



Polynesian Bones

Peter Stride^{1*}

¹University of Queensland, Australia.

Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

Article Information

DOI: 10.9734/BJMMR/2016/25651

Editor(s):

- (1) Panagiotis Korovessis, Chief Orthopaedic Surgeon, Orthopaedic Department, General Hospital "Agios Andreas" Patras, Greece.
- (2) Oswin Grollmuss, Head of Department of Pediatric and Adult Resuscitation Congenital Heart of Centre Chirurgical Marie Lannelongue, University Paris XI, France.

Reviewers:

- (1) Ronald L. Huston, University of Cincinnati, Cincinnati, USA.
- (2) Anonymous, Loma Linda University, USA.
- (3) Praveen Kumar Pandey, GGSIPU, India.
- (4) Taranjit Singh Tung, University of Manitoba, Canada.

Complete Peer review History: <http://sciencedomain.org/review-history/15149>

Review Article

Received 14th March 2016
Accepted 18th June 2016
Published 28th June 2016

ABSTRACT

Osteoporosis is a common bone disorder found predominantly in women in every corner of the globe both in the living and in skeletons of the last seven millennia found in global archaeological excavations, except Polynesia.

The Pacific Islands, or South Sea Islands, Polynesian people have an instantly recognisable phenotype characterised by a large bone and muscle mass frequently found in the front row of the rugby union scrum, or as security 'bouncers' on the door of night clubs. They are rarely seen in the orthopaedic wards of Australasia in spite of increasing migrant numbers and their passion for the two rugby football codes. This poses the questions of are their bones stronger and if that is the case, why is that?

Information directly from the islands is restricted by limited life expectancy, greater health priorities such as diabetes, limited diagnostic facilities and the lack of sophisticated computerised health information collection. However; this paper finds supportive data for the first question and identifies genetic and lifestyle factors as the possible answer to the second question.

Keywords: Polynesia; osteoporosis.

*Corresponding author: E-mail: pjostride@gmail.com;

1. INTRODUCTION

Polynesia encompasses a thousand islands with a population of some three million people on three hundred thousand square kilometres of land scattered over 25 million square kilometres of Pacific Ocean, in a triangle bounded by New Zealand, Hawaii and Easter Island. Most islands are still predominantly inhabited by these original settlers. The Maori ethnic race, one of the original Polynesian peoples, lived in New Zealand long before the arrival of European explorers and settlers, and still constitute fifteen percent of the New Zealand population, while another seven percent of them are migrants from other parts of Polynesia. These original occupants of New Zealand are the predominant source of accurate clinical information.

Polynesian people have well documented low fracture rates and high levels of bone density, though some research pre-dates the more modern measurements utilising DEXA scans and quantitative CT. Contemporary research and paleopathology reveal that osteoporosis has been present in every other continent for at least seven millennia, though it is less common in some African races [1]. The question arises therefore as to why Polynesians are different from most other ethnic groups in the world. Genetic and environmental effects are discussed, and the effect of increased muscle mass as the driver of increased bone mass is reviewed.

2. THE POLYNESIAN PHENOTYPE

The bone density and fracture rates of people of European and Asian ethnic origin has been extensively investigated, but the data of Africans and Pacific Islanders is much more limited, however there is still substantial data, much of it from New Zealand, demonstrating an increased bone and muscle mass and strength in Polynesians.

A study of 'fat free mass', essentially bones and muscles, discovered an average of 70 kg in a sample of Tongans compared with 62 kg in a control group of Caucasian origin Australians, a 13% difference [2]. Georgeson recorded bone density in the members of an Australian rugby league team on the Gold Coast of Queensland, the Titans, and found a bone mass one standard deviation greater than a control population, while

anecdotally noting the Tongan members of the team had the greatest bone density [3].

Norton noted lower rates of hip fracture in Polynesians unrelated to hip axis length [4], and Orr-Walker also noted both a higher lean body mass and fat mass in Polynesians [5]. Cundy studied women living in New Zealand of four different ethnic groups, Chinese, Indian, European and Polynesian, finding significantly greater bone mineral density (BMD) in the Polynesians, even after excluding the artefactual effect of the increased bone mass of Polynesians on bone density as measured by absorptiometry [6].

