# Bringing to bear: A biosocial examination of the developmental origins of the obstetric dilemma.

Sarah-Louise Decrausaz

Darwin College Phenotypic Adaptability, Variation and Evolution Research Group Department of Archaeology University of Cambridge

> This thesis is submitted for the degree of Doctor of Philosophy

> > August 2018



## Declaration

This thesis is the result of my own work and includes nothing that is the outcome of work done in collaboration except as declared here and in the relevant sections of the text. The contributions of other researchers are as follows. The comparative magnetic resonance imaging and dual-energy x-ray absorptiometry sample used in Chapter Two was provided by Megan Shirley and Professor Jonathan Wells. The Great Ormond Street Hospital reference sample and the associated data used in Chapters Three, Four and Five of this thesis was provided by Professor Mary Fewtrell, Professor Jonathan Wells and Dr. Jane Williams.

This thesis is not substantially the same as any that I have submitted, or, is being concurrently submitted for a degree or diploma or other qualification at the University of Cambridge or any other University or similar institution. I further state that no substantial part of my thesis has already been submitted, or, is being concurrently submitted for any such degree, diploma or other qualification at the University of Cambridge or any other University or similar institution.

This thesis does not exceed the prescribed word limit for the Archaeology Degree Committee (80,000 words).

Sarah-Louise Decrausaz

Cambridge, August 2018.

#### Summary

Childbirth is biomechanically challenging for humans, as mothers must deliver a relatively large-headed neonate through a narrow birth canal. Previous work has indicated that a narrow birth canal in humans is a result of an anatomical compromise for efficient bipedal locomotion and the ability to give birth to largebrained infants. This previous work also suggested that difficult and potentially risk delivery of altricial humans was both evidence of and the solution to the tight relationship between infant size and the maternal birth canal in humans. This is known as the obstetric dilemma (OD). More recent work has complicated the OD and pointed to ecologic context as an influence on pelvic dimensions, suggesting that the mismatch between the maternal birth canal and the size of the human infant may have arisen with the transition to agriculture. This thesis begins with the theme of OD and uses it as a platform to examine the developmental trajectory of the bony pelvis in growing women in the context of childbirth. This project investigates associations between pelvic dimensions, biological and biosocial variables in living girls (n=286) from southern England to clarify the factors affecting the birth canal throughout growth and development.

Dual energy x-ray absorptiometry (DXA) can be successfully used to collect pelvic dimensions measurements from healthy, living women. The same pelvic dimensions were collected from magnetic resonance (MRI) and DXA images. DXA measurements all had a higher technical error of measurement than MRI though were within acceptable error limits. Bi-iliac breadth was most closely matched in DXA and MRI measurements. Pelvic measurements collected from DXA scans represent the same biological variation at those collected from MRI scans.

Growth curves of pelvic dimensions show that bi-iliac breadth increases in a similar manner to shoulder breadth, whilst the pelvic canal increases in breadth most noticeably between 11 and 17 years of age. Peak growth velocity of the pelvic canal occurs between 11 to 12 years of age, whilst peak bi-iliac and shoulder breadth growth velocity occurs at approximately 8 to 9 years of age. The division in growth patterns between the non-canal and canal components of the pelvis suggest differing

patterns in canalization and potential for genetic compared to environmental impact on their morphology.

Biological factors that associated with shoulder breadth and bi-iliac breadth were height and thigh circumference, whilst pelvic canal dimensions associated with height and indicators of pubertal development. This suggests that pelvic canal dimensions increase more slowly whilst hip and shoulder breadth increase alongside stature growth in girls. No biosocial factors associated with shoulder breadth or pelvic dimensions, demonstrating stronger associations between biological factors than biosocial factors on pelvic development. These results also underline the unique developmental trajectory of the canal compared to the non-canal components of the pelvis, and suggest that pubertal development indicators (which include localized fat deposits relevant to breastfeeding) align in growth with pelvic canal dimensions.

In summary, the findings of this thesis contribute an important basis with which to consider the origins of childbirth difficulty. The results of this project contribute to scholarly work that is redefining the evolutionary origins of childbirth difficulty, proposing more nuanced origins than the original OD hypothesis. The implications of this thesis are that obstetrically relevant pelvic dimensions have a specific growth trajectory that may be impacted by environmental factors in early life and ultimately contribute to childbirth difficulty in adulthood. Results from this work also suggest that the key period of pelvic canal development occurs ahead of puberty whilst also demonstrating that growth priority for the pelvic canal is linked to indicators of pubertal development including localised pockets of adipose tissue. Methodologically this thesis provides a basis for more widespread use of medical imaging in investigating osteological variation. More broadly the results of this work demonstrate that an evolutionary medicine approach that integrates osteological methods and clinical data provides a richer framework to examine the intersections between growth and reproduction in humans.

#### Acknowledgements

They say it takes a village, though this experience has taught me it takes a village, a few cities, a couple of departments and a fair amount of beer. I have had profound luck in many things in my life and I count my PhD amongst them. I owe a large debt to my supervisor Dr. Jay Stock – his wit, humour and intellect have carried me through this experience and have given me the strength to keep going. I am so pleased that I can count you amongst my mentors. I thank Professor Jonathan Wells for his incredible generosity in introducing me to the sample I have examined in this thesis and for his truly saint-like patience in teaching me so much about child growth. The fact that I was mentored by two researchers whose very work inspired my proposed PhD project truly is, as they say back home, 'fully sick'.

This thesis would also not have been possible without the collaboration and warm welcome I received from Professor Mary Fewtrell and Dr. Jane Williams at the University College London Institute of Child Health and the Great Ormond Street Hospital. A special mention to Jane for all of her help with rebuilding the DXA database and for explaining the reference sample data in all its complexity. I also thank Dr. Megan Shirley for sharing part of her doctoral data with me and for her serene collaboration on getting those MRI measurements sorted out! I look forward to collaborating more in future. To all of the girls and women in the reference sample – it has been my privilege to see how you grew through the scans and data you provided for the original project and for this thesis. My thanks go to Paul Stevens at General Electric who despite my nigh-harassment-level emails confirmed so many technical DXA details for this project.

I would like to express my gratitude to the Parkes Foundation for the Small PhD Grant that supported my data collection as well as a number of other expenses. I also thank the Department of Archaeology at the University of Cambridge for the travel and conference funding that has supported the sharing of my thesis results at national and international conferences. The PAVE lab provided a professional and personal homebase throughout this PhD and I would like to thank all of those who have made this so. Special mentions to Dr. Jaap Saers, Steph Payne, Dr. Ali Macintosh, Dr. Laura Buck and Ella Brown for putting up with my endless nonsense. I want to thank Dr. Rebecca Roberts for being a willing ear and for helping me laugh so very much. An extra special shout out to Eóin Parkinson for his friendship and magnificence – you're the best of eggs. To Dr. Emma Pomeroy, it has been such a treat to count you as a mentor and a friend. Your genuine and gentle support through this project has helped me more than you know. I am very excited to keep working with you.

Beyond PAVE, I had the great fortune to work with a number of wonderful colleagues in the Department of Archaeology at the University of Cambridge. I am VERY much obliged to Fabio Lahr whose technical wizardry enabled me to transfer and rebuild the database I needed for this PhD project. I thank Barbora Sajfrtova for showing me the admin ropes when I arrived and for her warm support throughout these four years. A large thank-you to Jo Osborn for her humour, support and for thinking of me for employment within the department. I am very grateful to the support and excellence of Dr. Trish Biers over the last few months in particular - I want to be like you when I grow up. Another colleague who has been a peaceful and important source of support during this PhD is Dr. Robert Attenborough. It is always a pleasure sharing Antipodean memories with you and I am thankful for all of the work you have done and continue to do for doctoral students within this Department. I also want to thank Laure Bonner for her vim and endless encouragement - the Department is richer for having you in it. Thanks also to Jake Dunn for giving me a fun and rewarding television gig and to my fellow PhD crusaders in archaeology and biological anthropology.

I am grateful to the Oxbridge Academic Programs crew for giving me the chance to learn so much about teaching, teamwork and what really matters in life. I am similarly indebted to the Grantchester Life Drawing Group for giving me a meditative and empowering space and a chance to bang on about my research. In the background of all this was a heavy cloud of music and colour that helped me think, decide on hair dye and more importantly, dance. I will be forever grateful to RuPaul for being the mother of us all and for creating Drag Race – a vision of the world as it should be. I thank Donna Summer, Jimmy Somerville, Frankie Goes To Hollywood, Underworld, David Bowie, The Smiths, The Clash, The Whitlams, Paul Kelly, Kate Bush, New Order, The Scissor Sisters, Public Image Limited, Madness and every vaporwave compilation I could get my hands on for getting me through data analysis and writing this beast.

What with moving to a couple of different places to complete my university studies, I have been fortunate to have friends who have become my family. This thesis would not have been possible without the love and camaraderie of Anna Lyttle and Michael McQuaid, Christina Farley, Tom Siek, Tyler Kelly and Pablo Martínez, Vasa Lukich, Sara Rhodes, Kristen Prufrock, Samantha Leggett, Marissa Ledger, Natalie Ward and Devin Ward (no relation). I thank Massimo Fabio Lando for being an inspiration and for his mind-blowing cooking. I thank Michael Rivera, my brother from another mother, for sharing in this adventure with me and for always having my back – you make me a better person. A part of my heart belongs to Michelle Cameron, who has had more faith in me than I ever thought possible. I am honoured to call you my friend, collaborator and frankly, second brain. Those TV execs won't know what'll hit'em.

I owe a lot to my family. Their support has been complete and unwavering, their love strong even with so much distance between us. I thank my mother, Christine Decrausaz, for her endless strength and my father, Philippe Decrausaz, for his endless wisdom. I thank my brother Mark-Henry Decrausaz for setting #goals for coolness and for your love. I am so grateful to Kathleen, Denis and Ciara Sarrasin, as well as the Sweeney and Sarrasin clan for welcoming me into their family and equally for letting me ramble about my studies. I remain in awe of the love I have in my better half, Kyle Sarrasin. I can never repay you for your willingness to sign on to this adventure. This thesis is dedicated to you, as W.H. Auden would put it, my north, my south, my east and west.

