting severe rodent gnawing of the cranium and postcranium (grade 4 and 5).

*Tsukumo*

Thirty individuals (8 subadults (<15 years), 22 adults (>15 years)) from Tsukumo were included in the analysis. Tsukumo, like Ōta, is classified as a shell midden site (Kiyono 1969). Relative dating places this site in the Late to Final Jōmon Period (4000–2300 BP) during a period of climatic cooling (Kiyono 1969). The Tsukumo assemblage is comprised of predominantly adults. However, there is a greater number of preserved subadults than was the case for Ōta. A distinct lack of infants under 1 year of age and older children (6–10 year olds) does suggest some collection bias where subadults, unless very well preserved, may not have been identified during excavation. While similar to Ōta in regards to sedentary dwellings along the Inland Sea, and similar foraging and fishing tools were excavated from the shell midden (Kiyono *et al*. 1920), Tsukumo does have some distinct differences which indicate considerable social change from the Middle to the Late and Final Jōmon Periods in this region. Kiyono (1969) identified fashioned deer antlers placed along the right hip of three individuals at Tsukumo, which were described as hip ornamentsand animal bone implements were found in 33% of graves. These were not found at Ōta. Temple (2018) has argued that these artefacts indicate social differentiation in identity at Tsukumo. While there remains no evidence for socioeconomic inequality, this may indicate the beginnings of social stratification in the Late to Final Jōmon in western Japan (Temple 2018). In regards to the Tsukumo diet, Kusaka *et al*. (2010) found no differences in the carbon and nitrogen isotope values between males and females, contrasting the findings at Ōta. Tsukumo individuals also exhibited significantly lower nitrogen values than Ōta individuals suggesting an increased proportion of terrestrial sources in the Tsukumo diet. The Tsukumo assemblage exhibited a carious lesion prevalence rate of 9.6%, considerably higher than that of Ōta (Temple 2007). The increased rate of carious lesions of Late and Final Jōmon compared to Middle Period Jōmon may be related to an increased carbohydrate intake supported by the isotopic findings of Kusaka *et al*. (2010) for an increased reliance on terrestrial food resources. The prevalence of linear enamel hypoplasia per individual was similar to that of Ōta at 66.7% in the Tsukumo assemblage (Temple 2007).

Over 68% of the adults in the assemblage were complete (more than 75%) whereas only 27% of subadults were complete (after Buikstra & Ubelaker 1994). Forty-five percent of the subadult assemblage were incomplete or fragmented, and epiphyses were poorly preserved. Individuals exhibited grade 1 to 3 surface erosion (after McKinley 2004).

**Table S1: Dietary sources of the Jōmon during summer/ spring and winter/autumn periods (Habu 2004). (<6.5 to 10mg a day results in Vitamin C deficiency). Vitamin C estimates: (US Department of Agriculture 2019).**

|  |  |  |  |
| --- | --- | --- | --- |
| **Winter/Autumn** | | **Spring/Summer** | |
| ***Food Type*** | ***Vitamin C (mg) per 100g*** | ***Food Type*** | ***Vitamin C (mg) per 100g*** |
| *Acorns* | 0 | Mulberry Fruit | 36.4 |
| *Chestnuts* | 40.2 | Gourd | 8.5 |
| *Walnuts* | 1.3 | Yam | 17.1 |
| *Boar* | 0.6 | Mussel/ shellfish | 13.6 |
| *Venison* | 0 | Fish | 3.6 |
| *Fish* | 3.6 | Tuber (Sweet Potato) | 2.4 |
|  |  | Taro | 5 |

**Age-at-death and sex estimation**

*Subadult age-at-death estimation*

We applied the Ubelaker (1987) dental eruption standard for subadults with erupted teeth. Where loose teeth were available, the Moorrees *et al*. (1963) calcification standards for dental formation were also applied. We compared long bone lengths of individuals with dental eruption or calcification scores to those without dentition to estimate the ages of post-infant subadults without preserved teeth. Timing of epiphyseal fusion served as a primary form of age estimation in individuals over the age of approximately 12 years to early adulthood following Schaefer *et al*. (2009).