The question whether these changes developed before or after puberty was addressed by Grant who contrasted bone mineral content (BMC) and BMD in European and Polynesian children in NZ, finding increased whole body BMC but only increased BMD in the distal radius of Polynesians. These differences were attributed to the increased height, weight and lean body mass found in Polynesians, rather than any pre-pubescent bone differences, suggesting the difference in ethnic BMD is due to post pubescent alterations in bone growth [7].

Reid in 1986 assessed the bone mineral content (BMC) of the non-dominant distal radius and ulna in New Zealand Women aged 18-70 of either European or Polynesian origin using single photon absorptiometry [8]. The Polynesian women of Maori or Islander origin had similar BMC to each other, but both were 20% higher than the women of European origin, a significant difference with a p value < 0.0001. Following the menopause, which occurred at a similar age in the two groups, the BMC of Polynesian women decreased but only to the levels of premenopausal European women. Reid considered that this data was consistent with the low rate of fractured hips in Polynesians, but that the cause of the difference was unclear, although both European and Islander ethnic groups in New Zealand by 1986 had similar nutrition and lifestyles.

The increasing length of the femoral neck is hypothesised as being related to increasing risk of fracture, however Chin demonstrated that the Polynesians have longer average femoral neck length than Asian or European controls, hence this theory appears not to apply to South Sea Islanders, more evidence for a relatively unique bone phenotype [9].

3. MUSCLE MASS AND BONE DENSITY

The concurrence of increased bone and muscle mass poses the question of connection between the two, and if they are related, then which, if either, is the predominant driver, the chicken and the egg question for the muscle-bone unit.

Bones and muscles develop in proportion to each other in healthy children with a fairly constant bone mineral content to muscle cross sectional area, apart from a disproportionate increase in BMC in post-pubertal girls [10]. Karasik [11] reviewed the linkage and cross-talk between bones and muscles. Bone and muscle cells share the same mesenchymal cell precursor, so would be expected to share genetic controls. Two chromosomal regions, 5q35 and 10q24 have pleiotropic effects on both bone and muscle. Several genes, IGF1, Vitamin D receptor, glucocorticoid receptor, Wnt and resistin appear to affect both bone and muscle development. Inactivating mutations in mice of the myostatin gene, a muscle growth inhibitor, induce a hypermuscular phenotype with increased cortical BMD.

During adolescent bone growth, skeletal muscle comprises 45% of body weight, but in old age when osteoporosis is common, muscle mass is about 27% of total weight. Bone mass predicts 82% of the variation in femoral neck geometry. Mechanical loads can activate new bone formation via IGF1 stimulated proliferation of osteoblasts [11].

Ashby [12] demonstrated that bone and muscle mass accumulation in five to eighteen years olds proceeds in parallel, and that increases in BMC are proportional to lean body mass. Proctor [13] reported similar findings in adults age 21-93, in whom muscle mass was proportional to physical activity, and bone density was proportional to muscle mass. Muscle mass was found in a multivariate analysis to be the strongest determinate of bone density, accounting for up to 53% of the variance at different skeletal sites.

Lang [14] evaluated bone and muscle development in mice relative to activity and genetic markers. Mechanical loading increased bone strength. Genetic analysis may currently be uncertain as to which genes are most important in their effects on bones and muscles but their quantitative trait locus analysis detected several chromosome regions associated with increased bone and muscle mass, for example the D7Mit69

marker on chromosome seven, which is near the IGF1 receptor gene, is associated with increased bone strength and muscle volume and strength.

Vigorous unilateral arm exercise in sports, especially if commenced in the pre-pubertal period can promote marked bone hypertrophy [15]. There is a possibility that Vitamin D receptors are present on muscle cells, a reversible myopathy has been reported in osteomalacia and serum 1,25-dihydroxyvitamin D (1, 25(OH)₂ D) levels have been found to be associated with increased muscle mass and strength [16].

The relationship between bone density and muscle mass in Polynesian children aged three to seven using Caucasian controls of similar weight was evaluated by Grant [7]. The Pacific Island children had greater height, weight (both fat mass and lean body mass), BMI, bone mineral content, but after adjustment for lean body mass, there was no difference in bone mineral density. Lean body mass was closely related to bone density. Grant believed that lean body mass and muscular forces augmented bone mineral acquisition, and speculated that the higher muscle mass played a major role in the genesis of increased bone mass and density in Polynesians. Moustafa [17] demonstrated that bone loading in mice reduced sclerostin positive osteocytes and increased bone volume, providing further confirmation and a postulated mechanism for the genesis of bone through muscle loading.

