FEMALE BONE PHYSIOLOGY RESILIENCE IN 750-300 BP 'POLYNESIAN OUTLIER' COMMUNITY

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ABSTRACT

Remodelling is a fundamental biological process involved in the maintenance of bone physiology and function. We know that a range of health and lifestyle factors can impact this process in living and past societies, but there is a notable gap in bone remodelling data for populations from the Pacific Islands. We conducted the first examination of femoral cortical histology in n = 69 individuals from 750 – 300 BP Taumako in Solomon Islands, a remote 'Polynesian Outlier' island in Melanesia. We tested whether bone remodelling indicators differed between age-at-death groups, and biological sex validated using ancient DNA. Bone vascular canal and osteon size, vascular porosity, and localised osteon densities, corrected by femoral robusticity indices were examined. Females had statistically significantly higher vascular porosities when compared to males, but osteon densities and ratios of canal-to-osteon (~10%) did not differ between the sexes. Compared to males, the femora of Taumako females experienced higher frequencies of remodelling events, which mirrors bone health paradigms through the life-course today. However, contrary to modern populations, female femoral bone tissue did not decline with age. This matches findings in other archaeological samples, and is testament to ancient female bone physiology resilience also in the Pacific region.

KEYWORDS

Melanesia; Pacific; osteon; Haversian; vascularity; histomorphometry; bone remodelling

INTRODUCTION

Peak bone mass attainment in modern humans occurs around the third life decade and is marked by a striking sex-specific difference whereby biological females (hereafter 'females') typically accrue less bone than biological males (hereafter 'males')[1-3]. Bone density becomes further compromised around the fifth-sixth life decade when females experience menopause and a significant reduction in the osteoclast inhibiting estrogen[4-6]. The physiological maintenance of bone throughout the life-course is executed by remodelling, a process sensitive to a range of internal and external stimuli[7]. Bioarchaeological research on human skeletal remains with well-preserved bone microstructure has provided data on bone remodelling under a range of cultural and environmental conditions[8-12]. However, there is a notable gap in data for past populations from across the Pacific Islands, except for two recent small sample size-based studies that did not systematically asses Haversian bone histology parameters (n = 8 from Tonga[13]; and n = 1 from Marshall Islands[14]). Here, we report the first such data for an archaeological 'Polynesian Outlier' skeletal assemblage from a ca. 750 – 300 BP site on Taumako, Southeast Solomon Islands[15-17] (Figure 1).

[Insert Fig. 1]

We focus on this region because Oceania has been subject to complex migration histories[18]. Taumako Island in Melanesia is known as a 'Polynesian Outlier' because of a blow-back migration of people from Polynesia during the mid-second millennium AD. A notoriously high incidence of metabolic and infectious conditions is widespread across the Pacific Islands, evidence for which is available for both past and present human populations[19,20]. High frequencies of specific and non-specific indicators of disease have also been reported in the Taumako sample[21,22]. As such, the Taumako bone remodelling capacities should reflect endemic environmental and disease insults. Not only will our data add new insights into current knowledge of ancient human bone physiology across Pacific Island habitats, they will also expand our understanding of bone growth dynamics within spatially and temporally

distributed populations. Further, the Asia-Pacific region is of increasing interest to the International Osteoporosis Foundation, which is currently mapping the occurrence of fractures across the region[23]. To that end, research on contemporary humans will benefit from our data by identifying the range and nature of bone remodelling responses at a population level.

The importance of bone remodelling through human life-course

Based on bone mineral density (BMD) and fracture incidence data, it is well established that significant bone loss occurs with age[24,25]. Bone building capacities in early adulthood play a key role in determining the rate at which bone metabolic activity becomes out of balance later in life[2]. While early life skeletal mass accrual is largely genetically determined, other factors such as physical activity, diet, and lifestyle habits, can also impact bone metabolism[12,26]. There are three key areas that characterise bone mass change in modern humans – peak bone mass accrual in the third life decade, drastic bone loss after menopause in females, and significant bone loss in both sexes in old age[2]. The first three ontogenetic decades are spent creating a 'bone bank' that is used for the remainder of the life course[27]. The female preponderance of bone loss is due to life-course variability in estrogen levels which inhibit prolonged bone resorption[4]. The effect of menopause on bone health can be mediated through lifestyle factors and calcium supplements available to women today. Modern clinical techniques can diagnose osteoporotic bone from BMD T-scores[28] and bone remodelling histological markers to check whether osteoclast-mediated bone resorption outweighs bone deposition by osteoblasts.

While BMD has been previously examined in some archaeological samples (see reviews in[8-12,29]), histological characteristics of cortical remodelling assessed from thin sections by

105 histomorphometric and histomorphological methods have also been successfully
106 reconstructed[30,31]. Cortical bone not only experiences metabolic turnover events that ensure
107 suitable calcium reservoirs, it also responds to biomechanical stimuli that drive bone cell
108 activity[7]. As teams of osteoblasts and osteoclasts execute bone remodelling as part of Bone
109 Multicellular Units (BMUs) that travel through the cortex, they leave behind remodelling
110 products of circular structure – secondary osteons - that can be studied histologically, and
111 thus offer an insight into bone remodelling activity in an individual[32]. The area of secondary
112 osteons and Haversian canals within these can aid in determining whether a typical BMU
113 completes infilling of bone relatively quickly or slowly[33-35]. Secondary osteons are also
114 replaced by subsequent generations of secondary osteons, creating a total population of
115 remodeled bone per given region[36], whereas the densities of vascular pores (Haversian canals,
116 Volkmann's canals, primary/simple vessels) reflect the complex interconnected network
117 cortical bone uses to circulate blood and interstitial fluid containing oxygen and nutrients
118 important for bone homeostasis[37].
119
120 **Bone remodelling in archaeological humans**
121 In cases of archaeological human bone that is well preserved microstructurally, several
122 studies have been able to reconstruct bone remodelling capacities and link them to aspects
123 such as gender division of labour and sex-specific bone remodelling[38], changes in subsistence
124 strategies through time[39], or medieval lifestyles associated with socio-economic disparities[40].
125 For example, Mulhern and Van Gerven[38] found higher secondary osteon densities in femoral
126 cross-sections of males than females from Medieval Sudanese Nubia, but no sex differences
127 in Haversian canal dimensions, suggesting sex-specific activities with physically strenuous
128 tasks of males contributing to the observed remodelling patterns. Miszkiewicz et al[13] found
129 severely porous Haversian bone in adult females compared to denser bone samples of males

130 from 2,650 BP Tonga, indicating experiences of abnormal bone loss likely relayed to both
131 age and activity. However, bioarchaeological studies where bone remodelling has been
132 investigated through histological means have also cautioned that we do not yet fully
133 understand the spectrum of bone histology parameters manifested in archaeological
134 samples[41], and that relying on very specific interpretations (e.g. behaviour) from histological
135 structures is clouded by multiple other confounding variables[42] such as health, nutrition,
136 ancestry and individual or population-based variations in metabolic activity. Therefore,
137 interpretations of archaeological human bone histology data are usually context specific.
138 However, with an increasing number of sites/collections reported, we may be able to start
139 building a better understanding of possible changes in bone remodelling through time and
140 space in recent humans. For example, one prior analysis comparing tibial and femoral bone
141 histology between Pleistocene specimens (including Broken Hill, Shanidar 2, 3, 4, 5, 6,
142 Tabun 1, and Skhul 3, 6, 7) and a pre-Columbian Pecos human sample, reported similar
143 levels of bone remodelling characterising the two[43], but smaller size of osteonal structures in
144 the Pleistocene sample[44].
145
146 As bone histology research using archaeological samples gathers increasing amounts of data,
147 it is apparent that a significant gap remains for populations from across the Asia-Pacific
148 region. While access to large samples of human remains is limited in the remote areas of the
149 Pacific, excavation at the Taumako Island site in Southeast Solomon Islands previously
150 produced one of the largest well-preserved skeletal samples in the region that offers an
151 excellent opportunity to investigate bone remodelling[15-17,21].
152
153 Modern Pacific Island nations are impacted by widespread metabolic syndrome related
154 conditions, including type 2 diabetes and obesity[19]. Archaeological evidence demonstrates the