Declaration	1
Summary	2
Acknowledgements	4
Table of contents	8
List of tables	11
CHAPTER ONE:	20
The growing skeleton and childbirth	20
1.1. The structure of this thesis	20
1.2. The context and central research questions of this thesis	20
1.3. The human pelvis	22
1.4. The development of the human pelvis in utero, childhood, adolescence	and
early adulthood	24
1.5. The development of the human pelvis and the actions of puberty	27
1.6. Sexual dimorphism of the human pelvis	
1.7. The human pelvis and childbirth: Biological and social components	32
1.8. Variation in the human pelvis of global populations and hunter-gathered	
communities	
1.9. The obstetric difference of abildbirth difficulty	
1.11. Eassil avidence of the avalution of human shildhirth	41 42
1.12 Birth in non-human primates and mammals and birth difficulty in prima	40 atos 16
1.13 An evolutionary medicine approach: Examining the origins of the obst	atric
dilemma in growing girls	48
1 14 Research aims	49
1.15. Detailed research aims	
	-
CHAPTER TWO:	54
The use of medical imaging in measuring pelvic growth	54
2.1. The role of Chapter Two in this thesis	54
2.2. Introduction	54
2.3. Materials and methods	59
2.3.1. Study sample	59
2.3.2. Osteological measurements	62
2.3.3. Analytical methods	67
2.4. Results	71
2.4.1. Study sample	71
2.4.2. Replications of data	
2.4.3. Data visualization	
2.4.4. Measurement precision	
2.4.5. Measurement replication variation	
2.4.6. Measurement variation	
2.4.7. Measurement technique agreement	۵۸
2.4.0. Divariate driaryses	۱۵ عو
2.7 Collecting pelvic osteometric data from DXA in the following chapters of	00 If this
thesis	87

# Table of contents

CHAPTER THREE:	88
The growth patterns of the female pelvis	88
3.1. The role of Chapter Three in this thesis	88
3.2. Introduction	88
3.3. Materials & Methods	91
3.3.1. Sample used in this chapter	91
3.3.2. Study sample	91
3.3.2. Osteological measurements	94
3.3.3. Analytical methods	95
3.4. Results	99
3.4.1. Sample used in this chapter	99
3.4.2. LMS method	101
3.4.3. Growth velocity	109
3.5. Discussion	119
3.5.1. Tracking the growth of body and pervic dimensions	119
3.5.2. Growin velocity of the pervis and other parts of the body	101
2.5.4. Polyic dimensions growth and proparing the body for shildbirth	102
3.5.4. Pervic dimensions growin and preparing the body for childbirth	125
3.7 The growth trajectory of the pelvis and biological features that associate	with
pelvic breadth in growing girls	126
pervic breadin in growing gins	120
CHAPTER FOUR	127
The biological factors associated with female pelvic growth.	
4.1. The role of Chapter Four in this thesis	127
4.2. Introduction	127
4.2. Materials and methods	131
4.2.1. Study sample	131
4.2.2. Osteological measurements	134
4.3. Analytical methods	134
4.4. Results	138
4.4.1. Longitudinal dataset	138
4.4.2. Spaghetti plots	138
4.4.3. Conditional references	148
4.4.4. Cross-sectional dataset used in this chapter	150
4.4.5. Multivariate statistics	152
4.5. Discussion	199
4.5.1. Childhood pelvic dimensions to adult pelvic dimensions	199
4.5.2. Timelines of body breadth growth vs. pelvic dimension growth	200
4.5.3. Parental weight and height does not associate with pelvic dimension	s .202
4.5.4. Age at first menses does not associate with pelvic dimensions	202
4.6. Conclusion	203
4.7. Associations between biological variables and pelvic canal breadth varia	DIES
and associations between ecological variables and pelvic canal breadth varia	Selar
	204

CHAPTER FIVE:	.205
The biosocial factors associated with female pelvic growth	.205
5.1. The role of Chapter Five in this thesis	.205
5.2. Introduction	.205
5.3. Materials and methods	.210
5.3.1. Study sample	.210
Osteological data collection	.212
5.4. Analytical methods	.212
5.5. Results	.214
5.5.1. Cross-sectional dataset used in this chapter	.214
5.5.2. Univariate analyses - boxplots	.216
5.5.3. Multivariate analyses - scatterplots	.217
5.5.4. Univariate analyses	.242
5.6. Discussion	.251
5.6.1. Socioeconomic status and growth in girls	.251
5.6.2. Mother comes first – maternal education and growth	.252
5.6.3. Oral contraception and growth	.253
5.6.4. Biological variables have a greater effect on pelvic growth than biosoc	ial
variables	.253
5.7. Conclusion	.254
5.8. Associations between biosocial variables and pelvic canal breadth variable	es
vs. associations between biological variables and pelvic canal breadth variable	S
	.254
CHAPTER SIX	255
Summary and Conclusions	255
6.1 The role of Chapter Six in this thesis	255
6.2 Summary of project aims	255
6.3. Summary of findings	.256
6.3.1 Dual energy absorptiometry scans be used for osteological data collect	tion
	.256
6.3.2. Different growth trajectory of canal vs. non-canal parts of the pelvis	.257
6.3.3. The pelvic canal continues to grow into early adulthood	.258
6.4 Wider implications	259
6.4.1 Bi-iliac breadth is representative of body breadth during growth	259
6.4.2 Pelvic size and shape variation during growth suggests multiple cause	s of
birth difficulty	259
6.5 Areas for future study	260
6.5.1 Pelvic growth in boys	260
6.5.2 Female pelvic growth through early adulthood and into middle age	261
6.5.3 Tracking hormonal alterations alongside skeletal growth	261
6.6 Conclusion	261
Literature cited	.263
Appendix One	.299
Appendix One Appendix Two	.299 .303
Appendix One Appendix Two Appendix Three	.299 .303 .305

# List of tables

Table 2. 1. Linear measurements of pelvic dimensions and shoulder breadth andtheir descriptions.63
Table 2.2. Descriptive statistics of study sample and measurement variables71
Table 2.3. Results of the TEM calculation. 76
Table 2.4. Mean differences between initial and first replication sessions of DXA and MRI scans for each measurement variable.77
Table 2.5. Coefficient of variation, average difference between MRI and DXA pelvicbreadth measurements and bias of DXA measurements compared to MRImeasurements.77
Table 2.6. Results of Bland-Altman analysis, including man bias, limits of agreementand linear regression results. * Values are significant at <0.05.
Table 2. 7. Results of the Shapiro-Wilk's test for normaity of the baseline DXA andMRI pelvic dimension measurements.81
Table 2. 8. Results of the linear regression of the first replication of DXA pelvicdimension measurements regressed on MRI pelvic dimension measurements81
Table 3.1. Sample details of other smaller studies included in Fewtrell and colleaguesreference sample, as well as sample collected specifically for Fewtrell and colleaguesreference sample
Table 3.2. Linear measurements of pelvic dimensions, shoulder breadth and their descriptions.
Table 3.3. Descriptive statistics of the study sample. 100
Table 3.4. Shapiro-Wilk's normality test results of the cross-sectional dataset used in this chapter.      101
Table 3.5. Shoulder breadth reference data for study sample by zscore or standard deviation
Table 3.6. Bi-iliac breadth reference data for study sample by zscore or standard deviation
Table 3.7. Medio-lateral pelvic inlet reference data for study sample by zscore or standard deviation
Table 3.8. Bi-acetabular breadth reference data for study sample by zscore or standard deviation

Table 4.1. Description of anthropmetrics collected from participants in the reference study.      132
Table 4.2. Descriptive statistics for longitudinal data subsample
Table 4.3. Correlation matrix for shoulder breadth SDS at six ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age
Table 4.4. Correlation matrix for BIIB SDS at six ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age
Table 4.5. Correlation matrix for INML SDS at six different ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age
Table 4.6. Correlation matrix for BIAC SDS at six ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age
Table 4. 7. Sample size in the cross-sectional dataset organised by age group priorto removal of outliers
Table 4. 8. Results of the Shapiro-Wilk's test for normality of the cross-sectionaldataset used in this chapter.151
Table 4. 9. Descriptive statistics of the cross-sectional dataset used in this chapter.      151
Table 4.10. Linear regression results for shoulder breadth, including model summary, multicollinearity measures and Durbin-Watson statistics (n=175)
Table 4.11. Linear regression results for BIIB, including model summary,multicollinearity measures and Durbin-Watson statistics (n=178).198
Table 4.12. Linear regression results for INML, including model summary,multicollinearity measures and Durbin-Watson statistics (n=179).198
Table 4.13. Linear regression results for BIAC, including model summary,multicollinearity measures and Durbin-Watson statistics (n=179).199
Table 5.1. Educational attainment code of participant's parents
Table 5. 2. Social code of participants parents, modified from Standard OccupationalClassification, published by the National Office of Statistics (National Office ofStatistics, 2000).212

Table 5. 3. Sample size in the cross-sectional dataset organized by age group priorto the removal of outliers
Table 5. 4. Results of the Shapiro-Wilk's test for normality of the cross-sectionaldataset used in this chapter.215
Table 5. 5. Descriptive statistics of the cross-sectional dataset used in this chapter.
Table 5. 6. Results of the one-way ANOVA test of shoulder breadth, pelvic breadthdimensions and social code. Total degrees of freedom (df) represent sample size foranalysis.242
Table 5.7. Results of the one-way ANOVA test of shoulder breadth, pelvic breadthdimensions and maternal education. Total degrees of freedom (df) represent samplesize for analysis.243
Table 5. 8. Results of the one-way ANOVA test of shoulder breath, pelvic breadthdimensions and paternal education. Total degrees of freedom (df) represent samplesize for analysis
Table 5.9. Results for Tukey's range test for ANOVA analysis of shoulder breadth,pelvic breadth dimensions and social code.244
Table 5.10. Results for Tukey's range test for ANOVA analysis of shoulder breadth,pelvic breadth dimensions and maternal education
Table 5. 11. Results of Tukey's range test of ANOVA test of shoulder breadth, pelvicdimensions and paternal education.248
Table 5. 12. Group statistics for student t-test performed to examine meandifferences between shoulder breadth and pelvic dimensions in girls using or notusing oral contraception.250
Table 5. 13. Results of student t-test for equality of means of shoulder breadth, pelvicbreadth dimensions and oral contraceptive use. Degrees of freedom (df) representsample size250

List of figures Figure 1.1. The female pelvis. From Gray (1918)23
Figure 1.2. The male pelvis. From Gray (1918)23
Figure 1.3. The human pelvis in sagittal and coronal section, indicating the positions of the three pelvic planes: inlet, midplane and outlet. From Rosenberg (1992)
Figure 1.4. Timeline of chondrification and ossification events in the ontogenetic development of the human pelvis. From Verbuggen and Nowlan (2017)
Figure 1.5. The stages of labour in humans. From Rosenberg and Trevathan (1992)
Figure 1.6. Relative cranial dimensions in infant primates. From Wells et al. (2012)
Figure 2.1. Measurements collected from MRI and DXA scans; A) Bi-iliac breadth (BIB); B) Bi-acetabular breadth (BIAC); C) Mediolateral inlet breadth (INML)64
Figure 2.2. BIIB measurement as collected on MRI scans64
Figure 2.3. BIAC measurement as collected on MRI scans65
Figure 2.4. INML measurement as collected on MRI scans65
Figure 2.5. BIIB measurement as collected on DXA scans
Figure 2.6. BIAC measurement as collected on DXA scans
Figure 2.7. INML measurement as collected on DXA scans67
Figure 2.8. Plot of the differences between method A and method B vs. the mean of two measurements. From Giavarina (2015)70
Figure 2.9. Scatterplot of MRI measurements of BIIB in every individual in the sample72
Figure 2.10. Scatterplot of DXA measurements of BIIB in every individual in the sample
Figure 2.11. Scatterplot of MRI measurements of BIAC in every individual in the sample

Figure 2.12. Scatterplot of DXA measurements of BIAC breadth in every individual in the sample	n 1
Figure 2.13. Scatterplot of MRI measurements of INML in every individual in the sample	74
Figure 2.14. Scatterplot of DXA measurements of INML in every individual in the sample	75
Figure 2.15. Bland-Altman plot of MRI compared to DXA BIIB7	79
Figure 2.16. Bland-Altman plot of MRI compared to DXA BIAC7	79
Figure 2.17. Bland-Altman plot of MRI compared to DXA INML8	30
Figure 2.18. Linear regression plot of DXA BIIB regressed on MRI BIIB8	32
Figure 2.19. Linear regression plot of DXA BIAC regressed on MRI BIAC8	32
Figure 2.20. Linear regression plot of DXA INML regressed on MRI INML8	33
Figure 3.1. Height-for-age and weight-for-age growth centile chart for girls in the United Kingdom aged 8 to 18 years. From Royal College of Paediatric and Chile Health (2012)	ld 90
Figure 3.2. Centile chart of shoulder breadth	97
Figure 3.3. Centile chart of BIIB breadth	98
Figure 3.4. Centile chart of INML	99
Figure 3.5. Centile chart of BIAC10	00
Figure 3.6. Growth velocity chart of shoulder breadth10	)5
Figure 3.7. Growth velocity chart of BIIB10	06
Figure 3.8. Growth velocity chart of INML10	)7
Figure 3.9. Growth velocity chart of BIAC10	8
Figure 3.10. Growth velocity chart of weight10	9
Figure 3.11. Growth velocity chart of height11	0
Figure 3.12. Growth velocity chart of fat mass11	11
Figure 3.13. Growth velocity chart of lean mass	12