Windelinck [18] postulates a possible link between muscle strength and genetics, showing muscle strength is related to vitamin D polymorphisms.

Thus there is extensive evidence of a close relationship between muscle and bone mass development, with increasing evidence genes that control both tissues, and the cellular mechanisms by which loading cause bone growth. Therefore, the next question we should be asking is why do Polynesians have large muscles?

4. NATURAL SELECTION

The Polynesian people traversed thousands of miles of ocean in their great voyaging canoes, stabilised by recently developed outrigger floats, using the ancient skills of navigating by the stars and ocean currents, while observing cloud

formations and animal life behaviour. Their talents at crossing the ocean were absolutely outstanding [19]. These canoes were driven by sails and paddles, as well as using the ocean currents. It is possible that the concept of 'survival of the fittest' applied during these incredible feats of endurance and navigation, with the weaker individuals dying at sea, to be then buried at sea, or perhaps providing nutrition for the stronger sailors. The successful remaining survivors of navigating the thousands of kilometres across the Pacific would have been the burly ones with broad shoulders and chests and huge muscular limbs. The original seafarers departing Taiwan and Melanesia heading for Polynesia between five and twenty millennia ago may have had a variety of body habitus, a diverse phenotype which possibly was reduced in transit to only those with a large bone and muscle mass as seen today in Polynesia.

The wheel and horses, or other beasts of burden, did not arrive till the advent of Europeans in the Pacific. Therefore, all travel and portage depended on muscular activity. The Polynesian people were also very warlike with frequent conflict between different island tribes. Cannibalism was common up to the nineteenth century. Nature's concept of 'eat, survive, reproduce' may have led to the natural selection of the very muscular phenotype over two or three millennia.

5. THE ORIGINS OF THE POLYNESIAN PEOPLE

The origin of the Polynesians has been extensively researched with most accurate current evidence coming from genetic, linguistic and archaeological disciplines. However, it remains a topic of doubt, controversy and speculation.

An appraisal of the basis of the Polynesian physique must examine these theories of their geographical and genetic origins as their colonisation of the Pacific is relatively recent in the history of Homo sapiens. The Polynesians believe, according to their folklore, that they came from a mythical place called Hawaiki, though the location of this ancient home remains obscure.

Scientific evidence indicates that the Polynesian people are a mixture of Melanesian and Austronesian ethnic groups. The Melanesians have lived in the West Pacific area for 50 to 100

thousand years being derived for the Proto-Australoids who migrated from Africa. The Austronesian people migrated from South-East Asia, predominantly from Taiwan into Indonesia some three to five thousand years ago, pushing the Melanesians eastwards into Papua and the Solomon Islands where they still comprise most of the population. The Melanesians did not progress further eastward alone.

Linguistic and archaeology experts believe the Polynesians originated as a homogenous group of people driven from Taiwan by agricultural problems, in a rapid and relatively recent wave of migration some 6,000 years ago, the 'Express train to Polynesia' theory [20,21] but this theory probably represents the last wave of migrants in prehistory as archeo-genetics suggest the explanation is more complex. The Taiwanese indigenous group diaspora are believed to have been living on the South Sea Islands for up to 6,000 years [22]. Their linguistic and genetic ties are with the Austronesian peoples of the Philippines, Malaysia, Indonesia, Madagascar, Polynesia, and Oceania (Addison) [23].

5.1 Y Chromosome Origins

Hurles [24] assayed the non-recombining portion of the Y chromosome in Melanesians and Polynesians, identifying two predominant haplotypes, accounting for 82% of the total different haplotypes, hg 10 and hg 26 in the Polynesian people. The former originates in Melanesia, and the later from SE Asia including Taiwan.

Underhill [20] assessed genetic markers in the Maori people of New Zealand who settled there about a millennium ago, compared with other local nations. Y-chromosome analysis detected European haplotypes in 40% dating from the European settlement over the last two centuries, and a similar number of Melanesian haplotypes from Indonesia and New Guinea. The remainder were from East Asia.