155 occurrence of gout and diffuse idiopathic skeletal hyperostosis, as well as infectious and
156 nutritional conditions affecting health from the time of first settlement ca 3,000 BP in Remote
157 Oceania (the islands east of the Solomons chain)[19,45-47]. Island environments are associated
158 with food shortages, climate and environmental instability, affecting health in the past and
159 today[19,45,48]. Ancient humans from this geographical area, therefore, might have developed a
160 suite of bone physiological adaptations to withstand endemic disease and environmental
161 insults. Indeed, one prior study including the examination of 61 Taumako individuals
162 recorded cortical bone indices of the metacarpal and femur, in addition to femur length to
163 find that no distinct stress or functional adaptation signal could be detected specific to island
164 environments[49]. However, whether bone from Taumako individuals possibly experienced
165 metabolic fluctuations in this context has not been specifically tested.
166
167 Given the significance of archaeological human samples in improving modern bone biology
168 research, the Pacific Island gap in our knowledge relating to archaeological bone
169 remodelling, and the island environmental context of Solomon Islands, this study tested
170 whether (1) Taumako males and females remodel femoral bone differently, and (2) to what
171 extent Taumako femur remodelling changes with age. Our total sample size was n = 69 (33
172 males and 36 females). We selected the femur because of its biomechanical versatility
173 reflecting sex-specific lifestyles and sexual dimorphism, which we firstly evaluated in this
174 sample through basic gross measures of femoral size (midshaft circumference, cortical width,
175 maximum length[49]) and robusticity indices computed from these values. Next, we measured
176 standard static histological variables (vascular canal and osteon size to reconstruct bone
177 tissue-canal ratios, vascular porosity, and localised osteon density[36]) as proxies for bone
178 remodelling (Figure 2), which we correlated against one another to check whether they are in
179 mutual agreement and thus can be independently used as markers of remodelling. The

180 femoral size data were then used to adjust histology data to account for microscopic-
181 macroscopic scaling issues.
182 [Insert Fig. 2 here]
183 We hypothesised there should be significant differences in bone remodelling markers with
184 age and sex, with females showing higher remodelling with more resorption than bone
185 deposition, and older individuals experiencing higher bone resorption than those of younger
186 age. Our sex estimates are based upon standard gross anatomy methods[50] validated by
187 determining XY or XX karyotypes via ancient DNA (aDNA), yielding 88% of successful sex
188 matching through these two approaches. This study treats sex as a biological trait and does
189 not consider gender identity which is unknown for these Taumako individuals.
190
191 **RESULTS**
192 The Taumako males had ($p < 0.001$, Tables 1-3) larger femoral midshafts and thicker
193 posterior cortical walls (average circumference = 95.79 mm, average cortical width = 10.77
194 mm) compared to females (average circumference = 89.81 mm, average cortical width = 8.77
195 mm). Expectedly, females also had shorter femora than males, though this difference was not
196 tested statistically due to a small sub-sample size of the individuals with intact femora (n =
197 23). Robusticity indices calculated based upon midshaft circumference (22.85) and cortical
198 width (2.58) were descriptively greater in males when compared to females (midshaft
199 circumference = 20.66, cortical width = 2.08), but could not be validated statistically either
200 because they were based on the limited length data. We could not conduct age related
201 inferential analyses on the gross morphometric femoral data, except for circumference and
202 cortical width, which did not change statistically across any of the age-at-death classes in the
203 whole sample ($p > 0.05$, Table 1). Within females and males, there was no age effect on

midshaft circumference or cortical width either. Given the clear sexual dimorphism in the Taumako femora, adjustments of bone histology data by femoral size were justified[50,51].

The histology correlations returned three statistically significant and strong relationships (Table 3, Figure 3) for the area of osteons and their Haversian canals (positive), osteon area and population density (negative), and vascular porosity and osteon population density (positive). This means that we can be confident in using individual variables as reflective of bone remodelling activity across the sample with the size of osteons and canals increasing alongside one another, higher osteon densities being associated with smaller osteons (possibly strain suppressed), and higher porosities also corresponding to higher osteon densities.

[Insert Fig. 3 here]

Trends in bone remodelling at Taumako

Firstly, descriptively, all three histology variables were greater in females when compared to males (Table 2). Out of all three, vascular porosity adjusted by both cortical width (average = 2.34) and midshaft circumference (average = 22.02) were statistically significantly higher ($p < 0.0001$) in females than males (average vascular porosity adjusted by cortical width = 1.64, average vascular porosity adjusted by midshaft circumference = 18.34) (Table 3, Figure 4). However, the vascular canal-osteon ratios did not differ statistically between the sexes ($p > 0.05$, Tables 2, 3), with both sub-groups bordering an approximate 10% of Haversian canal relation to secondary osteon area (averages of 7.61% in males and 8.31% in females). We did not attempt an inferential statistical comparison of the osteon density data, and on further sub-divisions by age-at-death due to inadequately small sample size in the sub-groups (Table 2). Secondly, descriptively, there was a clear change in bone histology values from young to old individuals whereby all histological markers of remodelling peaked at the middle age

category (Tables 2, 3, Figure 4). While all histology data were lower in the young and old age sub-groups when compared to the middle-age sub-group, the old individuals showed the lowest values across the entire sample with the exception of canal-osteon ratios which were slightly higher in the older individuals. However, none of these descriptively apparent changes with age-at-death were statistically significant ($p > 0.05$) (Table 3). As above, we did not attempt an inferential statistical comparison of the osteon density data, and on further sub-divisions by sex due to inadequately small sample size in the sub-groups (Table 2). Collectively, our results only partly support our hypothesised expectations.

DISCUSSION

Our age-at-death and sex analyses of the Taumako bone histology data revealed that, overall, the Taumako females had higher vascular porosity of their femoral cortical bone compared to males, while variables such as osteon densities and canal-to-osteon area ratios did not differ significantly when compared between the sexes. This occurred despite males and females having sexually dimorphic femora at Taumako. A possible isometric effect of larger male femur size on bone histology can be excluded as underlying these results as our data were corrected by femoral midshaft size measures and robusticity indices[51].