Figure 3.14. Growth curves for different body tissues. From Cabana et al. (1993)
Figure 4.1. Tanner development stage chart. From Marshall and Tanner (1969)
Figure 4.2. Spaghetti plot of shoulder breadth134
Figure 4.3. Spaghetti plot of shoulder breadth SDS135
Figure 4.4. Spaghetti plot of BIIB136
Figure 4.5. Spaghetti plot of BIIB SDS137
Figure 4.6. Spaghetti plot of INML138
Figure 4.7. Spaghetti plot of INML SDS139
Figure 4.8. Spaghetti plot of BIAC140
Figure 4.9. Spaghetti plot of BIAC SDS141
Figure 4.10. Scatterplot of shoulder breadth SDS regressed on birth weight147
Figure 4.11. Scatterplot of bi-iliac breadth SDS regressed on birth weight148
Figure 4.12. Scatterplot of medio-lateral inlet breadth SDS regressed on birth weight
Figure 4.13. Scatterplot of bi-acetabular breadth SDS regressed on birth weight
Figure 4.14. Scatterplot of shoulder breadth SDS regressed on height SDS151
Figure 4.15. Scatterplot of bi-iliac breadth SDS regressed on height SDS152
Figure 4.16. Scatterplot of medio-lateral inlet breadth SDS regressed on height SDS
Figure 4.17. Scatterplot of bi-acetabular breadth SDS regressed on height SDS154
Figure 4.18. Scatterplot of shoulder breadth SDS regressed on head circumference SDS
Figure 4.19. Scatterplot of bi-iliac breadth SDS regressed on head circumference SDS

Figure 4.20. Scatterplot of medio-lateral inlet breadth SDS regressed on head circumference SDS157	,
Figure 4.21. Scatterplot of bi-acetabular breadth SDS regressed on head circumference SDS	}
Figure 4.22. Scatterplot of shoulder breadth SDS regressed on maternal weight159	)
Figure 4.23. Scatterplot of bi-iliac breadth SDS regressed on maternal weight160	)
Figure 4.24. Scatterplot of medio-lateral inlet breadth SDS regressed on maternal weight	
Figure 4.25. Scatterplot of bi-acetabular breadth SDS regressed on maternal weight	
Figure 4.26. Scatterplot of shoulder breadth SDS regressed on maternal height163	3
Figure 4.27. Scatterplot of bi-iliac breadth SDS regressed on maternal height164	1
Figure 4.28. Scatterplot of medio-lateral inlet breadth SDS regressed on maternal height	
Figure 4.29. Scatterplot of bi-acetabular breadth SDS regressed on maternal height166	
Figure 4.30. Scatterplot of shoulder breadth SDS regressed on paternal weight167	
Figure 4.31. Scatterplot of bi-iliac breadth SDS regressed on paternal weight168	,
Figure 4.32. Scatterplot of medio-lateral inlet breadth SDS regressed on paternal weight	)
Figure 4.33. Scatterplot of bi-acetabular breadth SDS regressed on paternal weight	)
Figure 4.34. Scatterplot of shoulder breadth SDS regressed on paternal height171	1
Figure 4.35. Scatterplot of bi-iliac breadth SDS regressed on paternal height172	2
Figure 4.36. Scatterplot of medio-lateral inlet breadth SDS regressed on paternal height	
Figure 4.37. Scatterplot of bi-acetabular breadth SDS regressed on paternal height	ŀ
Figure 4.38. Scatterplot of shoulder breadth SDS regressed on hip circumference SDS	5

Figure 4.39. Scatterplot of bi-iliac breadth SDS regressed on hip circumference SDS
Figure 4.40. Scatterplot of medio-lateral inlet breadth SDS regressed on hip circumference SDS
Figure 4.41. Scatterplot of bi-acetabular breadth SDS regressed on hip circumference SDS
Figure 4.42. Scatterplot of shoulder breadth SDS regressed on thigh circumference SDS
Figure 4.43. Scatterplot of bi-iliac breadth SDS regressed on thigh circumference SDS
Figure 4.44. Scatterplot of medio-lateral inlet breadth SDS regressed on thigh circumference SDS
Figure 4.45. Scatterplot of bi-acetabular breadth SDS regressed on thigh circumference SDS
Figure 4.46. Boxplots of Tanner development stage and shoulder breadth SDS183
Figure 4.47. Boxplots of Tanner development stage and bi-iliac breadth SDS184
Figure 4.48. Boxplots of Tanner development stage and medio-lateral inlet breadth SDS
Figure 4.49. Boxplots of Tanner development stage and medio-lateral inlet breadth SDS
Figure 4.50. Scatterplot of shoulder breadth SDS regressed on age at menarche187
Figure 4.51. Scatterplot of bi-iliac breadth SDS regressed on age at menarche188
Figure 4.52. Scatterplot of medio-lateral inlet breadth SDS regressed on age at menarche
Figure 4.53. Scatterplot of bi-acetabular breadth SDS regressed on age at menarche
Figure 5.1. Scatterplot of shoulder breadth SDS regressed on calcium intake218
Figure 5.2. Scatterplot of bi-iliac breadth SDS regressed on calcium intake214
Figure 5.3. Scatterplot of medio-lateral inlet breadth SDS regressed on calcium intake

Figure 5.4. Scatterplot of bi-acetabular breadth SDS regressed on calcium intake
Figure 5.5. Boxplots of social code and shoulder breadth SDS
Figure 5.6. Boxplots of social code and bi-iliac breadth SDS
Figure 5.7. Boxplots of social code and medio-lateral inlet breadth SDS221
Figure 5.8. Boxplots of social code and bi-acetabular breadth SDS222
Figure 5.9. Boxplots of maternal education and shoulder breadth SDS223
Figure 5.10. Boxplots of maternal education and bi-iliac breadth SDS224
Figure 5.11. Boxplots of maternal education and medio-lateral inlet breadth SDS225
Figure 5.12. Boxplots of maternal education and bi-acetabular breadth SDS226
Figure 5.13. Boxplots of paternal education and shoulder breadth SDS227
Figure 5.14. Boxplots of paternal education and bi-iliac breadth SDS228
Figure 5.15. Boxplots of paternal education and medio-lateral inlet breadth SDS229
Figure 5.16. Boxplots of paternal education and bi-acetabular breadth SDS230
Figure 5.17. Scatterplot of shoulder breadth SDS regressed on hours of vigorous physical activity per week231
Figure 5.18. Scatterplot of bi-iliac breadth SDS regressed on hours of vigorous physical activity per week232
Figure 5.19. Scatterplot of medio-lateral pelvic breadth SDS regressed on hours of vigorous physical activity per week
Figure 5.20. Scatterplot of bi-acetabular breadth SDS regressed on hours of vigorous physical activity per week234
Figure 5.21. Boxplots of oral contraceptive use and shoulder breadth SDS235
Figure 5.22. Boxplots of oral contraceptive use and bi-iliac breadth SDS236
Figure 5.23. Boxplots of oral contraceptive use and medio-lateral inlet breadth SDS
Figure 5.24. Boxplots of oral contraceptive use and bi-acetabular breadth SDS238

## CHAPTER ONE:

#### The growing skeleton and childbirth

#### 1.1. The structure of this thesis

This thesis does not follow the traditional structure of a doctoral thesis. Instead each chapter covers a research theme and is structured as a research article, with methods, results reporting and discussion included in each chapter. The bulk of the literature review for the theoretical basis of this thesis is covered in Chapter One, with theme-specific literature reviews covered in the Introduction sections of Chapters Two, Three, Four and Five. Chapter Two demonstrates the feasibility of the main method used in this thesis to collect data, using a dataset separate to that used in Chapters Three, Four and Five. Chapters Three, Four and Five use a dataset that is fully described in Chapter Three. The conclusions drawn from this thesis are drawn primarily from the results of Chapters Three, Four and Five.

## 1.2. The context and central research questions of this thesis

This thesis focuses on the growth of the human female pelvis in the context of the skeletal components involved in childbirth. In order to outline the full context of this thesis it is necessary to briefly define the obstetric dilemma and then clarify the context of this thesis in relation to this definition. The central research questions of this thesis are detailed after the definition of the obstetric dilemma. The structure of Chapter One is explained in the end of this section to bolster the contextual basis of this thesis.

Childbirth is biomechanically challenging in humans due to the tight fit between the maternal birth canal and the size of the neonatal head. Archaeological evidence of death as a result of childbirth in the past includes younger ages at death for women compared to men on a population level, as well as excavations of skeletons revealing foetal remains in the maternal pelvis (Wells et al., 2012). Whilst medical assistance has greatly reduced the risks associated with childbirth, childbirth still accounts for 9% of maternal mortality globally (WHO, 2014). Washburn (1960) suggested that the tight fit between maternal birth canal and neonatal head is unique to humans, as the human pelvis is predominantly adapted for energetic efficiency during bipedal walking. Simultaneous to bipedal adaptations, humans have also undergone strong selection for greater brain size (Gruss and Schmitt, 2015), resulting in conflicting pressures rendering childbirth biomechanically challenging. Washburn (1960) termed the result of these conflicting pressures the obstetric dilemma (OD). The OD remained a fixture in characterizing the evolution of modern humans, suggesting that childbirth difficulty is a necessary danger for *Homo sapiens* and that the major issue with childbirth in the past was the biomechanical result of the OD. More recent research has presented a set of alternative influences on the OD (Wells et al. 2012) including ecological influences. The OD does not represent the biomechanical reality of the tight fit between the human birth canal and the human neonatal head. The OD should not be used interchangeably with any medical definition of childbirth difficulty, as issues with successfully delivering an infant can be caused by multiple factors. It remains unclear whether the concurrent evolution of bipedality and increased brain size is the cause of the mismatch between the size of the maternal birth canal and the human neonate. This is further complicated by a relative scarcity of studies examining the influences on pelvic morphology in humans.

The contextual basis of this thesis is that there is no single OD. The basis of this thesis is that there are *multiple* possible developmental and ecological factors that may contribute to the biomechanical difficulties of childbirth, as well as conflicting pressures acting on both the maternal and neonatal sides. The content of the thesis relates to the OD in theme, though in its particulars examines the development of and associations with pelvic morphology with a view to elucidate possible factors contributing to childbirth difficulty. The OD is reviewed in this chapter and is mentioned throughout this thesis, however the OD should be viewed as a starting point for the research questions that this thesis addresses. In addition, the results of this thesis are not applicable to every living human population, or as a template that can be directly applied to archaeological populations.

This thesis seeks to answer three major questions: what is the growth trajectory of the female bony pelvis? What are the ecological factors that associate pelvic growth? Can these results demonstrate if childbirth difficulty as a result of obstetrically compromised pelvic dimensions are ecological in **origin?** The OD stems from a compromise in pelvic development and this project will illuminate which factors are the most important in pelvic development throughout growth and could affect CPD and OD in adulthood. This project also integrates the skeletal component of pelvic growth with overall body size and shape change as women grow into girls, effectively 'putting the skeleton back into' a fleshed body. Body size and shape change occurs as part of pubertal development and can be measured in soft and hard tissue changes. Previous work has demonstrated that these tissues interact during growth, meaning that pelvic development may track alongside body size and shape change as girls grow into women. Measuring pelvic development in living girls and women calls for methods that can visualise the pelvis in situ in a similar manner to dry bone. In this project medical imaging (dual-energy x-ray absorptiometry) methods are used to collect pelvic and body breadth data. This is a novel approach in biological anthropology and unites perspectives on growth and development in living and past human populations.