5.2 Mitochondrial DNA Origins

Underhill [20] also found a totally different picture in the mitochondrial DNA, with 85% having the 9-hp motif found in Polynesia and Asia, but not in Melanesia. The remaining 15% of mtDNA were of European origin.

Trejaut [25] found that the indigenous Taiwanese, the Melanesians and the

Polynesians shared three specific mutations in their mitochondrial DNA, not found in the mainland Chinese people who now comprise 98% of the population of Taiwan following the major Han Chinese immigration beginning in the 17th century.

5.3 Bone Density and Fracture Rates of the Polynesians and Their Original Ancestors

Comparative data on osteoporosis and fractures are limited in developing countries with limited health resources and a low life expectancy.

Firstly, according to the United Nations World Population Prospects 2012 Revision [26], the average life expectancy at birth over the period 2010–2013 for Fiji, Samoa, Tonga, Papua New Guinea and Indonesia was between 66 and 73 years, while the mean age for a series of 500 hip fractures in Australia was 83 [27]. Therefore, most Polynesians living in Polynesia will not reach the age at which minimal trauma fractures due to osteoporosis become common elsewhere in the world.

Secondly, measurements of bone density are much less common in developing countries, for example, the people of Indonesia have one DEXA machine per 8 million population compared with the European recommendation of one per 100,000 population. Most of these are in Jakarta and the cost is beyond most of the population, so data on the incidence of osteoporosis is very limited [28].

In Tonga in 2012, there were only seven radiographers in the whole country or 0.007 per thousand population, a tenth of ratio in Australia [29,30].

Osteoporosis is viewed as a low health priority in countries where infectious diseases and cardiovascular diseases related to the adoption of global company fast food in preference to healthier traditional diets are much greater problems. Much more precise methodology would be required to define the appropriate population and to distinguish minimal trauma from significant trauma.

Thirdly data is more likely to be valid and comparable when it is from a developed country with sophisticated computerised data collection than in the developing nations of Polynesia, hence information on the low rate of fractures within Polynesia is limited.

The most relevant information is likely to be found in New Zealand with a first world health system and data collection, a long life expectancy and as noted in the introduction, about twenty-two percent of the population have Polynesian ancestry. The Polynesian diaspora to other countries such as Australia and America is a more recent migration with no data yet on post-menopausal fracture rates. Raw data on fracture rates in Fiji would be confounded by the fact that approximately 40% of islanders are of Indian descent.

Data from island health systems would require individual chart review to distinguish minimal trauma fractures from those caused by significant trauma, and is not available.

Information relating to bone density and fractures found in the ancestral original homes of the Polynesians are limited and subject to confounding problems.

Details of the health status, and particularly information about bone disease in most displaced indigenous people all over the world is confounded by the low health and socioeconomic status found. They suffer from malnutrition, reduced life expectancy, unemployment, poverty, alcoholism and smoking. Information on the bone health of current indigenous people of Taiwan would be confounded by a few centuries of declining health statistics, and probably would be worse than half a millennium ago. Supporting evidence comes from Pietruszewsky [31] who examined 23 skeletons buried in Taiwan between two and five millennia ago, finding the indigenous people of Taiwan then were taller than their descendants of today, and none had any limb fractures suggesting at least average bone strength and growth before becoming a deprived race.

Data on fracture rates and bone strength in Melanesia is limited but fracture rates in the Solomon Islands were found by Barss [32] to be low suggesting these migratory ladies from Taiwan collected men with strong bones. Watters [33] in Papua New Guinea reported that osteoporosis, Colles' fractures and fractures of the neck of femur were rare. Fractures followed significant trauma rather than minimal trauma. Galeazzi and Monteggia radial fractures with distal or proximal radial dislocation were more common than Colles' fractures illustrating the need for precise data, rather than raw radial fracture rates.

Kanis and an International Osteoporosis Foundation working group collated data on hip fracture rates, finding that Indonesia had a low reported frequency of hip fractures, coming fifty-third out of 63 nations with approximately 190 fractures per 100 thousand women compared with just under 600/100,000 in Denmark and 20/100,000 in South Africa [34]. In 2012 there were 43,000 hip fractures in Indonesia. These limited figures age imply an ethnic group, who genes contributed to the Polynesian phenotype, with above average strength bones.