Sex-specific trends in cortical bone remodelling

The femoral samples of Taumako females were more vascularised than those of males, which suggests experiences of higher frequencies of intra-cortical remodelling events in the females, agreeing with sex-specific bone remodelling through the life-course[52,53]. We do note, however, that our data for the cortical bone porosity are made up of Haversian canals with the possibility of including some primary vessels. As such, our measure of porosity is that of an accumulation of vascular cavities up until the point of death, rather than just reflecting recent

254 remodelling events, and we cannot be sure which canals had been replaced in the first few
255 life decades in these individuals. Further, despite the greater density of cortical pores in
256 females, neither the secondary osteon population density nor the geometric properties of
257 secondary osteons differed statistically between the sexes. In fact, the ratio relationship
258 between Haversian canals and osteon area was almost the same when comparing the sexes
259 (approximately 10%). We expected higher Haversian canal area in females than males
260 indicating prolonged osteoclasts-mediated bone resorption. This suggests that at least the
261 intra-cortical femoral bone at midshaft in the Taumako males and females experienced
262 similar remodelling events. Considering that the vascular porosity data include sub-periosteal
263 and sub-endosteal bone regions, they appeared to contribute to the overall vascularity
264 difference between the sexes, which was partly our hypothesised expectation.
265
266 Prior bioarchaeological research reported inconsistencies in osteonal morphometry between
267 the sexes similar to those we present for Taumako. For example, data for males in the
268 Medieval Sudanese sample introduced earlier showed higher osteonal densities than females,
269 but females had larger osteons than males[38]. However, similar to us, Mulhern and Van
270 Gerven[38] reported a lack of statistically significant differences in the geometric properties of
271 Haversian canals between the sexes. Similarly, 14th-19th centuries Pecos females (New
272 Mexico) had large secondary osteons, but with smaller Haversian canals when compared to
273 males[54]. Burr et al.[54] observed a lack of distinct bone loss in the Pecos females, citing a
274 physically active lifestyle as a possible factor driving the maintenance of good bone density.
275 In the 700 BC to 19th century Canadian Baffin Island male and female skeletons, no
276 significant differences were noted when considering the density of Haversian canals and the
277 area of secondary osteons[55]. There is a possibility that some of these results are underlain by
278 shorter longevity in the past compared to today, whereby ancient females might not have

279 regularly survived to post-menopause age. Nevertheless, as previously noted by Pfeiffer[41],
280 there is clearly variability in how bone histology is expressed in archaeological and modern
281 populations, complicating inter-population comparisons. This suggests that a population-
282 specific approach needs to be taken to provide possible explanations of our results.
283
284 Outside of a genetic basis to the effective age of adult compact bone which we cannot
285 validate, alternative interpretations for the Taumako intra-cortical bone remodelling
286 capacities may include some effects of social status, increased male frailty, and/or bone
287 density adaptation shielding from excessive calcium loss in females. Indeed, in previous
288 studies, diet did not greatly differentiate between Taumako males and females[17], with both
289 sexes likely consuming foods from a similar trophic level. This is something we might see
290 reflected in the lack of significant differences of intra-cortical bone remodelling in our study.
291 However, Kinaston et al.[17] also reported 20% of wealthy Taumako individuals, who were
292 predominantly male, to be associated with a consumption of high status-specific foods (such
293 as higher trophic level animals including pig, fish, and turtle). This means that social status
294 may play some role in our results. Prior research comparing high and low social status human
295 bone histology in medieval Canterbury, England noted a negative effect of upper-class foods
296 (e.g. increased consumption of protein) on bone remodelling, adversely affecting males in
297 particular[40,56]. As lifestyle is closely tied with social status, the lack of intra-cortical bone
298 remodelling differences between the sexes could point towards Taumako males engaging in
299 more residential and sedentary lifestyles than females.
300
301 A combination of male frailty and females being equipped with equally dense intra-cortical
302 femoral bone to buffer excessive bone loss can be supported by previous reports of lesser
303 survivorship likelihood of unwell adolescent and young males from Taumako who may have

304 been fed a poorer diet[16]. Further evidence for this observation in the archaeological
305 samples[16], can be found in contemporary anthropometric research in Solomon Island
306 communities where sex-ratios are biased[57]. For example, the growth and nutritional status of
307 females on Roviana Island, was previously reported to be much better than that of young
308 males[57]. Furusawa and Aswani[57] used body-mass index, muscle and skinfold measurements
309 of body fat, and growth scores calculated from height and weight, to show that young females
310 were better nourished and grew faster than males. Taumako also has a long history of malaria
311 and yaws exposure to which might have impacted the modelling and remodelling events of
312 young males. Experiences of inconsistent dietary intake in childhood might have led to poor
313 bone maintenance later in life[58]. Indeed, males are typically considered to be more
314 susceptible to environmental and disease insults than females as hormones are a key factor
315 regulating immunity[59,60]. Even though no sex-specific differences in pathology (such as linear
316 enamel hypoplasia or lesions indicative of yaws)[22,61] have been previously noted in the
317 Taumako assemblage, our microscopic markers of bone health indicate long-term
318 physiological trends that do not manifest on the external skeletal or dental structures.
319 Complementarily, the Taumako female bone remodelling may have served as a buffer to
320 calcium loss through the life-course. We know from experimental research that loss of
321 calcium is compensated for by increasing remodelling in lactating females, which ultimately
322 restores compromised bone tissue during reproduction[62,63]. Taken together, a combination of
323 male and female specific bone physiology responses at Taumako may plausibly explain our
324 results.
325
326 **Bone remodelling with age at Taumako**
327 The two ontogenetic areas of key concern to life-long bone building capacities are the third
328 and fifth-sixth decades reflecting peak bone mass accrual and female menopause,

329 respectively[2]. Bone mass in modern humans through the life-course is easier to map than in
330 ancient populations as we cannot observe life-long change to bone mass accrual per
331 individual in the past. However, our sample size is large enough to begin unravelling
332 Taumako bone remodelling differences across the three anthropological age-at-death
333 categories. Overall, the entire sample followed an expected trend in secondary bone
334 remodelling with age, whereby Haversian bone structures increase in densities through
335 ontogeny[64]. Further, all the bone histology data appear to peak at the middle-age category,
336 which somewhat mirrors the expectation based on modern bone health through the life-course
337 paradigms. We acknowledge such comparison cannot be exact given the broad age-at-death
338 anthropological categories, but the end of the young, and the start of the middle-aged age-at-
339 death category, overlaps with the peak age for bone mass accrual in living humans[2]. When
340 considering the Taumako similarities in intra-cortical bone histology, it becomes apparent
341 that Taumako females maintain similar localised amounts of bone as males.
342
343 We can only provide cautious comments on bone histology in our old males and females,
344 because secondary osteons were measured in a limited number of well-preserved samples.
345 The one old female whose histology data were corrected by femoral robusticity, matched
346 bone remodelling trends of the female middle-aged category, without any indication of early
347 stages of osteoporosis (i.e. cortical bone trabecularisation or the presence of 'giant'
348 coalescing pores, see[13]) due to menopause. As a preliminary observation, we note that at least
349 some old Taumako females might have been shielded from significant estrogen loss insults,
350 or perhaps experienced it later in their lifespan. Otherwise, we would observe severe porosity
351 intra-cortically[65], in addition to the overall higher vascular porosities in females. This would
352 be difficult to validate as the anthropological methods of age-at-death estimation cannot
353 provide specific chronological ages or decades.