The structure of Chapter One is designed to establish a basis of information on the human pelvis and then detail different knowledge areas pertaining to human pelvic variation, birth in humans (both in the past and today), previous and more recent work on the OD, birth in non-human primates and non-human mammals and an evolutionary medicine perspective on the development of the pelvis.

## 1.3. The human pelvis

The pelvis is made of four main elements: the sacrum, the coccyx and the right and left os coxae (or hip bones). The os coxae and the sacrum are connected together to create a ring that provides the anchoring point for reproductive and some digestive organs and transfers loads from the trunk to the lower limbs. Each os coxae is in turn constructed from the ilium, ischium and the pubis (see Figures 1.1 -1.2). The canal in the middle of the pelvis is divided into three planes – the inlet, the midplane and the outlet (see Figure 1.3). These planes are of particular interest for obstetric adaptations, as the human neonate rotates through the midplane of the pelvis during childbirth (see Figure 1.5).



Figure 1.1. The female pelvis. From Gray (1918). Permission for this image is out of copyright.



Figure 1.2. The male pelvis. From Gray (1918). Permission for this image is out of copyright.



Figure 1.3. The human pelvis in sagittal and coronal section, indicating the positions of the three pelvic planes: inlet, midplane and outlet. From Rosenberg (1992). Permission to reproduce this figure has been granted by the rights holder, John Wiley and Sons.

# 1.4. The development of the human pelvis in utero, childhood, adolescence and early adulthood

The pelvis develops from the same tissue as the other lower limb bones, beginning as a small bud of tissue in the maternal intra-uterine wall during the third week of gestation (Strayer, 1943, 1971). Chondrification (the formation of cartilage from condensed mesenchyme tissue) of the structures leading to the pelvis occurs between intra-uterine sixth and seventh weeks of gestation (Bardeen, 1905). The cartilage model of the pelvis nears completion at approximately the onset of the third uterine month (Adair, 1918). The primary centres of ossification in the pelvis are located in the centre of the ilium, ischium and the pubis. The ilium ossifies first during the beginning of the third month of gestation, whilst ossification of the ischium occurs during the fourth to fifth months of gestation (Verbruggen and Nowlan, 2017). The pubis ossifies last during development in the fifth to sixth month of gestation whilst the sacrum continues to ossify from the third month of gestation onwards (Verbruggen and Nowlan, 2017). At birth, the primary centres of ossification are clearly visible via radiograph and overall growth of these centres is noticeably rapid until three years of age, followed by a reduction in growth speed (Verbruggen and Nowlan, 2017). Fusion of the three centres of ossification at the acetabulum can occur as early as three years of age though more commonly takes place between five and eight years of age. Complete fusion of the acetabulum is usually achieved between 11 to 14 years old in females and 14 to 17 years of age in males (Stevenson, 1924; Flecker, 1932; Freedman, 1934). See Figure 1.5. for a more complete outline of the developmental timeline of the human pelvis.

Whilst the trajectory of pelvic growth is similar for girls and boys throughout childhood, some important sexually dimorphic differences have been noted throughout the growth process. Hromada (1939) found increasingly discernible sexual dimorphism in growth phases between 7 months and birth. Moerman (1982) found that within two years of menarche, girls reached 91-99% their adult pelvic dimensions, demonstrating a slower pattern of growth than that seen in stature in girls. This same pattern of growth was found in more recent studies examining linear skeletal development compared to skeletal breadth (Völgyi et al., 2010). There are very few other studies that have examined the growth phases of the bony pelvis throughout childhood and adolescence.



Figure 1.4. Timeline of chondrification and ossification events in the ontogenetic development of the human pelvis. From Verbuggen and Nowlan (2017). Permission to reproduce this figure has been granted by the rights holder, John Wiley and Sons.

#### 1.5. The development of the human pelvis and the actions of puberty

The growth and development of the pelvis are greatly affected by the life history period known as puberty. Puberty refers to the activation of the hypothalamicpituitary-gonadal-axis that stimulates development of the gonads and the effects of sex steroids (Hochberg and Belsky, 2013). During puberty somatic growth increases markedly and changes in the quantity and distribution of fat throughout the body occurs (Marshall, 1978). The term adolescence refers to the biosocial components of maturation, including social learning, intimacy, social support and the development of skills needed for reproduction (Hochberg and Belsky, 2013).

The length of adolescence after the onset of puberty in humans has been characterised as a unique trait, suggesting that it arose with Homo sapiens (Bogin, 1999a). Humans display a defined period of adolescence that takes place after the juvenile growth stage and shortly after the onset of puberty, and also includes an adolescent growth spurt (Bogin, 1999a). There is no distinct period of adolescence amongst New and Old World monkeys (Bogin, 1999a) though some work has pointed to an adolescent-like growth spurt amongst chimpanzees (Hamada and Udono, 2002). Delaying full reproductive maturity presents a number of advantages for humans as it allows time for adolescents to learn the biosocial skills necessary for raising children. The length of puberty in humans also includes the variation in the timing of different changes for girls compared to boys. For example, puberty in girls was thought to begin with the larche (the development of breast buds) (Hochberg and Belsky, 2013). However ovaries are already generating oestrogen approximately two years prior to the larche, meaning that puberty would technically begin two years prior to the larche as it is during this time that the maturation of the hypothalamic-pituitarygonadal-axis occurs (Hochberg and Belsky, 2013). This results in the acceleration in growth (known as peak height velocity or PHV) in girls taking place approximately six months after the larche, with menarche following about a year after PHV (Hochberg and Belsky, 2013). Frequently this means that girls may have the external appearance of sexual maturity but may not be fertile. Conversely boys become fertile around 14 to 15 years of age, about two years after their PHV though around this age they may not externally appear sexually mature (Hochberg and Belsky, 2013). Bogin

(1994) suggests that the sexual dimorphism in adult height may be a consequence of this difference in timing for PHV.

Fat distribution is another key change that occurs with puberty and relates to reproduction in humans. The processes underlying reproduction and food intake in mammals are controlled by a sensory system that monitors metabolic signals. These signals are generated by changes in metabolic fuel availability within body cells rather than by changes in the amount of body fat or by changes in any aspect of body composition (Schneider et al., 2000). This metabolic monitoring system in turn allows energy to be apportioned to different physiological activities, including reproduction. During times with low access to food, reproduction is not prioritized. Studying the effects of food deprivation on reproduction in Syrian and golden hamsters, Schneider and Wade (1989; 1990) found that food deprivation and weight loss inhibited oestrus. Amongst humans the effects of food deprivation on this metabolic monitoring system are more complex. Menstrual dysfunction has been found to occur amongst girls suffering from anorexia nervosa (an eating disorder) (Kimmel et al., 2016), however menstrual dysfunction does not always occur amongst girls with restricted diets (such as those competing in high-level sports). For example, amongst 220 university-aged competitive soccer players who had reached menarche, only 19.3% presented with menstrual dysfunction (Prather et al., 2016).

In opposition to this metabolic perspective to the link between energy and reproduction, Kennedy (1953) Frisch (1990) put forward that food intake and reproduction were controlled by a hypothetical monitor of body fat which reduced the likelihood that reproduction could take place below a certain level of body fat. More recent work suggested that plasma leptin levels provide information about body fat content (see Schneider et al. (2000) for sources). Leptin is a hormone constructed primarily of adipose cells that regulates energy balance by inhibiting hunger and through this maintains fat storage in the body. Previous work has demonstrated that leptin treatment on food-deprived anoestrous rats and hamsters reverses the effects of food deprivation on fertility (see Schneider et al. (2000) for sources). Schneider et al. (2000) suggested that leptin secretion was controlled by the intracellular availability of oxidizable metabolic fuels rather than by levels of adiposity, making it a better candidate for examining the energy balance struck between fat availability and

reproduction in mammals and possibly in humans.

Fat tissue also acts as energy storage – energy can be released in the short or long term between access to food resources by storing or using fat tissue (Norgan, 1997). Fat covers essential organs such the heart, liver, lungs, kidneys to function as shock absorption, and fatty tissue is also necessary for the central nervous system (Norgan, 1997). Women carry sex-specific fat around the breasts, hips, thighs and buttocks whilst men accrue fat tissue around their trunk (Norgan, 1997). Higher body fat in women acts as energy storage for pregnancy and lactation. It is seems to be linked with the onset of menarche. Menarche amongst European and American girls was found to occur with critical body fat percentage of 17% with regular menstrual cycles only possible with 22% body fat (Frisch and McArthur, 1974). Ellison (2001) did not find evidence to support this claim though Wells (2010) outlines that Frisch's (Frisch and McArthur, 1974) hypotheses may be more relevant for fertility issues in girls suffering from eating disorders and malnutrition. Wells' (2010) point on fertility issues being greater amongst girls suffering from eating disorders would align with Schneider and Wade's (1989;1990) work on non-human mammals, showing that food deprivation and weight loss inhibits oestrus.

Skeletal tissue is also affected by puberty through hormonal action. A selection of hormones regulate the process of bone deposition and bone resorption and allow for bone growth and bone strengthening alongside mechanical loading. Continuous secretion of parathyroid hormone (PTH) stimulates bone resorption, which maintains serum calcium levels. Bone formation occurs when PTH is secreted intermittently (Hadjidakis and Androulakis, 2006). Growth factors IGF-1 (insulin-like growth factor) and IFG-2 also regulate bone resorption and formation at the cartilaginous end plates and during endochondral bone formation. They function in a systemic and local feedback control mechanism, down-regulating certain actions of each hormone throughout the growth process (Giustina et al., 2008). IGF-1 and IGF-2 also significantly influence adult bone mass (Wang et al., 2004). During development IGF-1 and IGF-2 work independently from other growth hormones, and during puberty GH (a single chain peptide of 191 amino acids) and IGF-1 significantly determine longitudinal skeletal growth (Giustina et al., 2008).

During the adolescent growth spurt the hypothalamus is stimulated to produce gonadotropin-releasing hormone, which is then released throughout the adolescent period in pulses. Different regions of the skeleton attain peak growth rate at different stages of the adolescent growth phase (Satake et al., 1994). Pubertal growth patterns have been outlined for populations in the United Kingdom (Tanner, 1962; Tanner and Whitehouse, 1976), the United States of America (Simmons and Greulich, 1943; Reynolds and Wines, 1948; Nicolson and Hanley, 1953; Abbassi, 1998; Juul et al., 2006) and throughout Europe (Bielicki, 1975; Taranger et al., 1976; Bundak et al., 2007; Tóth et al., 2012). Many of these studies demonstrate the variation in pubertal growth phases as results of differing socioeconomic status, geography, diet, health status, time period and sex. Androgen and oestrogen hormones have a direct influence on bone growth by influencing cell division and maturation at the bone growth plate (Bogin, 1999b). On average, girls begin the adolescent growth spurt two years before boys, and this is accompanied by an increased bone density in boys (Bogin, 1999b). The principal effect of testosterone in boys during early adolescence is to promote bone growth, and during later adolescence testosterone stimulates epiphyseal fusion (Preece et al., 1984). High levels of estradiol (an oestrogen hormone) are responsible for the adolescent growth spurt in girls (Prader, 1984). Hormone variation in populations may contribute to the timing of skeletal growth in boys and girls, particularly the differentiation between linear growth compared to growth in body breadth.

## 1.6. Sexual dimorphism of the human pelvis

Much of the examination of selection for pelvic sexual dimorphism compares pelvic dimorphism with body size dimorphism. Previous work demonstrated the link between pelvic sexual dimorphism and body size sexual dimorphism amongst primates (Schultz, 1949; Leutenegger, 1973): higher body size dimorphism corresponded with higher pelvic dimorphism and vice versa. However, Tague (2005) proposed that the default primate pelvic anatomy is female, and that male pelvic shape was attained by systematic stimulation and reduction of pelvic growth by testosterone levels throughout development.