*Adult age-at-death estimation*

We estimated adult age using standards for the pelvis (Lovejoy *et al*. 1985; Brooks & Suchey 1990). We recorded Scott’s (1979)dental attritionscores for all adult individuals with molars present. Individuals within each assemblage were seriated based on their degree of tooth wear. We compared relative tooth wear scores between those with predetermined age categories from the auricular surface and pubic symphysis regions to those where the pelvis was not preserved. Tooth wear seriation age estimation was only used where the other age estimation methods were not available.

*Sex estimation*

We estimated sex in individuals over the age of 15 years only. Both cranial and pelvic morphological assessments were performed, with pelvic estimates given priority to estimation. The Phenice method (1969) was considered the most reliable estimate and took precedence over other methods and was supplemented with scoring of the greater sciatic and pre-auricular sulcus following standards outlined in Buikstra and Ubelaker (1994). For cranial estimation, we employed the method of Walrath and colleagues (2004).

**Section 2: differential diagnosis of lesions**

*Lesion distribution and radiographic observations*

Forty-seven percent (15/32) of the Ōta assemblage, all adults, exhibited subperiosteal new bone (SPNB) deposits. SPNB deposits were present on both the crania and postcrania. However, these lesions were predominantly unilateral and present on long bones, particularly the anterior tibiae. Most lesions, both cranial and postcranial, were discrete and remodelled with 20% (3/15) of individuals with SPNB exhibiting diffuse lesions across more than two skeletal elements. These three individuals also exhibited diffuse SPNB deposits across multiple internal and external ribs. Cranial lesions were also commonly symmetrical and accompanied with abnormal cortical porosity.

All Tsukumo individuals, both adults and subadults, exhibited at least one osteoblastic lesion. While some individuals exhibited diffuse deposits of SPNB across the long bones, the lesions were overwhelmingly symmetrical and discrete deposits of SPNB across the crania and postcrania that were often directly associated with abnormal cortical porosity, similar to those at Ōta. Bands of radiolucent metaphyses and radiodense metaphyseal lines were observed in the long bone radiographs of three subadults aged between approximately 2 to 3 years (38% (3/8) of subadults). Generalised osteopenia—low bone density—was radiographically observed in all subadults.

*Traditional differential diagnosis*

Certain nutritional diseases, congenital disorders, infections and trauma can cause systemic subperiosteal new bone lesions in adults and subadults such as those observed in the Jōmon samples and are therefore considered in the differential diagnosis. Rickets is a demineralisation disorder of childhood which results from an imbalance of vitamin D, calcium, phosphorus and parathyroid hormones (Klein & Simmons 1993; Wharton & Bishop 2003). Having a genetic or nutritional basis, the disease can lead to deposition of newly mineralised osteoid deposits on the shafts of the bone during recovery (Brickley *et al*. 2020). The disease is most characteristic for causing long bone bending, cupping, fraying, porosity and flaring and swelling of metaphyseal plates. Only one child, from Tsukumo, exhibited mild cupping of the proximal tibiae. However, this bone change can also occur in scurvy (Brickley *et al*. 2020). Therefore, while rickets must be considered wherever childhood nutritional diseases such as scurvy are suspected (Lewis 2017), it can be ruled out as a cause for the subadult pathology in the Jōmon samples. Infantile cortical hyperostosis (Caffey’s disease), a disease of unknown aetiology which causes systemic new bone production, can be ruled out as all affected subadults were over 1 year of age (Kamoun-Goldrat & Le Merrer 2008). Pellagra is a nutritional disease caused by niacin and tryptophan deficiency causes new bone deposits on long bones. The bone changes of pellagra are not well described in the palaeopathological literature but do not appear to be distinctly discrete and associated with cortical porosity as observed in the Jōmon assemblages (Paine & Brenton 2006). Infection as a primary cause of the lesions is also unlikely due to the discrete and symmetrical pattern of the new bone, rather than diffuse and non-symmetrical patterning in systemic infection (Vlok *et al*. 2020). It is possible that many SPNB lesions observed in Ōta are a result of trauma or localised infection, as they are unilateral and isolated suggesting a single event inducing an inflammatory bone response. These lesions are not diagnostic and can only be considered as a consequence of non-specific stress (Weston 2008, 2012). However, the multiple symmetrical and discrete deposits of new bone with cortical porosity and vascular impressions, particularly in the crania observed in Ōta and Tsukumo, are not consistent with traumatic injury (e.g. accidental or interpersonal) as multiple episodes of symmetrical microtrauma are necessary for such a lesion pattern to occur (Snoddy *et al*. 2017; Brickley *et al*. 2020). The consistent distribution observed in the children with the absence of healed fractures also rules out the likelihood of repeated child abuse (Kemp *et al*. 2008; Snoddy *et al*. 2017).