354 The descriptive trend in bone histology changes from young to old Taumako males, but a
355 lack of statistically supported differences, aligns with the same age-related observation in
356 1250–1450 AD Sudanese Kulubnarti, Nubia, where no statistically significant changes in the
357 geometric parameters of osteons were observed[38]. Our data also match some of the findings
358 reported by Burr et al.[54] for the Pecos females who appeared to maintain bone well into
359 adulthood and showed an age-related increase in bone remodelling. Generally, a smaller
360 secondary osteon size has been previously noted to occur with age in both males and
361 females[64], which applies to our results, and is similar to previous reports for the Pecos
362 males[54]. We cannot exclude the effect of osteon population density asymptote on the data in
363 the old category, whereby the evidence of pre-existing secondary osteons may have been
364 erased by subsequent generations of remodeled bone[66].
365
366 Our study highlights the significance of combining gross anatomical and microscopic
367 approaches to understanding bone biology in archaeological contexts. Robb et al.[49] reported
368 some effect of age on metacarpal cortical bone indices, and femoral length, in the Taumako
369 sample without accessing microstructural indicators of cortical bone remodelling. Our
370 robusticity indices were calculated for femora instead of just reporting length, and followed a
371 robusticity methodological recommendation based on a published thorough technical
372 evaluation of different robusticity measures[67]. It will be important for future
373 bioarchaeological studies to combine macro- and microscopic technical approaches as limb
374 bone size and shape complete modelling after the first two life decades[68].
375
376 **LIMITATIONS AND REMARKS ON TEMPORAL AND SPATIAL DATA**
377 We acknowledge that bone histology interpretations in archaeological settings need to be
378 conducted at a population level, but given our study presents the first osteonal remodelling

379 data for the Pacific region, we can establish that the Taumako data fall into a global range of
380 secondary osteon parameters for archaeological humans[34,38,40,54,55]. Some examples include:
381 the Taumako male and female combined osteon area (28,433 μm²) data are similar to
382 27,303.27 μm² reported for medieval Canterbury, England[34,40]; the male and female
383 combined area of Haversian canals in Taumako is 2,220.64 μm² which compares closely to
384 2,100 μm² in medieval (1250–1450 AD) Sudanese Kulubnarti, Nubia[38], 2,335.50 μm² in 14th-
385 19th centuries Pecos, New Mexico[54], and 2,334.12 μm² in medieval Canterbury, England[34,40].
386 Similarities can also be noted in raw osteon density data, whereby the Taumako data of
387 13.64/mm^2 are close to 11.78/mm^2 in Sudanese Nubia[38]. We acknowledge the above studies
388 used slightly different region of interest (ROI) selection techniques, but all considered
389 femoral midshaft cortical bone.
390
391 Further, we cannot exclude a series of confounding factors that have impacted our results and
392 interpretations. The estimates of age-at-death and sex for a portion of the sample at Taumako
393 rely on anthropological standards, as such they are probability scores. However, the aDNA
394 validation of the bulk of the sex estimates in this study overcame some of the uncertainty of
395 gross methods. Age-at-death assessments were validated as much as possible by ensuring that
396 each individual's histology profile generally matched its age-at-death status established from
397 the gross anatomical methods (e.g. thin sections were inspected for possible presence of
398 primary bone in samples from older adults). Unfortunately, we cannot overcome the
399 inconsistencies in sample size in each age-at-death and sex sub-group either, and do not have
400 access to better preserved bone histology. Finally, we only use two-dimensional (2D)
401 methods of thin sectioning, but a wider volumetric dataset providing three-dimensional (3D)
402 perspectives on vascularity connectedness, in combination with mineral density information,

would provide a more in-depth picture of bone building and remodelling capacities in the Taumako sample.

CONCLUSIONS

The Pacific islands are yet to be thoroughly studied for ancient human bone histological markers. Our study forms the first, largest sample size based, report of ancient human intra-cortical secondary osteon data in this part of the world. We have found that archaeological females at Taumako show highly vascularised femoral midshaft bone, but also have localised areas of intra-cortical bone that remodels similarly to that of males. This finding mirrors bone remodelling data from other archaeological sites from across North America, Europe, and Africa, and somewhat conforms to our modern understanding of bone loss through the life-course. These new data fall in the range of bone histology archaeological variability reported globally, extending currently available bone histology data by this site from the Pacific Islands. The intra-cortical bone histology in males might reflect their higher frailty in the cultural and environmental context of Taumako history. Ongoing efforts examining bone histology in Asia-Pacific will further our understanding of ancient human bone remodelling capacities in this region, contributing to modern efforts investigating the conditions under which human experience significant bone loss.

MATERIALS AND METHODS

Taumako is one of the remote Duff Islands, which lie northeast of Santa Cruz Islands in the far southeast Solomon Islands[15] (Figure 1). While the island is located within the Melanesian geographical boundary, Taumako is known as a 'Polynesian Outlier' representing a probable 'blow-back' migration of populations from Polynesia around the mid-second millennium AD[15,69,70]. The modern inhabitants of Taumako speak a Polynesian language, but as a result

of admixture with established populations, share similar cultural traditions to nearby Melanesian islands in the Duff and Santa Cruz groups[71].

The Namu burial mound, an archaeological site dated to 750 – 300 BP[15], yielded a significant number of human remains and associated grave goods that have been since examined to reconstruct the lives of the past inhabitants of Taumako. This has included social status stratification[15,17] reflected in dietary and child feeding practices reconstructed from bone stable isotope data[16,17,72]; abnormalities of the alveolar bone suggesting possible experiences of periodontitis[73]; evidence for interpersonal violence and warfare inferred from skeletal patterns of trauma[74]; a high prevalence of yaws (*Treponema pertenue*)[22]; and, more recently, gender specific migration patterns from neighbouring islands[75]. The site is known for archaeological evidence of using shell money and ornamentation practices that include a *tavi* - a neck ornament thought to represent high status (see[17]). The one aforementioned study also analysed Taumako femur length and metacarpal and femur cortical indices and noted a lack of distinct bone functional adaptation in remote Pacific Island environments[49].

With permissions from, and in collaboration with, the Solomon Islands National Museum, n = 69 Taumako adults were sampled in the present study. There were 19 left and 50 right femora. Both sides were pooled due to no statistically significant bilateral differences in the recorded data (Supplementary Information (SI) Tables 1, 2). Standard methods for estimation of biological sex and age-at-death were followed to assign the individuals into young (20-35 years old), middle-aged (35-50 years old) and old (50+ years old) age-at-death categories, and male or female estimates[50]. The methods are based on well-established age and sex related morphological changes to the post-cranial, cranial, and dental human structures[50]. With permission from the Solomon Islands Museum, the sex estimates based upon gross

methods were further validated through aDNA obtained from genome-wide data that were produced for a subset of samples investigated histologically. Individuals were sampled for DNA by drilling the petrous part of the temporal bone[76] (see dataset[77]). DNA was extracted from the sampled powder and prepared for next-generation sequencing by producing a double-stranded DNA library following established protocols[78-80]. Deaminated cytosines were enzymatically partially removed and retained only in the terminal positions as described in[81]. All libraries were directly shotgun sequenced on an Illumina HiSeq 4000 platform (1 × 75 + 8 + 8 cycles). The sequenced reads were mapped to the human genome reference hg19 using EAGER[82]. The retained damage was excluded from the analysis by masking the two terminal positions of each read[83]. The genetic sex was inferred using two independent methods: 1) The number of reads covering each position was counted across a total of around 1.24 million genome-wide SNP positions[84-86] and subsequently averaged for each sex chromosome and all autosomal ones. The Y- and the X- chromosome average coverages were normalized by the average autosomal coverage and compared to determine the sex assignment[87]. 2) An approach specifically designed for low-covered shotgun genomes in which the ratio between the average coverage across the entire X-chromosome and the coverage averaged across the autosomes was calculated as in[88] (see SI for extended aDNA methods). This was possible for n = 48 individuals. There was an 88% success rate (42/48) in corroborating the macroscopic and aDNA sex results, with only six individuals misclassified by the gross methodologies (see SI Table 3). Therefore, the presented sex classification can be treated as fairly reliable. We acknowledge we do not attempt to classify these as 'gender', but treat them as a biological entity in relation to bone metabolic processes. As a result, this study comprised n = 34 young adults, n = 13 middle-aged adults, n = 22 old adults, and n = 36 females and n = 33 males. Further sub-division by age-at-death within each sex group can be seen in the dataset[77].