Comparing fracture rates between different countries yields data in which it is difficult to distinguish between real differences and methodological differences, for example Bacon [35] evaluated the difference in fracture rates across 9 countries in Europe, the Americas and Hong Kong, finding an apparent tenfold difference between the highest and the lowest rates, yet was not clear if this was a genuine difference or due to varying data collection methods.

Three reviews of worldwide bone disease failed to isolate data from Polynesia from other parts of Australasia and South East Asia.

Johnell [36] in a review of the worldwide prevalence of osteoporotic fractures states that 28% of global osteoporotic fractures occur in the Western Pacific area, but his classification of Western Pacific countries includes not only Fiji, Samoa and Tonga, but also China, Japan, Korea and Australia, thus not giving any specific information about Polynesian bones.

Johnell [36] also reported that 4.2% of world hip fractures in 1990 occurred in 'other Asia and Islands', a group including all Asia except China, Japan and India, again a heterogeneous group with no specific Polynesian data.

Gullberg [37] endeavoured to predict the worldwide present and future risks of hip fracture, but the data for 'Oceania' was derived solely from Australian data.

Wilkinson in 1998 reported only four orthopaedic operations requiring a pin or plate out of a total of 770 general surgical operations in Tonga, but information is not available on age, bone involved or degree of trauma [38]. Therefore, available data on fractures and bone density in the 'Polynesian Islands' and the original homes of the Polynesian peoples is very limited, and

may not be an accurate indication of the presence of osteoporosis or increased bone strength.

There is however some information of relevance about fracture rates in one group of Polynesians, those from New Zealand, the Maoris.

Stott in 1980 found a significantly lower rate of hip fractures in New Zealanders of Maori origin, compared with those of non-Maori origin [39]. Barber evaluated hip fracture data for the period 1989-1991 in New Zealand. The age specific hip fracture rate per 100,000 Maori males was 197, compared with 288 for non-Maori males, and the rate per 100,000 Maori females was 516 compared with 827 for non-Maoris, a highly significant sixty percent higher rate in non-Maori females [40].

Therefore, the optimum available data implies the Polynesians and their distant ancestors had stronger bones and less fractures than most Asians and Europeans.

6. OTHER FACTORS

6.1 Diet

The dietary habits in Polynesia are changing as global commercial business becomes the driving paradigm overtaking successful traditions; such that the dietary fat content has increased in recent decades. There is no clear evidence yet of change in bone density with a less healthy diet. The traditional diet in the Pacific Islands was 75 to 80% starch, 7 to 12% fat, and 12 to 15% protein. The root vegetables, taro, cassava, yam; fruits such as breadfruit, green bananas, pawpaw, pineapple and a wide variety of citrus fruits, seaweeds, and nuts such as peanuts, and macadamia provide the starch. There is currently no clear data that a diet with a high percentage of root crops has an effect on bone density. Protein and fat come from pork, chicken, beef and the abundant fish. Milk and other dairy products are uncommon and there is a high prevalence of lactose intolerance.

Roberts [41] found 34% of a group of Polynesians had the MCM6 -13910CC genotype, the cause of lactose intolerance. Cattle are used for beef, rather than dairy. Calcium is found in coconuts and coconut milk. Cresswell [42] found coconuts contained 0.16% calcium, therefore 100 mls of coconut milk contains 16 mg calcium, less than the 100 mgs

found in cows' milk but apparently adequate for nutrition and growth, and undoubtedly excluding a high calcium diet as the cause of increased bone mass.

6.2 Vitamin D and Sunlight

Polynesia has adequate amounts of sunlight for vitamin D synthesis throughout the year. Both Samoa and Fiji average eight hours' sunshine daily in summer, down to around five hours daily in Samoa and six in Fiji during 'winter' [43].

6.3 Exercise

Polynesians have a culture of high levels of muscular activity for three millennia prior to the advent of western machinery in the last century. They rowed, fought and carried continually, generating muscle and bone bulk.

7. CONCLUSIONS

The linguistic patterns found in Polynesia, unlike the usual pattern elsewhere in the world, therefore are derived from the female sector. The interpretation of the genetic data, a counter-intuitive and unique theory, is that a predominantly female group of Indigenous Taiwanese, travelled to Melanesia, 'abducted' a similar size group of muscular males, and 'coerced' them to paddle ocean-going canoes to Polynesia, clearly a group of early Holocene feminists.