Selection for specific pelvic features in humans has also been associated with bipedal locomotion. The narrowness of the human birth canal was long thought of as

a biomechanical compromise necessary for bipedal locomotion (Meindl et al., 1985; Lovejoy, 1988, 2005; Rosenberg and Trevathan, 1995; Correia et al., 2005; Wittman and Wall, 2007; Grabowski, 2013). Warrener et al. (2015) demonstrated that there was no locomotor cost imposed by a wider pelvis, suggesting that the variation seen in female pelvic shape (including that which is not obstetrically sufficient) is caused by factors other than a biomechanical necessity. The anatomically modern pelvic canal does not appear in the fossil record until approximately 200,000 years ago in *Homo sapiens* groups evolving in the Middle East and parts of Africa, which has been suggested to reflect selective pressures for a narrow body type to better deplete body heat in warm climates (Gruss and Schmitt, 2015).

The compromise between body size and pelvic canal size as a selective force is frequently discussed with respect to obstetric capacity in human females. The following features are larger in the female pelvic canal than the male pelvic canal: circumferences, breadths and posterior lengths of the pelvic inlet, midplane and outlet, bi-acetabular breadth, the anterior to posterior length of the midplane and outlet, linea terminalis length, pubic bone length, breadth of the sciatic notch, subpubic angle and sacrum angulation and lower iliac height length (see Kurki, 2005 for full list of references). The fit between the long-axis of the neonate cranium and the planes of the pelvic canal are such that human birth takes place via a 'rotational' mechanism; the neonate's head first rotates through the pelvic midplane and out of the canal, followed by the shoulders entering the pelvic canal, with the shoulders then beginning to rotate internally within the canal to align anterior-posteriorly with the outlet (Rosenberg, 1992; Rosenberg and Trevathan, 1995). The midplane is then the most obstetrically important plane of the pelvic canal, as its dimensions must allow for the passage of the neonate between the ischial spines (Walrath and Glanz, 1996; Taque, 2000; Kurki, 2007). Independent of body size, the most sexually dimorphic and obstetrically important features of the pelvic canal are the posterior space of the inlet, the medio-lateral diameter of outlet, the subpubic angle, and the sacral angle (Tague, 1992).

Selective pressures may still have affected overall male pelvis and pelvic canal shape, as selection pressures acting on one sex produces associated responses in the opposite sex (Lande, 1980). Kurki (2013) tested the hypothesis that

skeletal variability in the female pelvic canal would be limited due to stabilizing selection. She suggested that variation in pelvic canal size and shape amongst females would be lower than amongst males as stabilizing selection would act to reduce variation in the female pelvic canal to ensure adequate obstetric dimensions (Kurki 2013). Kurki's (2013) study demonstrated that found that the pelvic canal is the most variable skeletal region in both males and females, and that whilst patterns of variability across the canal and non-canal aspects of the male pelvis did not match those of the female pelvis, there remained some variation present within the male pelvis itself. The pelvic canal was also found to be more variable in shape and size than the non-canal portions of the pelvis in both males and females (Kurki and Decrausaz, 2016).

#### 1.7. The human pelvis and childbirth: Biological and social components

The process of childbirth amongst humans has a set timeline though variations may occur as a result of biomechanical difficulties, health issues and various factors that predominantly impact the mother or the infant. This section first details the process of birth in humans and then outlines some of the biosocial components which can affect childbirth.

Birth occurs in humans after approximately 40 weeks of gestation. In natural childbirth the neonate passes through the maternal birth canal and is born through the vagina. Labour can last between six and 20 hours in length and is divided in to three stages. The latent phase occurs first, where the cervix canal reduces in length and the concentration hormone relaxin increases to soften the collagen components of the cervix. This is followed by the first stage labour, indicated by the onset of regular uterine contractions and the dilation of the uterine cervix. The second stage of labour begins with the full dilation of the cervix and continues with regular uterine contractions to aid in the mother in pushing the neonate out of the birth canal. The third and final stage of labour takes place with birth of the infant and the delivery of the placenta (Martini et al., 2009).

The fit between the long-axis of the neonate cranium and the planes of the maternal pelvic canal are such that human birth takes place via a 'rotational' mechanism; the neonate's head first rotates through the pelvic midplane and out of

the canal, followed by the shoulders entering the pelvic canal, with the shoulders then beginning to rotate internally within the canal to align anterior-posteriorly with the outlet (Rosenberg, 1992; Rosenberg and Trevathan, 1995) (see Figure 1.5.).



Figure 1.5. The stages of labour in humans. In each box, a sagittal section through the maternal body during labour is shown. Anterior is at the top of each picture and inferior is to the right. The maternal public bone and vertebral column are shown in black. In the upper right hand corner of each box is a "midwife's eye" view of the foetus as it rotates within and emerges through the birth canal. From Rosenberg and Trevathan (1992). Permission to reproduce this figure has been granted by the rights holder, John Wiley and Sons.

There is diversity in the risk of childbirth amongst living populations. In developed countries women have a 1 in 4900 lifetime risk of dying in pregnancy and childbirth, whilst in developing nations women have a 1 in 45 lifetime risk of dying in pregnancy and childbirth (WHO, 2014). The largest risk factor for death for pregnant women globally is a pre-existing condition that is exacerbated by pregnancy, such as diabetes, malaria and obesity (28%), followed by severe bleeding (27%), pregnancyinduced high blood pressure (14%), infections occurring mostly after childbirth (11%), obstructed labour (9%), abortion complications (8%) and blood clots (3%) (WHO, 2014). Half of all maternal deaths due to childbirth complications in 2008 occurred in India, Pakistan, Afghanistan, Nigeria, Ethiopia and the Democratic Republic of the Congo (Hogan et al., 2010), countries that are known to be particularly economically restricted and have large populations. Bhutta et al. (2004) identified malnutrition and infectious disease rates and factors pertaining to gender inequality such as female illiteracy, poverty as barriers to major improvements in maternal health and childbirth difficulties in Asia. In a multi-country study, Khan et al. (2006) reported that obstructed labour was the cause of death in 4.1% of maternal deaths in Africa, 9.4% of maternal deaths in Asia, 13.4% of maternal deaths in Latin America and Caribbean and 0% of deaths in developed countries. Haemorrhage caused the highest percentage of maternal deaths in Africa (33.9%), Asia (30.8%) and Latin America and the Caribbean (Khan et al., 2006).

Human childbirth is a social event as a result of the assistance needed in the delivery of a neonate (Rosenberg and Trevathan, 1995). Childbirth practices have been greatly altered by medical intervention. Historically, medical doctors were only involved in the event of childbirth if a natural birth was impossible, if midwives and other female relatives were unable to help the woman in labour (Ellison 2001).

An element of childbirth that has been affected by medical intervention is delivery position. Some birth positions may demand greater or lesser muscular force, and in addition may increase or decrease the relative size of the obstetric outlet. Michel et al. (2002) found that a squatting position, and a position that allows a woman in labour to pull back her knee with her hand, increased the dimensions of the sagittal and interspinous outlet of the pelvis, which could be beneficial particularly in the second stage of labour (full cervical dilation). Indeed, on average human mothers squat when delivering a neonate (Rosenberg and Trevathan, 1995). It seems that the supine position for childbirth has been adopted as a consequence of anaesthetic administration rather than obstetric advantage (Michel et al. 2002). Depending on the time period and location, birth position may still affect maternal and neonate mortality during childbirth.

# 1.8. Variation in the human pelvis of global populations and hunter-gatherer communities

Variation in female pelvic size and shape has been examined in many living populations as a means of identifying possible obstetric difficulty. Maternal height (as a reflection of maternal pelvic outlet size) has been clinically shown to predict obstructed labour (Toh-Adam et al., 2012), suggesting that pelvic outlet size and skeletal body size are developmentally linked. However, pelvic canal dimorphism does not consistently correlate with female body size. Baragi and colleagues (2002) identified pelvic floor shape differences between European-American and African-American women from the Hamann-Todd Osteological Collection: African-American women were found to have a shorter bi-spinous breadth and ischial spine-to-sacrum length. Rizk et al. (2004) found that European/White women living in the United Arab Emirates had larger pelvic inlet and outlet breadths than women from non-European/White ethnicities also living in the United Arab Emirates.

On the subject of maternal mortality due to childbirth in hunter-gather populations, Headland (1989) does not examine pelvic dimensions amongst the Casiguran Agta, a Phillipine Negrito hunter-gatherer community, but calculated that childbirth caused 14% of deaths amongst adult females 40 years prior to 1977 and 12% of deaths from 1977 to 1984. Headland (1989) identified poor maternal nutritional status and poor sanitary practices surrounding the birth experience. Hill et al. (2007) estimated that the risk death as a result of childbirth was 4.4 per 1000 reproductive-aged women amongst the Hiwi hunter-gatherers from Colombia and Venezuela.
#### **1.9.** The obstetric dilemma, the human pelvis and caesarean deliveries

The OD outlines the interplay between the differing pressures acting on the female pelvis; obstetrics and locomotion on one hand, and the delivery of a comparatively encephalized infant on the other. This results in a potentially difficult childbirth process for humans (Washburn, 1960). It should be noted that a mismatch between the size of a neonate and the maternal birth canal is not unique to humans: the interpubic ligament of free-tailed bats must stretch to 15 times its original length during birth (Crelin, 1969). The supposed uniqueness of the difficulty of human childbirth prompted scholars to characterise the OD as an anatomical compromise for efficient bipedality (Washburn, 1960). The narrowness of the human birth canal was long thought of as a biomechanical compromise necessary for bipedal locomotion (Meindl et al., 1985; Lovejoy, 1988, 2005; Rosenberg and Trevathan, 1995; Correia et al., 2005; Wittman and Wall, 2007; Grabowski, 2013). This anatomical compromise has also been identified as the reason for the rotational mechanism of human birth.

It would be logical to suppose that the female pelvis demonstrates a particular trend in shape for optimized parturition that also balances the morphological necessities for bipedal locomotion (an average shape matrix with little variation around it), reducing the potential for labour complications resulting in the death of mother or infant. Warrener et al. (2015) demonstrated that there was no locomotor cost imposed by a wider pelvis, suggesting that the variation seen in female pelvic shape (including that which is not obstetrically sufficient) is caused by factors other than a biomechanical necessity. Fischer and Mitteroecker (2015) also investigated the link between two factors in childbirth and consequently the OD. They examined the covariation between human pelvis shape (including both males and females in their analysis) and head circumference. Stature was significantly associated with pelvis shape in both males and females but head circumference was significantly associated with pelvis shape in females (Fischer and Mitteroecker, 2015). Results from their work also demonstrated that females with a larger head circumference had birth canals that were shaped to better accommodate large-headed infants (Fischer and Mitteroecker, 2015). In response to the results of Fischer and Mitteroecker's (2015) work, Ponce de León et al., (2016) stated that Fischer and Mitteroecker's

argument that a postreproductive lifespan had only limited inclusive fitness was invalid given the long postreproductive lifespan in humans and suggesting that Fischer and Mitteroecker's conclusions were presented as biological spandrels as per Gould's work (Gould and Lewontin, 1979).

It has also been suggested that the OD reflects plastic processes in response to historical environments (Wells et al., 2012). Wells and colleagues proposed that the OD varies in magnitude with different ecological settings and is not fixed throughout human history. Wells et al. (2012) use the example of the advent of agriculture to outline the changing OD. With the advent of agriculture, female growth and development was compromised by poor diet quality (compared to higher quality diets prior to the advent of agriculture). Skeletal evidence shows that the transition to agriculture resulted in a decline in overall health in many populations (Larsen, 2006; Shuler et al., 2012), with an increase in infectious disease (Larsen, 2006; Helle et al., 2014). Wells et al. (2012) suggest that the resulting delay skeletal development led to compromised female pelvic capacity. Whilst diet previous to agriculture allowed for adequate female growth and development, labour complications did occur. However, the *risk* for labour complications created by compromised pelvic capacity was *lower* prior to agricultural practices than the risk present after the advent of agriculture (Wells et al., 2012). Wells et al. (2012) suggested that maternal growth is more plastic than originally thought and that the appearance of the OD is not universal, but appears to increase with a specific human ecological transition such as the advent of agriculture. Further discussion on archaeological evidence of childbirth difficulty of past populations is outlined in Section 1.9.