Prior to histological analyses we recorded a series of femur morphometric measurements to characterise the size of each femoral midshaft and calculate femoral robusticity indices where possible[51,67]. Four variables were included: midshaft circumference (Circ) in mm, posterior cortical width (Ct.W) in mm, and femur maximum length in cm. These were measured using standard osteological laboratory equipment composed of an osteometric board, digital calipers, and a soft measuring tape. Two robusticity indices were computed using the Stock and Shaw[67] recommendation and following prior methods combining femoral bone histology and robusticity measures[40]: femoral robusticity index based on Circ where the circumference values are divided by femoral length, and a femoral robusticity index based on Ct.W where cortical width values are divided by femoral length and multiplied by 100. The latter included multiplication by 100 to increase decimals in the resulting robusticity index values for the ease of our statistical analysis. Only 23 femora were of a suitable preservation for measuring the maximum length, and so only these were used in the robusticity index calculations.

Next, posterior cortical bone samples from the midshaft of each femur were extracted using a Dremel tool with a rotary blade, resulting in approximately 1cm thick cortical quadrants (see[40]). The posterior femur is of interest to our study because it overlaps the *linea aspera*, a rich leg muscle site insertion anatomical landmark. Bone remodelling detected there should capture stimulation resulting from lifestyle[89-90], which will strengthen our analyses of by age and sex. Our minimal invasive approach ensured the femora remained as intact as possible, limiting the amount of archaeological bone being taken for the histological analysis[91].

Standard histological methods relevant to archaeological human remains were then followed to produce ~100 μm thin sections[30]. Each sample was embedded in Buehler epoxy resin, cut using a Kemet MICRACUT precision cutter equipped with a diamond blade, glued to a microscope slide, further reduced, ground, and polished to obtain a clear view of bone histology. The thin sections were examined using an Olympus BX53 microscope with a

DP74 camera using transmitted and linearly polarised light at a magnification of 10x (100x total magnification). Once histology slides were prepared, it became apparent that not all microstructures could be measured in all sections. Well preserved ROIs where cement lines of secondary osteons were easily identifiable were the case for only 21 individuals, but 68 individuals had consistently and suitably preserved Haversian canals. A diagenetically obscured band that ran along the outer posterior and endosteal layers of bone samples was also observed in the thin sections. However, the intra-cortical regions of bone were of an almost pristine preservation, which allowed us to focus on intra-cortical remodelling activity away from the immediately sub-periosteal and sub-endosteal regions of cortical bone. As such, we designed the ROI selection procedure so that data can be collected from the mid-portion of each sample by scanning a full cortical strip down the midline and then capturing three ROIs within its centre (Figure 2). The examination of cortical strips as ROIs, and intra-cortical bone regions generally, were successful in prior archaeological studies[39,92].

The Olympus cellSens software allows to automatically stitch images in live scanning mode, which we used to record each 'strip'. A thin section was placed on the microscope stage so that the mid-point of the periosteal border was in the field of view. The stage was then slowly moved forward (away from the observer) until the endosteal end of the border was reached. The area of the ROI strips ranged from 6.76 mm^2 to 25.84 mm^2 in our sample given variation in cortical wall thickness (mean = 14 mm^2, standard deviation = 3.67 mm^2). From within the strip, the first ROI was located at the midpoint (by dividing the length of the entire strip by two), and then one ROI was taken either side of this midpoint, ensuring no overlap in histology shown in the field of view (Figure 2). Using FIJI/ImageJ tools that included the "Multi-Point Count" and "Polygon" selections, three histomorphometric variables indicative of cortical bone remodelling events were measured (we use bone histomorphometry nomenclature recommended by Dempster et al.[93] Figure 2):

- **Vascular porosity (**V.Po) per mm^2 (e.g.[13,54,94,95]): total number of intact Haversian and primary canals across a full strip ROI of bone measured from the posterior to the endosteal borders of the section, and divided by the strip area in mm^2. Volkmann's canals were excluded because they were rarely visible in the sample. Because we worked with archaeological specimens and 2D histology sections, true vascular porosity, including other minute capillaries is not possible to obtain. In instances where cement lines of osteons were not visible, we cannot be entirely confident that a counted canal derives from a secondary osteon structure. As such, we use V.Po to represent all major vascular canals seen in the ROI strip.
- **Osteon population density** (OPD) per mm^2 (e.g.[38,40]): sum of intact osteon and fragmentary osteon numbers counted from three intra-cortical ROIs of 2.05 mm^2 area each (totalling 6.15 mm^2). Each sum was divided by the ROI area in mm^2.
- **Haversian canal:Osteon area ratio** (H.Ar:On.Ar), measured in μm^2 separately, and then converted to unitless ratio values (e.g.[40,51,96]): H.Ar is the average area (total area/total number of measured canals) of intact Haversian canals measured from three intra-cortical ROIs of 2.05 mm^2 area each (totalling 6.15 mm^2); On.Ar is the secondary osteon area in μm^2 created from average area (total area/total number of measured secondary osteons) of intact secondary osteons with complete cement lines measured from three intra-cortical ROIs of 2.05 mm^2 area each (totalling 6.15 mm^2). Secondary osteons cut off by an image border were excluded. The average values of H.Ar are then divided by the average values of On.Ar and multiplied by 100 to indicate percentage of canal to osteon area.

Recommended standards for reporting of bone histomorphometric data stipulate a minimum of 25 osteons examined per thin section[36]. Our study meets those standards by examining a minimum of 47 and maximum of 126 secondary osteons across the samples for the purposes