Data suggests that the increased bone and muscle development found in the Polynesian people is partly a genetic effect on the bone-muscle precursor mesenchymal cells which produce larger than average bone and muscle bulk, once found in the indigenous people of Taiwan and still found in the Melanesians, the original sources of the female and male DNA respectively, and partly the selection effect of paddling thousands of kilometres of ocean for exploration, tribal warfare and the use of muscles for portage and all forms of travel.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. Stride PJO, Patel N, Kingston D. The history of osteoporosis: Why do Egyptian mummy mummies have thin bones? Diagnosis, definition, diagenesis, density and drugs. *J Royal Coll Physicians Edinb.* 2013;43:254-61.
2. Craig P, Halavatau V, Comino E, et al. Differences in body composition between Tongans and Australians: Time to rethink the healthy weight ranges? *Int J Obesity.* 2001;25:1806-1814.
3. Georgeson EC, Weeks BK, McLellan C, et al. Seasonal change in bone, muscle and fat in professional rugby league players and its relationship to injury: A cohort study. *BMJ Open.* 2012;2:e001400. DOI: 10.1136/bmjopen-2012-001400
4. Norton R, Campbell AJ, Lee-Joe T, et al. Circumstances of falls resulting in hip fractures among older people. *J Am Geriatr Soc.* 1997;45(9):1108-12.
5. Orr-Walker B, Evans M, Reid I, et al. Increased abdominal fat in young women of Indian origin. *Asia Pac J Clin Nutr.* 2005;14(1):69-73.
6. Cundy T, Cornish J, Evans MC et al. Sources of interracial variation in bone mineral density. *J Bone Miner Res.* 1995;10(3):368-373.
7. Grant A, Gordon F, Ferguson E, et al. Do young New Zealand Pacific Island and European children differ in bone size or bone mineral? *Calcif Tissue Int.* 2005;76:397-403
8. Reid I, Mackie M, Ibbertson H. Bone mineral content in Polynesian and white New Zealand women. *BMJ.* 1986;292:1547-48
9. Chin K, Evans MC, Cornish J, et al. Differences in hip axis and femoral neck length in premenopausal women of Polynesian, Asian and European origin. *Osteoporos Int.* 1997;7(4):344-7.
10. Schoenau E, Neu CM, Beck B, et al. Bone mineral content per muscle cross-sectional area as an index of the functional muscle-bone unit. *J Bone Miner Res.* 2002;17(6):1095-101.
11. Karasik D, Kiel D. Genetics of the musculoskeletal system: A pleiotropic