The OD cannot be examined without also considering the plasticity of neonate. Human neonate altriciality is another unique element of human life history and the process of childbirth. Dunsworth et al. (2012) have suggested that human altriciality is a consequence of the metabolic draw on the developing infant's mother, prompting human birth to occur at an early developmental stage. This energetic model would also fit with the notion proposed by Wells and colleagues, given ecological shifts affecting food resource availability. Neonatal mass likely increased as a result of shifts from high-protein to high-carbohydrate diets (Wells et al. 2012), as high glycemic diets (such as those high in carbohydrates) are known to promote heavier

birth weight (Scholl et al., 2004; Moses et al., 2006). Wells et al. (2012) suggest that alongside the increase in neonatal size there was a decrease in maternal height due to the decline in health overall with the onset of agriculture, creating a scenario of pelvic and neonatal size mismatch. This ultimately led to an aggravation of OD in the last few thousand years, which would increase the potential for labour complications due to obstetrically inefficient pelvic capacity. This must also be contextualized alongside the potential for plasticity in foetal growth in *parallel* to maternal pelvic size (Wells, 2015). Wells (2017) has also pointed to the influence of the dual burden of malnutrition on changing parameters of the OD in women living today. Women with short stature through stunting are more likely to experience CPD. Obese mothers are also more likely to experience a mismatch between maternal birth canal size and neonatal size, as obese women tend to give birth to larger-than-average (macrosomic) babies (Wells 2017).

It is also important to recognize the evolutionary conflict between infant and mother on the subject of energetics, particularly in relation to pregnancy, childbirth and the OD. Trivers (1974) demonstrated that the theory of natural selection predicts a conflict of interest between mother and offspring amongst vertebrates. In Triver's study the mother reduces her care for her first offspring once her influence on its own survival has reduced, and switches her care to the next offspring in order to maximize maternal inclusive fitness. The offspring's fitness is maximized by obtaining care from its mother until it is detrimental to its siblings (Trivers, 1974). Trivers (1974) suggested that natural selection favours offspring that prompt maternal care until the cost of this care to maternal fitness is double the benefit to the offspring's own inclusive fitness. Blurton Jones (1978) applied this to human birth weight. Mother and infant only share 50% of their genetic information, which prompts mothers to divide her care between multiple offspring. However each human offspring requires care from its mother to maximize its only fitness. Blurton Jones (1978) suggested that offspring fitness is maximized with a high birth weight, whilst for a mother it would be more beneficial to deliver an low birth weight baby in order to apportion greater energy to current and future offspring. Blurton Jones (1978) proposes that birth weight might be a compromise between the neonate's ideal size the maternal investment of energy. Blurton Jones' (1978) application of Trivers' (1974) parent-

offspring conflict theory aligns effectively with Dunsworth et al.'s (2012) and Wells' (2015) points on the presence of and changing levels of maternal and neonatal plasticity in relation to the OD.

Other researchers have proposed developmental models to explain the OD and the variation in maternal pelvic size and shape. Huseynov et al. (2016) collected pelvic dimensions from medical imaging data of 257 people ranging in age from late foetal stages to late adulthood to examine changing obstetric needs throughout a female's lifetime. They found that only moderate sexual dimorphism was present prior to puberty and that with the onset of pubertal changes obstetrically-relevant dimensions of the pelvis expanded from the age of 25 to 30 years (Huseynov et al., 2016). From 40 years of age onwards, females resumed a mode of pelvic development similar to males, causing a reduction in size of obstetric dimensions in the pelvis (Huseynov et al., 2016). Mitteroecker and Fischer (2016) repeated Huseynov et al.'s (2016) study using data from their own 2015 project and were able to reproduce some but not all of Huseynov et al.'s (2016) result, and characterised Huseynov et al.'s (2016) conclusion that the decrease in obstetric dimensions in the pelvis in later adulthood as adaptive as not valid but a Gouldian spandrel.

The evolutionary origins of the OD are also examined through the lens of obstetric medicine. Clinical investigations of the OD investigate dystocia, which occurs as a result of a slow or lack of progression of labour prior to childbirth. Dystocia is often caused by complications with uterine muscular force (the power), cephalo-pelvic disproportion or CPD (the passage) or irregular presentation of the foetus (the passenger) (Thomsen et al., 2014). Women of short stature are at greater risk for CPD (Sheiner et al., 2005; Toh-Adam et al., 2012). CPD often results in obstructed labour. One of the major medical strategies to manage CPD and obstructed labour birth via caesarean section (c-section), which involves an incision through the midline of the uterus and manual surgical assistance to remove the infant from the mother's body. C-section deliveries take place for a multitude of reasons, initiated by both medical staff caring for the mother as well as the mother themselves (Grossman, 2017). The clinical decisions relating to c-section deliveries are beyond the scope of this thesis but c-section deliveries have been used as a measure to

examine the interaction between evolutionary forces and medical intervention in the arena of childbirth.

The WHO recommends 10-15% c-section deliveries within a population (WHO, 2015). C-section deliveries rates above 10% of the population in multiple countries are not associated with decreases in maternal and neonatal mortality (Ye et al., 2016) and mothers delivering via c-section in 24 countries had increased risk of death, blood transfusion and hysterectomy (Souza et al., 2010). Liston (2003) suggests that the increase in c-section rates in different countries was a result of an evolutionary mismatch. Liston (2003) suggested that richer diets and increased population density leading to higher levels of disease with the onset of agriculture reduced maternal height and maternal pelvic capacity. Rosenberg and Trevathan (2018) outline a selection of possible reasons for the increase in c-sections: reduced periods of waiting between labour stages, a lack of continuity-of-care models for childbirth, CPD, shoulder dystocia (an injury-causing mismatch between the maternal birth canal and the shoulders of the neonate), abnormal foetal heart rate, issues with placental implantation and abnormal positioning of the foetus. Zaffarini and Mitteroecker (2019) have more recently found that secular trends in body height can predict global rates of c-section deliveries. Their study hypothesizes that the variation in secular trends in height in different countries caused an intergenerational change of body size, affecting obstructed labour and that with the environmentally induced increase in body height is associated with an increase in CPD and therefore the need for c-section deliveries (Zaffarini and Mitteroecker, 2019). Using data from the WHO, Unicef and the United Nations Zaffarini and Mitteroecker (2019) found that between the years of 1971 and 1996 c-section delivery rate and average body height change had a strong linear positive association. Mitteroecker et al. (2016) also proposed a theoretical model for the high incidence of obstructed labour (as a component of the OD). The cliff-edge model of obstetric selection is on the positive association between the highly variable relationship between neonatal size and maternal birth canal size. This relationship functions until it reaches a critical value beyond which natural birth is no longer possible. As a result of this highly asymmetric cliff-edge fitness distribution, the symmetrical phenotypic distribution of neonatal size and maternal birth canal dimensions cannot effectively match the fitness distribution. The

ideal distribution of neonatal size and maternal birth canal size involves a portion of individuals falling outside of the fitness distribution and experiencing CPD (Mitteroecker et al., 2016). Using the cliff-edge model Mitteroecker et al. (2016) projected an increase in rates of CPD due to the regular use of c-section deliveries acting as a reduction to maternal mortality. Grossman (2017) critiqued Mitteroecker et al.'s (2016) study, outlining that CPD rates have increased due to several reasons according to the obstetrical literature, that the cliff-edge model did not account for variation in maternal soft tissue that could contribute to obstructed labour nor the clinical realities of managing the second stage of labour that could include CPD and that c-section rates have also increased due to the loss of surgical training in forceps delivery.

#### 1.10. Archaeological evidence of childbirth difficulty

It is challenging to examine childbirth in past populations. Firstly, variable preservation of skeletal remains renders clear identification of a pregnant women and an unborn or recently born infant difficult. Secondly, documentation relating to childbirth (such as demographic records, baptism records etc.) may not be accurate or available. Thirdly, whilst childbirth is often a social event amongst humans, there are specific circumstances were women might deliver an infant alone. Examples of this include infanticide amongst hunter-gatherer populations to maintain a low population density (Hayden, 1972) though other scholars have pointed to the need for nuance to better address infanticide in the wide range of hunter-gatherer groups in the past (Denham, 1974; Riches, 1974). However it is essential to consider evidence of childbirth difficulty in the past to address the OD. It should be noted that the skeletal remains of a pregnant woman also constitute evidence of childbirth difficulty from the actual position of the neonate in the maternal birth cavity.

Evidence of childbirth difficulty from Bronze Age Spain is present in the skeleton of a woman with a foetus in transverse presentation in her pelvic cavity, implying that dystocia may have been the cause of death (Malgosa et al., 2004). A further three examples were found in different time periods in Spain, including medieval (Flores and Sánchez, 2007), Roman (Campillo et al., 1998) and Islamic (Seguí et al., 2005). The remains of a pregnant woman from the Phoenician-Punic

necropolis of Monte Sirai dated from 6<sup>th</sup> to 5<sup>th</sup> century BCE Italy also presents another example of possible childbirth difficulty (Piga et al., 2016). Liston and Papadopoulos (2004) outlined their re-examination of the cremated remains of a woman from the Agora region of Early Iron Age Athens buried with a foetus four to eight weeks short of full term and concluded that she died as a result of childbirth difficulties. Lieverse et al. (2015) presented confirmation of dystocia in the remains of a 20 to 25 year old woman from Neolithic Siberia (dated to 7725 to 7630 cal BP) as her skeleton was found with neonatal bones suggestive of twins, with some long bones situated outside of the uterine area and others within the abdominal area. Willis and Oxenham (2013) found the skeleton of a young woman in Neolithic southern Vietnam (dated to 2100-1050 BCE) interred with an unborn full-term breech foetus at the site of An Son. Owsley and Bradtmiller (1983) examined the remains of a selection of females from Arikara communities, prehistoric hunter-gatherer communities spread throughout South Dakota in the United States of America, found evidence of two pregnant women though concluded that childbirth was not their cause of death. Conversely Arriaza et al. (1988) identified maternal cause of death as childbirth in 14% of the total sample of Indian mummies from pre-Columbian Arica in Chile. Alduc-Le Bagousse and Blondiaux (2002) found evidence of coffin birth (postmortem foetal extrusion) in a woman from Calvados in France, whose burial dated to the 4<sup>th</sup> century. Sayer and Dickinson (2013) also detail a case of coffin birth from Early Anglo-Saxon Cambridgeshire, dated to 450 to 700 AD. Hogberg et al. (1987) summarised an example of an either premature or stillborn infant buried with a woman in medieval Sweden, whilst Pérez and Martín (2007) used a multidisciplinary approach to identify coffin birth in a necropolis in Spain dated to the 11<sup>th</sup> to 12<sup>th</sup> century. More recent cases of possible or identifiable archaeological evidence of childbirth difficulties include a mother and child buried in a churchyard in 19<sup>th</sup> century Portugal caused by dystocia (Cruz and Codinha, 2010).