Anaemia can cause marrow hyperplasia and cortical thinning (rarefaction) resulting in deep porosity of the skull (porotic hyperostosis) and the orbits (cribra orbitalia), which may sometimes overlap with lesions observed in scurvy (Klaus 2017). The only sign of anaemia in subadults were mild cribra orbitalia in one adolescent, but anaemia is also a common co-morbidity of scurvy due to internal blood loss and knock-on effects to folate metabolism, so the presence of cribra orbitalia does not negate a scurvy diagnosis (Cox *et al*. 1967; Khalife *et al*. 2019). Overall, skeletal expression described above in both of the adults and subadults are most likely due to haemorrhaging as a consequence of muscle movement and multiple episodes of vessel rupture in association with habitually used muscles in scurvy. The Snoddy *et al*. (2018) criteria is applied below in consideration of the biological basis of inflammatory lesions, cortical porosity and growth disruptions which occur in scurvy.

*Diagnosis following the Snoddy* et al*. (2018) criteria*

The Snoddy *et al.* (2018) criteria requires a case to present with at minimum two or more *diagnostic* lesions to be considered a probable case. A possible case fails to meet this criteria but presents with one *diagnostic* lesion and/or two or more *suggestive* lesions. Diagnostic lesions are provided in Snoddy *et al.* (2018), and are lesions that have been well demonstrated in clinical and palaeopathological literature to be associated with cases of scurvy, *and* are anatomically intuitive. Suggestive lesions are lesions that have been associated with scurvy cases, but are yet to be intensively researched to support the claims they are associated with scurvy specifically.

Ōta adults

Discrete symmetrical SPNB and abnormal porosity on the cranium were the most common diagnostic lesions exhibited. These lesions presented symmetrically on the orbital roofs, anterior and posterior zygomatic bones, anterior and posterior maxillae, lateral greater wings and pterygoid processes of the sphenoid bones, and the medial coronoid processes and mylohyoid lines of the mandibles (Figure 3; Table S2). Vascular impressions were infrequently observed in association with these lesions. Possible and probable cases of scurvy were identified predominantly in adults between 30 and 50 years of age (Table 1). Two individuals exhibited abnormal cortical porosity without SPNB deposits.

Tsukumo subadults

Macroscopic lesions diagnostic for scurvy consisted of bilateral and symmetrical SPNB deposits associated with abnormal cortical porosity and sometimes vascular impressions on the sphenoid bones, temporal bones, maxillae, zygomatic bones, mandibles, and superior orbits (Table S2). Abnormal endochondral porosity exceeding 10mm from the metaphyseal plates of long bones was present in these six individuals supporting a diagnosis of probable scurvy. The radiographic signs observed are consistent with white lines of Fraenkel in combination with Trümmerfeld zones (Figure 2).

Tsukumo adults

Discrete bilateral and symmetrical SPNB were present on both the crania and postcrania. As with Tsukumo subadults, SPNB deposits and/or abnormal cortical porosity was present symmetrically on the sphenoid bones, temporal bones, maxillae and mandibles of adults (Figure 3; Table S2).

**Section 3: statistical methods and analyses**

Parameters for logistic regression in StatsDirect v3.0

Response data: individual

Precision of estimates: 0.0000001

Calculate intercepts: yes

Parameters for sample size analysis in StatsDirect v3.0

Correlation coefficient under null hypothesis: 0

Correlation coefficient under alternative hypothesis: 0.5

Power: 90%

Alpha: 5%

**Table S3. Odds ratios (ORs) of scurvy across sites (adults only)**

|  |  |  |  |
| --- | --- | --- | --- |
| Sample  Exposed group: Tsukumo (≥15 years)  Control group: Ota (≥15 years) | OR | P-value | 95%CI |
|
|  |  |  |  |
| *Probable only* | **10.67** | **0.0006** | **2.75 – 41.43** |