555 of osteon density calculations, and minimum 25 and maximum 50 for the purpose of ratio
556 calculations from area measurements of osteon units. The V.Po and OPD data are used in our
557 study as products of bone remodelling events that indicate the amount of bone produced and
558 remodeled per mm^2 intra-cortically[40]. The area of Haversian canals and secondary osteons
559 can be used as indicators of the rate at which, and stage of, a BMU travelling through the
560 cortex filling bone tunnels[32]. Larger areas of osteons and canals can be associated with longer
561 periods of BMU activity, and smaller areas would indicate a shorter-term BMU activity,
562 particularly if it is strain-suppressed[34,97].
563
564 Prior to addressing the main questions of our study, we ran non-parametric Spearman's *Rho*
565 tests (due to sample size smaller than 30 in at least one sub-group that was being included in
566 the correlations) correlating all the histology variables to check how well they reflect
567 remodelling relationships (i.e. whether porosities, densities, and area measures increased or
568 decreased in values when considered alongside each other). This step was necessary as we
569 have histology data from two different types of ROIs (the 'strip' and three localised ROIs
570 within), so we needed to check that each variable can be an independently reliable indicator
571 of bone remodelling activity. Statistically significant relationships and those of $Rho > 0.35$[98]
572 were taken to indicate that each variable was reflective of BMU activity and as such could be
573 interpreted on its own.
574
575 Next, the V.Po and OPD variables were then adjusted by the previously measured midshaft
576 variables and calculated RIs to account for a possible isometric relationship between femur
577 size and the underlying histological structures[51,52]. It is possible that larger femora could
578 simply show higher values of canals and osteons as a result of inherent size variation across
579 the sample. This was also important as previous research indicated that sexually dimorphic

bones may still build bone tissue of similar quality[99]. The H.Ar:On.Ar ratio variable did not require adjustments as it is in itself already a quantitative relation between two histology measures of size. The V.Po variable was adjusted by raw Circ and Ct.W (creating V.Po/Circ, and V.Po/Ct.W), whereas OPD was adjusted by robusticity index (Circ) and robusticity index (Ct.W) where the femoral maximum length was available for robusticity index calculations. A brief descriptive analysis summarising data using mean, minimum, maximum, and standard deviation (SD) values was conducted in first instance. The quantitative variables in n > 30 (Circ, Ct.W, V.Po, V.Po/Ct.W, V.Po/Circ) were tested for normality using the Kolmogorov-Smirnov test. Parametric tests were then selected for normally distributed variables (Circ, Ct.W, V.Po, V.Po/Circ), and non-parametric tests were applied to V.Po/Ct.W where data were not normally distributed. For data in sub-groups of n < 30, non-parametric inferential tests were selected without normality tests given the sample size. As a result, Mann-Whitney *U* tests or *t*-tests were applied when comparing bone macro- and microstructure between the sexes. When comparing the three age-at-death groups, we used a non-parametric Kruskal-Wallis test with a post-hoc pairwise comparison. For the gross femoral analyses we report significant results only, whereas for the histology analyses we show all results because they are interpreted to answer our research questions. We did not run statistical analyses on the OPD data, and age-at-death and sex sub-divisions due to inadequate sample size in the sub-groups.

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AUTHOR CONTRIBUTIONS

J.J.M. led project and consultation, secured funding, carried out histology lab work, data analysis, wrote first draft of manuscript; H.B. supervised project, interpreted data; M.F. performed aDNA analysis; S.C. performed initial in-silico screening for aDNA; K.N. and E.B. assisted with aDNA lab work; N.R.D.G. and M.M.W. assisted with histology lab work; L.K. assisted with osteology, led research permissions and consultation, interpreted data; A.P. secured funding and coordinated the sample collection; C.P. supervised the aDNA data generation and aDNA analysis; R.L. K. supervised project, secured funding, conducted osteology, collected samples, interpreted data, organised research permissions and consultation. All edited the manuscript and gave approval for publication.

DATA AVAILABILITY STATEMENT

Data are available open access from Figshare (Miszkiewicz et al. 2022[77]) https://doi.org/10.6084/m9.figshare.16815295.

CONFLICT OF INTEREST DISCLOSURE

None.

ETHICS APPROVAL STATEMENT

Approval to conduct this research was obtained from the Solomon Islands National Museum. The analysis and release of data were in consultation with and co-authorship by community representative (Lawrence Kiko), with whom also a layman's report summarising the findings was filed. The thin sections will be repatriated to the Solomon Islands National Museum upon the completion of this project. All research followed ethical guidelines of the American Association of Biological Anthropologists and the Australasian Society for Human Biology.

TABLES

Table 1. Descriptive summary of gross femoral data sub-divided by estimated sex and age-at-death. SD: standard deviation, MAX.: maximum, MIN.: minimum, Ct.W_RI: robusticity index (RI) calculated using cortical width (Ct.W) data, Circ_RI: robusticity index (RI) calculated using midshaft circumference (Circ). The RI variables are unitless.

GROSS FEMORAL MEASURES						
Sub-divided by sex and age-at-death		**N**	**Min.**	**Max.**	**Mean**	**SD**
FEMALE	Femur max length (cm)	6	40.60	44.50	42.42	15.41
	Circ (mm)	36	69.00	106.00	89.81	7.86
	Ct.W (mm)	36	3.95	13.23	8.77	1.96
	Circ_RI	6	17.93	22.33	20.66	1.91
	Ct_W_RI	6	1.47	2.57	2.08	0.46
MALE	Femur max length (cm)	17	30.40	47.00	42.92	4.70
	Circ (mm)	33	85.00	106.00	95.79	5.53
	Ct.W (mm)	33	6.56	15.27	10.77	1.65
	Circ_RI	17	19.43	34.21	22.85	3.84
	Ct_W_RI	17	1.75	4.16	2.58	0.56
YOUNG ADULT	Femur max length (cm)	16	304.00	477.00	426.81	48.13
	Circ (mm)	34	69.00	106.00	91.29	8.05
	Ct.W (mm)	34	3.95	15.27	9.61	2.21
	Circ_RI	16	17.93	34.21	22.40	4.18
	Ct_W_RI	16	1.47	4.16	2.45	0.67
MIDDLE-AGED ADULT	Femur max length (cm)	2	406.00	421.00	413.50	10.61
	Circ (mm)	13	81.00	106.00	93.15	7.54
	Ct.W (mm)	13	6.64	13.23	9.42	2.07
	Circ_RI	2	21.43	22.33	21.88	0.64
	Ct_W_RI	2	2.43	2.57	2.50	0.10
OLD ADULT	Femur max length (cm)	5	420.00	458.00	437.00	15.31
	Circ (mm)	22	83.00	104.00	94.50	6.15
	Ct.W (mm)	22	6.73	13.03	10.07	1.86
	Circ_RI	5	19.43	23.80	22.05	1.78
	Ct_W_RI	5	2.21	2.78	2.44	0.25

Table 2. Descriptive summary of histology data sub-divided by estimated sex and age-at-death groups. SD: standard deviation, MAX.: maximum, MIN.: minimum, V.Po: density of Haversian canals per mm^2, H.Ar/On.Ar: Haversian canal to osteon area in μm^2, OPD: osteon population density per mm^2, Ct.W_RI: robusticity index (RI) calculated using cortical width (Ct.W) data, Circ_RI: robusticity index (RI) calculated using midshaft circumference (Circ). All variables are unitless.