Tague (1994) evaluated the link between age-at-death and pelvic size amongst adult remains from the prehistoric Amerindian Indian Knoll, Pecos Pueblo and Libben populations, dividing the samples in to younger (18 to 24 years old) and older adults (25 years of age and older). Tague (1994) found that the subpubic angle narrowed with age in both sexes and that the linea terminalis was significantly shorter in younger adults. Tague (2004) suggested that females with a short linea terminalis likely had a small pelvic inlet and may have died at a younger age due to childbirth difficulties. Auerbach et al. (2018) examined pelvic dimensions and age-at-death in North American prehistoric early agriculturalist groups ranging from Alaska, Nunavut and New Mexico. They also found that females who died earlier in adulthood have smaller pelvic canals, particularly in the medio-lateral inlet and anteroposterior outlet dimensions, suggesting childbirth was also a cause of death from women in these communities. Pfeiffer et al. (2014) used a similar technique to assess mortality risk associated with childbirth amongst Later Stone Age foragers from South Africa. They found a higher mortality risk for younger women that may have been tied to childbirth, a dietary protein deficiency compared to older women or a combination of both factors. Pfeiffer (2011) also outlined a case study of the remains of a single smallbodied female coastal forager from Later Stone Age South Africa. This woman showed evidence of pelvic asymmetry as a result of narrowing of the right sacral costal process compared to the left an eburnation (Pfeiffer, 2011). Pfeiffer (2011) suggested that this injury may have been caused by habitual activities or that childbirth had exacerbated the instability of the pelvis. It is possible that there was some variation in pelvic dimensions with respect to obstetric sufficiency in Later Stone Age South Africa, as Kurki (2007) found evidence of protection of obstetric capacity in particularly small-bodied populations from this area and time period. McFadden and Oxenham (2019) used linear regression to successfully determine an equation for calculating maternal mortality as a result of childbirth complications in the past that will prove useful for continuing work examining evidence of the OD in past populations.

## 1.11. Fossil evidence of the evolution of human childbirth

It is challenging to examine the evolution of human childbirth due to the scarcity of complete and well-preserved fossilised pelves. There is additional difficulty in identifying the variation in pelvic size and shape as a result of locomotor adaptations with the transition from arboreal to terrestrial lifestyles, compared to pelvic variation as an adaptation to delivering increasingly large-headed neonates. The earliest example of the hominin pelvis appears with the remains of *Ardipithecus ramidus* from Ethiopia at 4.4 million years ago (Lovejoy et al., 2009; White et al.,

2009). Pelvic structure in this species has been described as a 'mosaic', displaying an ischium length closer to that of extant apes and laterally flared iliac blades better positioned to improve gluteal leverage, reflecting a combination of features that enabled both bipedal locomotion and arboreal living (Lovejoy et al. 2009; White et al. 2009). The pelvis of *A. ramidus* displays greater lordosis curvature in the lower spine when compared to the pelves of extant monkeys or apes, though this is unlikely to have affected a deviation from a non-human primate birth process (Parente et al., 2011). Estimates of the relative infant-to-mother mass of *A. ramidus* suggest small infants (DeSilva, 2011) that may also have affected the birth process. For Australopithecus afarensis, a bipedal species dating to 3.2 million years ago, the birth process may have been more similar to that of an anatomically modern human. The birth canal of *A. afarensis* has a curvature more similar to that of anatomically modern humans than to extant apes, suggesting a greater likelihood that infant would have to rotate in the birth canal during birth (Bouhallier et al., 2004). Predictions of neonatal head size in *A. afarensis* indicate similarity to ape rather than anatomically modern human infant head size (Tague and Lovejoy, 1986; Leutenegger, 1987; DeSilva and Lesnik, 2008).

The male and female fossil remains of *Australopithecis sediba*, dated to 1.9 million years, unfortunately have not provided greater detail on changing birth processes. The male pelvis is that of a juvenile and it is unclear whether sexual maturation rates in anatomically modern humans could be used as effective comparisons for *A. sediba* (Kibii et al., 2011), making it difficult to identify the extent of sexual dimorphism between males and females that could affect the birth process. Kibii et al. (2011) found the pelvic inlet in the female pelvis of *A. sediba* to be rounder than that of *A. afarensis* and more similar to the pelvis of *Homo* species. However the relatively small adult brain size in this species suggests that there was not yet a selective pressure for delivering comparatively large-headed infants, including a rotational birth mechanism similar to that seen in modern humans (Kibii et al. 2011).

The mechanisms for delivering an infant for early *Homo* species are complicated by continuing taxonomic uncertainty and a lack of comparative cranial remains that can be used to model possible birth processes (Gruss and Schmidt 2015). Overall the pelves of early *Homo* species exhibit mediolaterally broad anteroposteriorly narrow dimensions that imply a non-rotational birth mechanism (Ruff, 1995, 2010; Rosenberg, 2007; Weaver and Hublin, 2009; Haille-Selassie et al., 2010). Some expansion in the anteroposterior direction of the birth canal did occur and is probably linked to the purported increase in neonatal brain size (Kappleman, 1996; Ruff et al., 1997; Ruff, 2010). A female Homo erectus pelvis (dated to 1.4 to 1.9 million years ago from Gona, Ethiopia) is an exception to the pattern seen amongst other *Homo* pelvic remains. The Gona pelvis is generally broader, with a rounder birth canal and a comparatively wide pelvic outlet (Simpson et al., 2008). Simpson and colleagues (2008) proposed that these particular dimensions (especially considered with a comparatively small body size) are evidence of selection for delivering large-headed infants. The relationship between neonatal head size and maternal birth canal dimensions amongst members of Homo neanderthalensis differs slightly to H. erectus due to the overall increased size of the pelvis, including a wider birth canal (Gruss and Schmitt, 2015). The increase in size did expand the birth canal compared to earlier Homo species yet models of the Neanderthal neonatal size indicate that birth was a similar biomechanical challenge amongst this species as amongst modern humans (Gruss and Schmitt, 2015). The modern human birth process (as indicated by pelvic size and shape) likely arose approximately 100,000 years ago with archaic Homo sapiens.

There are a number of factors that likely contributed to hominin pelvic size and shape and potentially childbirth processes. The relative width of the Australopithecine pelvis has been characterised increasing stride length whilst maintaining energy efficiency per stride length (McHenry, 1982; WL, 1982; McHenry and Coffing, 2000)as well as reducing the energetic cost of walking when carrying an infant (Wall-Scheffler et al 2007; Wall-Scheffler and Myers 2013). Grabowski et al. (2011) has put forward the reduction in integration in the human pelvis when compared to other hominids, suggesting that the appearance of lower integration in pelvic elements in anatomically modern humans enabled a smoother transition to bipedal locomotion. Grabowski and Roseman (2015) further developed this proposal by using genetic models to test whether strong directional selection on specific traits within the pelvis contributed to the dimensions of the modern human pelvis. Their results demonstrated that there were multiple, complex directions for evolutionary forces,

and that many but not all traits hypothesized to play a role in the evolution of the hominin hip and pelvis evolved as a direct result of natural selection (Grabowski and Roseman, 2015).

# 1.12. Birth in non-human primates and mammals and birth difficulty in primates

The process of human birth is not utterly dissimilar to birth in non-human primates and other mammals. Birth varies from a solitary to social event amongst primates (Trevathan, 2015) as a means of reducing infant and maternal risk as well as predation risk. The birth process in humans has been identified as unique amongst mammals as a result of the mismatch in dimensions between the head of a human neonate and the birth canal of a human mother. Washburn (1960) suggested that the human birth canal was narrow when compared to other primates such as gorillas and chimpazees as a result of selection for energetically-efficient bipedality. Increased energetic efficiency as a biped would only be possible with a narrower pelvis overall, reducing the size of the birth canal and rendering childbirth a riskier venture in humans compared to other mammals. Yet human birth is rarely contextualised with birth in other mammals.

Whilst gestation length, labour duration and the biomechanical processes of parturition vary between species, birth behaviour is remarkably similar. The onset of labour in humans is variously brought on by muscular cramping, as well as the loss of a mucus plug from the cervix (a collection of cervical mucus that accumulates throughout pregnancy and is thought to play a role in adaptive immunity) (Becher et al., 2009). The onset of labour in elephants is similarly demarcated with the loss of a cervical mucous plug (Hermes et al., 2008). Women will shift positions prior to delivering an infant, though this will be influenced by their delivery location in and the presence and nature of medical assistance (Desseauve et al., 2017), as will giraffes (Hall-Martin and Skinner, 1978). Many mammals will experience a change or loss in appetite prior to labour, including sheep (Sharafeldin et al., 1971). In humans one of the effects of oxytocin is appetite suppression (Pedersen and Boccia, 2002). Labour stages may be more readily identified in particular species (such as domesticated horses (Frazer et al., 1999) as a result of greater research attention, however the presence of distinct stages is mirrored in human labour stages (Martini et al., 2009).

Most primate and other land-dwelling mammal mothers will engage in placentophagy (consuming the placenta). Possible reasons for this behaviour include maintaining a sanitary area for caring for the newborn, reducing likelihood of scents attracting predators, hunger after labour and acquiring hormones present in the placenta (Kristal et al., 2012). It has been suggested that placentophagy would have reduced with the introduction of fire approximately 2 million years ago (Young et al., 2012). The placenta acts as a filter during pregnancy and accumulates toxicants as the pregnancy continues. Young et al. (2012) suggest that with increased exposure to smoke and ash from fires, placentophagy would have reduced the reproductive fitness of mothers who engaged in this practice. Placentophagy has not been documented amongst hunter-gatherer or agricultural communities (Selander et al., 2013) though increasing interest in placentophagy has been recorded via internet search in the United States of America since 2011 (Farr et al., 2017).

Great apes demonstrate important differences in pregnancy, parturition and pelvic shape and size. These are results of differences in locomotion (bipedality vs. knucklewalking) and reproductive physiology. In humans, the pelvic inlet is wider transversally, whilst the pelvic outlet is much wider anteroposteriorly than it is in apes, which necessitates rotational movement by the human infant during birth (Trevathan 1996; Trevathan and Rosenberg 2000; Tague 2007; Parente et al. 2011). In nonhuman primates, both the pelvic inlet and outlet are wider anteroposteriorly, and the pelvis is lengthened and flattened compared to humans (Parente et al., 2011). The combination of the shape of the nonhuman primate pelvis and the relative size of the neonate allows for a more comfortable fit between the maternal canal and the infant head, which does not create the same need for assistance in the birth process for nonhuman primates as it does in humans (Rosenberg & Trevathan, 2002). Indeed, nonhuman primates primarily assume a squatting position whilst giving birth, which includes the mother assisting the delivery by pulling the infant out of the birth canal (Goodall and Athumani, 1980), and they usually give birth alone (Rosenberg, 1992). However recent observations of three captive birthing female bonobos (Pan paniscus) show birth attendance by other females, including holding the infant and swatting away flies from the maternal birth canal (Demuru et al., 2018). Reports of birth difficulty amongst primates are complicated by a key component of birth behaviour. Non-human primates living in the wild (and some living in captivity) frequently deliver their infants at night (Jolly, 1973) which makes it challenging to report the events occurring during delivery difficult. This lack of data on non-human primate birth makes comparisons with human birth behaviour and birth difficulty problematic (Lindburg, 1982). Some reports of birth difficulty in different species on non-human primate have been published. Nash (1974) reported the delivery of a dead, breech-born, premature infant to a feral baboon (*Papio anubis*) in Gombe Stream National Park in Tanzania, whilst Mitchell and Brandt (1975) suggest that breech presentations amongst macaques usually result in infant mortality. Bowden et al., (1967) reported an infant squirrel monkey stillborn to a female in breech presentation. The scarcity of studies on non-human primate birth behaviour more generally hinders a wider comparison of primate birth.