**Table S4. !!help!-> 1043 <-!help!! !!redo!-> "UnivariateSummary"  <-!redo!! Descriptive statistics for Ota**

|  |  |  |
| --- | --- | --- |
| **Variables** | **Age at death** | **Probable scurvy** |
| ***Valid data*** | **25** | **25** |
| ***Missing data*** | **0** | **0** |
| ***Sum*** | **916** | **5** |
| ***Mean*** | **36.64** | **0.2** |
| ***Variance*** | **91.656667** | **0.166667** |
| ***Standard deviation*** | **9.573749** | **0.408248** |
| ***Variance coefficient*** | **0.261292** | **2.041241** |
| ***Standard error of mean*** | **1.91475** | **0.08165** |
| ***Upper 95% CL of mean*** | **40.591849** | **0.368517** |
| ***Lower 95% CL of mean*** | **32.688151** | **0.031483** |
| ***Geometric mean*** | **35.390281** | **\*** |
| ***Skewness*** | **0.167967** | **1.5** |
| ***Kurtosis*** | **2.451232** | **3.25** |

**Table S5. Descriptive statistics for Tsukumo**

|  |  |  |
| --- | --- | --- |
| **Variables** | **Age at death** | **Probable scurvy** |
| ***Valid data*** | **22** | **22** |
| ***Missing data*** | **0** | **0** |
| ***Sum*** | **634** | **16** |
| ***Mean*** | **28.818182** | **0.727273** |
| ***Variance*** | **100.251082** | **0.207792** |
| ***Standard deviation*** | **10.012546** | **0.455842** |
| ***Variance coefficient*** | **0.347439** | **0.626783** |
| ***Standard error of mean*** | **2.134682** | **0.097186** |
| ***Upper 95% CL of mean*** | **33.257496** | **0.929382** |
| ***Lower 95% CL of mean*** | **24.378868** | **0.525164** |
| ***Geometric mean*** | **27.217171** | **\*** |
| ***Skewness*** | **0.591345** | **-1.020621** |
| ***Kurtosis*** | **2.511344** | **2.041667** |

**Table S6. Pearson residual for logistic regression**

|  |  |  |  |
| --- | --- | --- | --- |
| **Index** | **Pearson residual** | **Leverage** | **Std Pearson residual** |
| **1** | **-0.445945** | **0.096197** | **-0.469077** |
| **2** | **2.117585** | **0.054501** | **2.177761** |
| **3** | **-0.475251** | **0.051429** | **-0.487965** |
| **4** | **-0.448792** | **0.090424** | **-0.470572** |
| **5** | **-0.663605** | **0.115818** | **-0.705731** |
| **6** | **3.507937** | **0.12181** | **3.743325** |
| **7** | **-0.685064** | **0.08866** | **-0.717614** |
| **8** | **1.128679** | **0.092669** | **1.184917** |
| **9** | **-0.86616** | **0.120361** | **-0.923519** |
| **10** | **-0.536345** | **0.069768** | **-0.556094** |
| **11** | **-0.532942** | **0.06462** | **-0.551043** |
| **12** | **-0.911406** | **0.167619** | **-0.998966** |
| **13** | **-0.730085** | **0.09296** | **-0.766585** |
| **14** | **-0.793065** | **0.242292** | **-0.911083** |
| **15** | **-0.636418** | **0.133181** | **-0.683563** |
| **16** | **0.406371** | **0.246818** | **0.468245** |
| **17** | **0.618401** | **0.046414** | **0.633272** |
| **18** | **0.614477** | **0.045749** | **0.629035** |
| **19** | **-0.251054** | **0.137981** | **-0.270402** |
| **20** | **0.986042** | **0.228871** | **1.122876** |
| **21** | **-0.587378** | **0.197439** | **-0.65566** |
| **22** | **0.534186** | **0.147985** | **0.57872** |
| **23** | **-1.507729** | **0.091055** | **-1.581446** |
| **24** | **0.638398** | **0.057968** | **0.657747** |
| **25** | **-1.73435** | **0.066608** | **-1.795166** |
| **26** | **0.541029** | **0.130803** | **0.580312** |

*Figure S1. Residuals plot for logistic regression analysis*

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