- approach. *J Bone Min Res.* 2008;23:788–802.
12. Ashby R, Adams J, Roberts SA, et al. The muscle-bone unit of peripheral and central skeletal sites in children and young adults. *Osteoporos Int.* 2011;22(1):121-32.
 13. Proctor D, Melton L, Khosla S, et al. Relative influence of physical activity, muscle mass and strength on bone density. *Osteoporos Int.* 2000;11(11):944-52.
 14. Lang D, Conroy D, Lionikas. A bone, muscle, and physical activity: Structural equation modelling of relationships and genetic influence with age. *J Bone Min Res.* 2009;24(9):1608-1617.
 15. Stride P. Bone density, King Richard III, Rafael Nadal, and modern imaging of exercise-induced osteogenesis. *The Ricardian Sept.* 2009;3:27-30.
 16. Marantes I, Achenbach SJ, Atkinson EJ, et al. Is vitamin D a determinant of muscle mass and strength? *J Bone Miner Res.* 2011;26(12):2860-71.
 17. Moustafa A, Sugiyama T, Prasad J. Mechanical loading-related changes in osteocyte sclerostin expression in mice are more closely associated with the subsequent osteogenic response than the peak strains engendered. *Osteoporos Int.* 2012;23:1225–1234.
 18. Windelinckx A, De Mars G, Beunen G, et al. Polymorphisms in the vitamin D receptor gene are associated with muscle strength in men and women. *Osteoporos Int.* 2007;18:1235–1242.
 19. Buck Sir Peter. *Vikings of the Sunrise Whitcombe and Tombs*; 1938.
 20. Underhill P, Passarino G, Lin A. Maori origins, Y-chromosome haplotypes and implications for human history in the Pacific. *Hum Mutat.* 200;17(4):271-80.
 21. Friedlander J, Friedlaender F, Reed F, et al. The genetic structure of Pacific Islanders. *PLoS Genet.* 2008;4:e19.
 22. Blust R. Austronesian: A sleeping giant? *Language and Linguistics Compass.* 2011; 5 (8):538–550.
 23. Addison D, Matisoo-Smith E. Rethinking Polynesians origins: A West-Polynesia Triple-I model. *Archaeology in Oceania.* 2010;45(1):1–12.
 24. Hurler M, Nicholson J, Bosch E, et al. Y chromosomal evidence for the origins of oceanic-speaking peoples. *Genetics.* 2002; 160(1):289-303.
 25. Trejaut J, Kivisild T, Loo J, et al. Traces of archaic mitochondrial lineages persist in Austronesian-speaking Formosan populations. *PLoS Biol.* 2005;3(8):1362-72.
 26. World Population Prospects Economic & Social Affairs The 2012 Revision Highlights and Advance Tables Reviewed, February; 2016. Available:http://esa.un.org/unpd/wpp/publications/Files/WPP2012_HIGHLIGHTS.pdf
 27. Stride P, Houston A, Ratnapala D, Perron J. SAHFE and SIGN in Australia. International benchmarking of 500 cases of hip fracture. *J R Coll Physicians Edinb.* 2007;37:98–102.
 28. Available:http://www.iofbonehealth.org/sites/default/files/media/PDFs/Regional%20Audits/2013-Asia_Pacific_Audit-Indonesia_0_0.pdf viewed February 2016
 29. Available:http://www.wpro.who.int/health_services/service_delivery_profile_tonga.pdf
 30. Available:<http://www.aihw.gov.au/workforce/medical/types-of-medical-practitioners>
 31. Pietrusewsky M, Tsang C. A preliminary assessment of health and disease in human skeletal remains from Shi San Hang: A prehistoric aboriginal site on Taiwan. *Anthropol Sci.* 2003;111(2):203-223.
 32. Barss P. Fractured hips in rural Melanesians: A nonepidemic. *Trop Geogr Med.* 1985;37(2):156-9.
 33. Watters D, Kapitgau W, Kaminiel P. Surgical capability and surgical pathology in Papua New Guinea in the year 2000. *ANZ J Surg.* 2001;71(5):274–280. DOI: 10.1046/j.1440-1622.2001.02101.x
 34. Kanis J, Odén A, McCloskey V, et al. On behalf of the IOF working group on epidemiology and quality of life. A systematic review of hip fracture incidence and probability of fracture worldwide. *Osteoporos Int.* 2012;23:2239–2256. DOI: 10.1007/s00198-012-1964-3
 35. Bacon W, Maggi S, Looker A, et al. International comparison of hip fracture rates in 1988 – 1999. *Osteoporos Int.* 1996;6:69-75
 36. Johnell O, Kanis J. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporos Int.* 2006;17:1726–1733.

37. Gullberg B, Johnell O, Kanis J. World-wide projections for Hip fracture. *Osteoporos Int.* 1997;7:407–413.
38. Wilkinson D, Pifeleti F. General surgery in Tonga: An audit. *Pacific Health Dialog.* 1999;6(2):199-201.
39. Stott S, Gray D. The incidence of femoral neck fractures in New Zealand. *N Z Med J.* 1980;91(651):6-9.
40. Barber J, Mills H, Horne G, et al. The incidence of hip fractures in Maori and non-Maori in New Zealand. *N Z Med J.* 1995;108(1007):367-8.
41. Roberts R, Merriman T, Upton J. High frequency of MCM6 lactose intolerance genotype in Polynesian people. *Aliment Pharmacol Ther.* 2010;32: 828–830.
42. Creswell D, Brooks C. Composition, apparent digestibility and energy evaluation of coconut oil and coconut meal. *J Anim Sci.* 1971;33: 366-369.
43. Available:<http://www.weather-and-climate.com/average-monthly-hours-Sunshine> 32

© 2016 Stride; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

*The peer review history for this paper can be accessed here:
<http://sciencedomain.org/review-history/15149>*