FEMUR HISTOLOGY MEASURES						
Sub-divided by sex and age-at-death		**N**	**Min.**	**Max.**	**Mean**	**SD**
FEMALE	V.Po/Ct.W	36	1.29	4.42	2.34	0.77
	V.Po/Circ	36	12.86	39.05	22.02	5.80
	H.Ar/On.Ar	9	6.50	10.40	8.31	1.36
	OPD/Ct.W_RI	4	6.34	6.84	6.62	0.24
	OPD/Circ_RI	4	5.53	7.86	6.76	0.53
MALE	V.Po/Ct.W	32	0.83	3.16	1.64	0.49
	V.Po/Circ	32	10.46	33.41	18.34	4.49
	H.Ar/On.Ar	12	4.57	9.46	7.61	1.52
	OPD/Ct.W_RI	8	3.40	6.69	5.31	1.20
	OPD/Circ_RI	9	4.78	7.66	6.34	1.08
YOUNG ADULT	V.Po/Ct.W	33	1.07	4.42	2.02	0.74
	V.Po/Circ	33	12.77	29.11	20.37	4.27
	H.Ar/On.Ar	13	4.57	9.85	7.83	1.40
	OPD/Ct.W_RI	8	3.40	6.69	5.75	1.16
	OPD/Circ_RI	9	4.90	7.66	6.44	0.96
MIDDLE-AGED ADULT	V.Po/Ct.W	13	1.29	3.40	2.22	0.69
	V.Po/Circ	13	13.98	28.19	21.53	4.75
	H.Ar/On.Ar	4	6.50	10.40	8.17	1.83
	OPD/Ct.W_RI	1	6.84	6.84	6.84	n/a
	OPD/Circ_RI	1	7.86	7.86	7.86	n/a
OLD ADULT	V.Po/Ct.W	22	.83	3.33	1.88	0.76
	V.Po/Circ	22	10.46	39.05	19.43	7.37
	H.Ar/On.Ar	4	5.42	9.36	7.92	1.73
	OPD/Ct.W_RI	3	4.04	6.81	5.36	1.39
	OPD/Circ_RI	3	4.78	7.39	6.10	1.30

Table 3. All results of inferential analyses comparing femoral size, and statistically significant results of bone histological markers compared between the sex and age-at-death groups. SD: standard deviation, MAX.: maximum, MIN.: minimum, V.Po: density of Haversian canals per mm^2, H.Ar/On.Ar: Haversian canal to osteon area in μm^2, OPD: osteon population density per mm^2, *t*: independent samples *t*-test, *Rho*: Spearman's Rho test, *U*: Mann Whitney *U* test, *H*: Kruskal-Wallis test, $*p < 0.5$, $**p < 0.01$, $***p < 0.001$.

COMPARISONS	Test statistic	n	*p*
Males vs. females			
Circ (mm)	$t = 3.626$	F = 36, M = 33	< 0.001***
Ct.W (mm)	$t = 4.577$	F = 36, M = 33	< 0.001***
Histology correlations across the sample			
H.Ar and On.Ar	*Rho* = 0.435	21	0.049*
On.Ar and OPD	*Rho* = -0.670	21	<0.001***
V.Po and OPD	*Rho = 0.531*	20	0.016**
Bone histology markers compared between males and females			
V.Po/Ct.W (unitless)	$U = 249$	F = 36, M = 32	<0.0001***
V.Po/Circ (unitless)	$t = 2.905$	F = 36, M = 32	0.005**
H.Ar/On.Ar (unitless)	$U = 40$	F = 9, M = 12	0.345
Bone histology markers compared between age groups			
V.Po/Ct.W (unitless)	$H = 2.4$	Y = 33, MA = 13, O = 22	0.301
V.Po/Circ (unitless)	$H = 3.278$	Y = 33, MA = 13, O = 22	0.194

FIGURES

Figure 1. Location of Taumako (red dashed outline), part of the Duff Islands (red marker) complex in Melanesia.

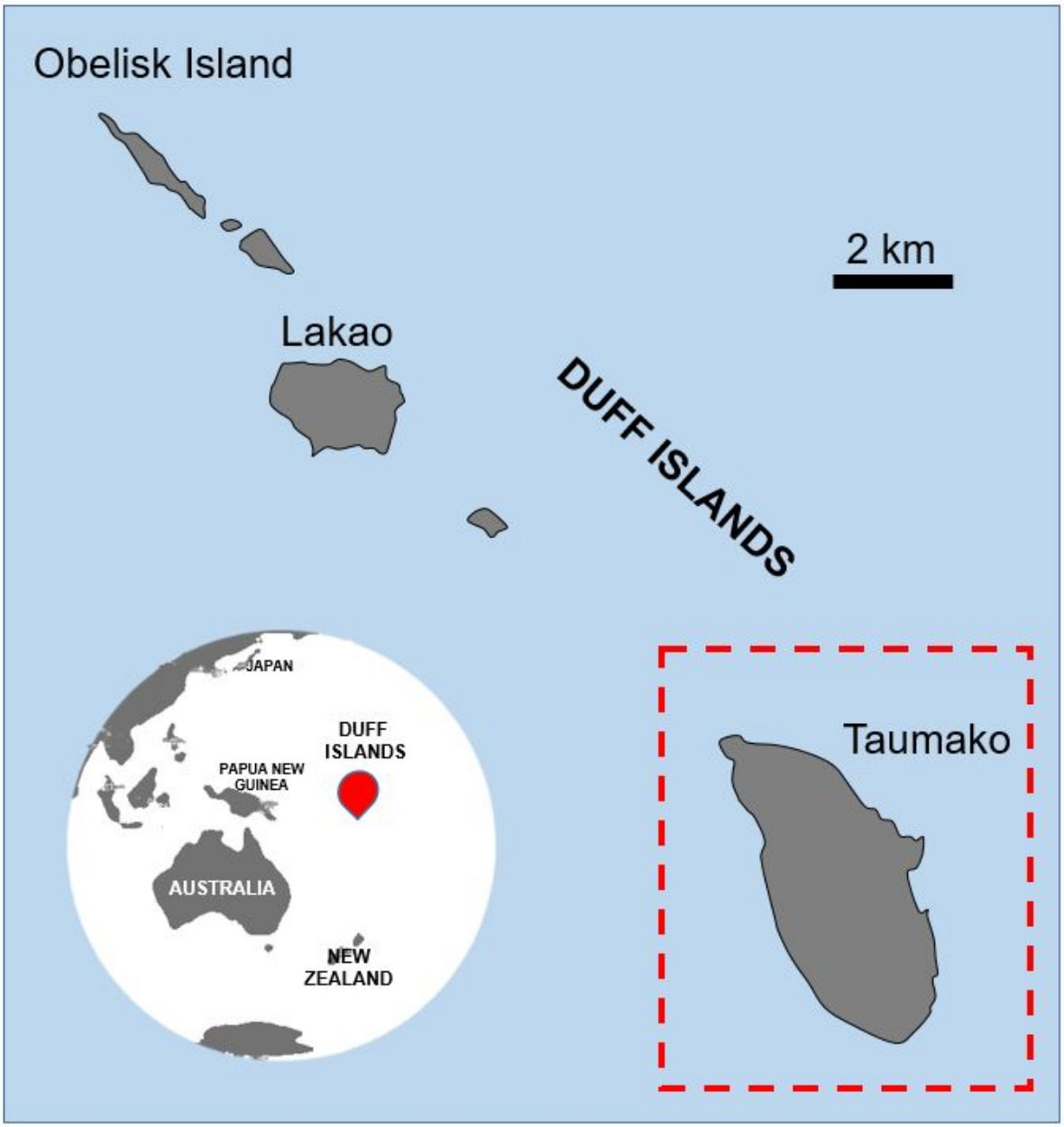

Figure 2. Summary of histomorphometric techniques used in this study. From left sketch of right posterior human femur – a: posterior cortical bone quadrant showing a strip (red dashed lines) of bone surface examined histologically from which vascular porosity (V.Po) was collected; b: three intra-cortical regions of interest (black rectangles) contained within the larger strip examined for osteon population density (OPD), Haversian canal area (H.Ar), and secondary osteon area (On.Ar); c: bone histology under transmitted light showing Haversian canals counted for V.Po (white triangle markers) and measured for area (c.1); d. bone histology under linearly polarised light showing secondary osteon area (d.1). Scale bars in c-d are 200µm.

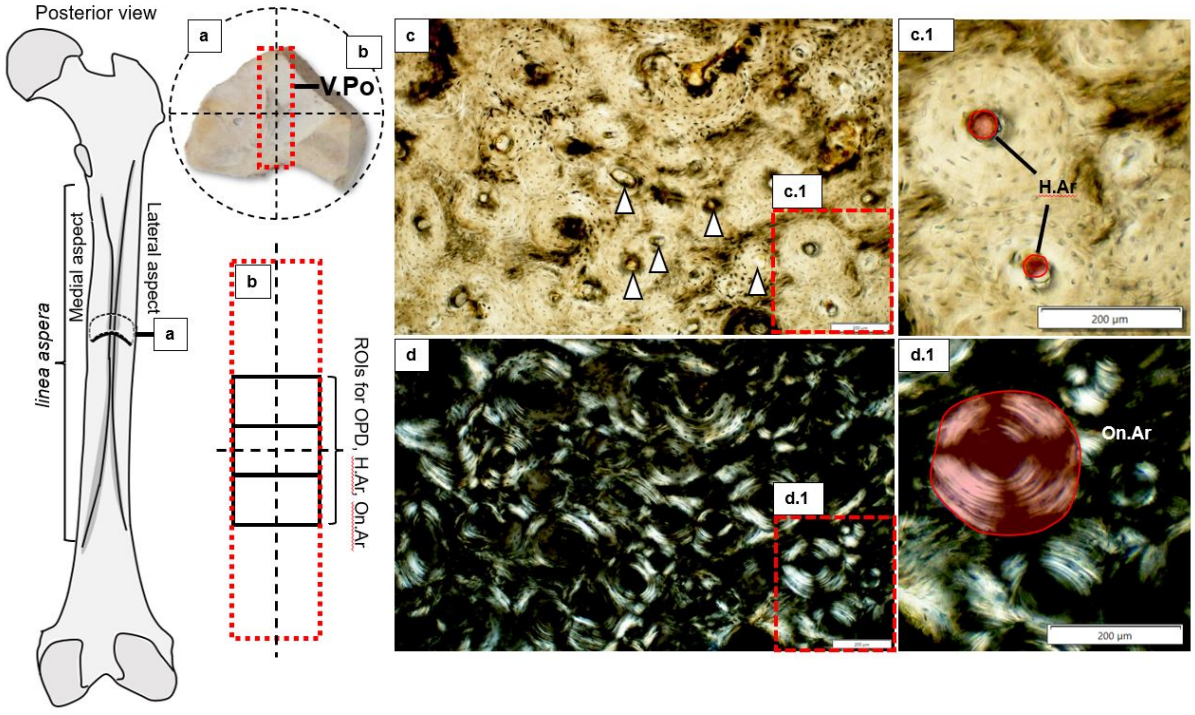

Figure 3. Montage combining simple correlations between the key histomorphometric variables examined in this study. We do not show y and x axis values as this figure is intended as a simple illustrative overview of how well the variables agree with each other. *p<0.05, **p<0.01, ***p<0.001 using Spearman's *Rho* tests (see Table 3).

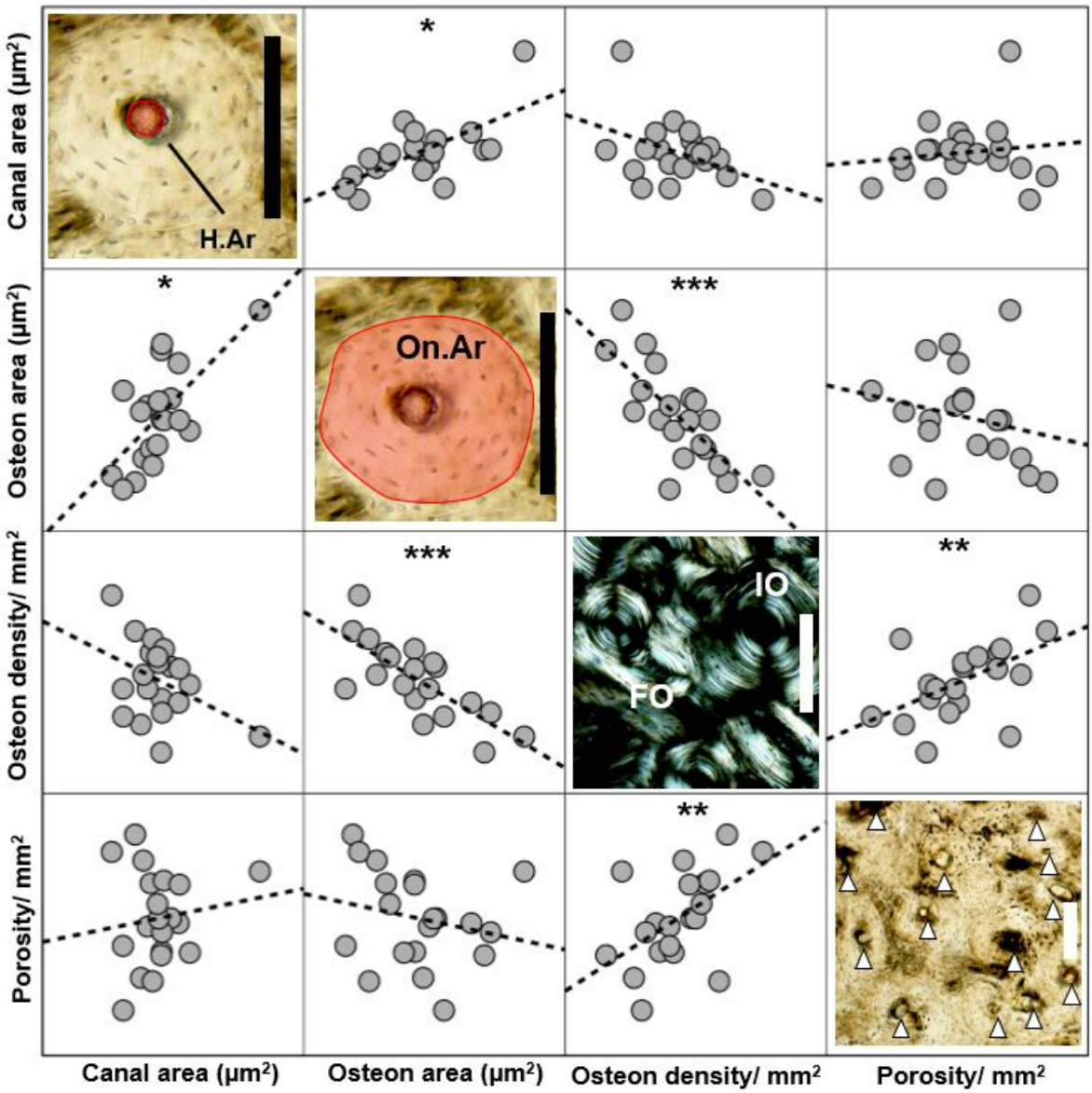

Figure 4. Simple boxplots illustrative differences in vascular porosities adjusted by different measures of femoral bone size (Circ: circumference of midshaft, Ct.W: cortical width), and ratio of Haversian canal area to osteon area, compared between the sexes (boxplots a-c), and age-at-death categories (d-f). ***p<0.001 using Mann Whitey U test (see Table 3).