# 1.13. An evolutionary medicine approach: Examining the origins of the obstetric dilemma in growing girls

Evolutionary medicine is a field that applies the principles of evolutionary biology to health and disease, often with the goal of integrating evolutionary theory with medicine (Grunspan et al., 2018) to posit more effective treatment for disease. At its core, evolutionary medicine harnesses an evolutionary approach to medical issues "to understand why the body is not better designed and why, therefore, diseases exist at all" (Nesse, 2001:358). This approach has been proposed to better address adaptive costs for the host-parasite contest in infection as well as chronic diseases (such as obesity) that have arisen as a result of the comparatively rapid change in human lifestyle since the Stone Age (Williams and Nesse, 1991). It is a burgeoning field that is continuing to define its core tenets (see Grunspan et al., 2018) though provides a promising platform to integrate perspectives from biological anthropology and clinical medicine.

This is especially promising as an approach for examining the origins of the OD as it provides the theoretical and methodological breadth to assess pelvic growth and development in growing girls as a baseline to understand the OD in human evolution. It is not advisable to use data gathered on pelvic growth in living girls as a template for pelvic growth in the past as it cannot be assumed that the ecological

influences on growth are similar in the past and today. Evolutionary medicine provides a basis for using clinical data on the skeletal growth of living children to test assumptions about ecological factors that may act as evolutionary forces. The results from these studies cannot provide an immediately applicable template to use for examining growth in historic populations, however they offer an informed perspective that includes information that is very challenging to match in studies in palaeoanthropology, biological anthropology or bioarchaeology (age of birth, age of death, lifestyle influence, health background etc).

## 1.14. Research aims

Childbirth has been characterised as biomechanically challenging for humans, prompting Krogman (1951) to describe human childbirth as a scar of human evolution. Human mothers give birth to large-headed infants through a comparatively narrow birth canal (see Figure 1.6.). Washburn (1960) suggested that a narrow birth canal in humans arose as an anatomical compromise for the bipedality: narrow hips were necessary to maintain energetic efficiency with bipedal locomotion (the OD). The OD has remained a cornerstone in biological anthropology as a defining feature of anatomically modern humans. However more recent work has questioned the nature of the OD in humans today and in the past, suggesting that there are numerous reasons that childbirth may be biomechanically difficult for mothers and adding complexity to maternal and infant energetic relationships (Dunsworth et al., 2012; Wells et al., 2012a, 2017; Warrener et al., 2015; Wells, 2017). One of the foremost ideas to arise from this rethink is the suggestion that ecological factors in early life (including nutrition and disease) may impact on skeletal growth and development and ultimately influence a woman's pelvic dimensions as an adult (Wells et al. 2012).



Figure 1.6. Relative cranial dimensions in fant primates (filled ovals) are superimposed on pelvic openings (outer oval), with the offspring head in anterior-posterior orientation (upper row) and transverse orientation (lower row). From Wells et al. (2012). Permission to reproduce this figure has been granted by the rights holder, John Wiley and Sons.

Early life influences on the pelvis that could affect childbirth are poorly understood. Previous studies found that pelvic canal breadth increased in girls around the onset of menstruation (known as menarche) (Greulich and Thoms, 1944) and that changes to the female pelvic canal during growth accrued in the midplane and outlet (Coleman, 1969) (see Figure 1.3.). Moerman (1982) and more recently Völgyi et al. (2010) have collected longitudinal data on pelvic growth in living girls. Their results demonstrate that the pelvis has a distinctly different growth trajectory than linear growth, showing that pelvis breadth increases at a slower rate than stature in growing girls and that the pelvic canal continues to grow in breadth once growth in height has ended. As of the time of writing, no studies have examined the influence of fat mass, markers of puberty or biosocial influences on female pelvic growth.

This rethink of the OD provides an ideal opportunity to examine the origins of the OD through examining the growth and the development of the female pelvis. A more nuanced understanding of the growth trajectory of the female pelvis throughout childhood into adulthood will clarify key time points and indicate important influences on pelvic growth. Difficulty with childbirth is not an event relegated to the human past. Childbirth still represents a potentially dangerous event for mothers today. The mismatch between the infant head and maternal birth canal (cephalopelvic disproportion [CPD]) accounts for approximately 9% of maternal mortality globally (WHO, 2014). This underscores the importance of work that approaches the OD from a holistic perspective and seeks to understand the origins of the OD throughout growth and development.

#### 1.15. Detailed research aims

This thesis is structured as a series of papers to facilitate peer-reviewed publication of the results of this project, though is also designed to minimize repetition within independent chapters. The bulk of the literature review for these chapters is contained in Chapter One, with literature specific to each Chapter reviewed therein. Materials for the project as a whole are outlined in Chapter Three, with more specific information contained in each individual chapter. Methods, results reporting and discussion are included in each chapter.

**Chapter Two** of this thesis is a methodological examination of medical imaging technology in collecting pelvic osteometric data from living people, with particular focus on comparing the similarity of collecting skeletal measurements from dual-energy x-ray absorptiometry (DXA) compared to collecting the same measurements from magnetic resonance imaging (MRI). The results of Chapter Two will demonstrate the validity of data collected DXA in investigations osteological variation. Völgyi et al. (2010) have collected pelvic osteometric data from a longitudinal sample of living Finnish girls and women, but their analyses did not include a method-test of DXA measurement similarity to another type of medical imaging technology, such as MRI. Chapter Two predicts that DXA measures of biiliac breadth (BIIB) will be similar to the MRI BIIB measurement due to the ease of identifying the anatomical landmarks for this measurement on both imaging methods. DXA Medio-lateral pelvic inlet (INML) and biacetabular breadth (BIAC) measurements will be less similar than MRI INML and BIAC measurements as DXA image quality will render the landmarks for these measurements more difficult to locate. The results of this chapter guide the methodological considerations for Chapters Three to Five.

**Chapter Three** examines the growth patterns of the pelvis in girls as they grow into women. The purpose of this chapter is to develop centile curves for pelvic breadth and body breadth for girls and women living in the United Kingdom today. These centile charts will be used alongside established growth curves for height, weight, body mass index (BMI), fat mass and lean mass (Cole et al., 1995, 1998; Wells et al., 2012b) of growing girls in the United Kingdom to examine how pelvic breadth develops alongside body size and shape. Growth velocity will also be calculated for pelvic and body breadth measures, identifying the ages at which specific breadth increase takes place. Tracking the growth trajectory and growth velocity of pelvic breadth throughout growth will give context to the manner in which the OD develops in living populations. Chapter Three predicts that shoulder breadth (a measure of body breadth) will increase as stature increases and pelvic breadth will increase after the onset of menarche (Völgyi et al., 2010).

Chapter Four is the first of two chapters examining the factors associated with pelvic breadth throughout growth. This chapter focuses on the biological factors associated with pelvic growth. Skeletal and soft tissue interact throughout growth, particularly puberty, as a function of hormone activity (Prader, 1984; Bogin, 1999b). The goal of this chapter is to test if pelvic breadth can be predicted by birthweight, parental body size and shape and age at menarche. Chapter Four predicts that birthweight and pelvic growth have a statistical association, as previous studies have demonstrated relationships between maternal pelvic size and neonate head circumference (Fischer and Mitteroecker, 2015). This would also follow the trend identified in other studies of neonate head circumference and limb proportions associating with parental anthropometry (Pomeroy et al., 2015a). It is expected that pelvic growth will associate differentially with age at menarche, as Völgyi et al. (2010) demonstrated the difference in growth velocity of the greater compared to the lesser pelvis with the onset of menarche. The results of this chapter will clarify the biological factors that interact with and impact upon pelvic growth during childhood and adolescence, illuminating components of the ecological model that has been proposed for the development of the OD (Wells et al., 2012a; Wells, 2015).

**Chapter Five** surveys the biosocial factors that affect the growth of pelvic breadth. A biosocial approach to analyses of human variation takes social, political

and economic variables into account, recognising that intertwining of biological and cultural actors in human variation (Goodman and Leatherman, 1998; Zuckerman and Armelagos, 2011). This chapter is designed in parallel to Chapter Four. The aim of Chapter Five is to expand on an investigation of biological factors impacting on pelvic growth and recognise the importance of social factors on health and development. This is especially pertinent with regards to the OD, as in living populations biosocial factors such as poor maternal nutrition, early age at marriage, gender inequality and maternal chronic health issues are major contributors to the new OD (Marphatia et al., 2016; Wells, 2017). This chapter uses a biosocial approach to examine the associations between social and economic factors an female pelvic development, including oral contraceptive use, dietary calcium intake, physical activity and socioeconomic status. Chapter Five predicts that socioeconomic status will associate with pelvic growth, as pelvic capacity amongst women is constrained by socioeconomic variables in different populations including access to nutrients (see Kurki 2011 for references). It is also predicted that dietary calcium intake will be associated positively with pelvic breadth, as calcium consumption predicts stature (Wiley, 2005) and increased dairy intake can lead to early age at menarche (Wiley, 2011).

The results from these individual chapters will be summarised in discussion in **Chapter Six** with further contextualization from the clinical and anthropological literature on skeletal growth and pubertal development. Limitations on this work as well as areas for future study based on the results of this project will also be outlined.

## CHAPTER TWO:

### The use of medical imaging in measuring pelvic growth

#### 2.1. The role of Chapter Two in this thesis

This chapter establishes the feasibility of collecting pelvic osteometric data from dual-energy x-ray absorptiometry, which is the principle method of data collection used in this thesis. The dataset used in this chapter differs from the dataset used in Chapters Three, Four and Five. The dataset used in the chapter was chosen specifically because it included two different types of medical imaging of the pelves of the same individuals, which is ideal for comparing medical imaging parameters for pelvic osteometric data collection. The methods explained in this Chapter are used in Chapters Three, Four and Five on a different dataset.

## 2.2. Introduction

The trajectory of human growth is particular in the length of specific periods compared to other animals with an S-shape growth curve. For example, growth velocity in humans is at its highest during prenatal development and preparation for reproduction (puberty) occurs during a period of low growth velocity (Bogin, 1999a). The differences between human and non-human mammal growth trajectories are frequently associated with the specific shape of human life history. Maturation is extended and reproductive age is delayed as part of a need for slow and steady development to better provide energy for increasing cognitive capacities (Mace, 2000). In addition, human growth varies with health status (frequently as a result of socioeconomic conditions), geographic location and population history (Waterlow et al., 1977; Tanner, 1981a; Wadsworth et al., 2002).

Tracking human changes in height and weight throughout growth has been an important component of clinical practice for some time (Marshall and Tanner, 1969, 1970; Tanner, 1981a). The identification of growth faltering throughout childhood is a means of recognizing acute or chronic health issues that could result in further health problems in adulthood. For example, low birthweight in obese adults is associated with cardiovascular disease (Barker et al., 2002). Further clinical research has examined changes in body composition (such as fat mass and lean mass) throughout growth into adulthood. Relative amounts of fat mass and lean mass

change throughout growth, particularly as girls grow into women. Fat mass increases in girls during puberty as a means of preparing the body for lactation (and pregnancy) (Ulijaszek et al., 1998). Lean mass has been found to increase more substantially with age amongst males (Wells et al., 2012b).

Clinical studies of growth make use of a number of different technologies to track stature, weight and body composition changes throughout life. Height is usually measured using a stadiometer whilst weight is measured using an electronic scale. The size of shape of limbs and trunk can be tracked throughout growth by using anthropometrics (Tanner, 1981a), which makes use of spreading and digital calipers and tape measure. Measurement techniques for body composition are dependent on the targeted bodily tissue, though are overall designed to partition body mass or volume in to different components. Body composition is divided into five models with increasing complexity - atomic, molecular, cellular, tissue system and whole body (Heymsfield et al., 1997). Generally, higher complexity tissues are composed of lower order components. For example, adipose tissue is a tissue-system level component but is made of adipocytes at the cellular level, lipids at the molecular level and carbon at the atomic level (Heymsfield et al., 1997). This means that measuring different components of body composition throughout growth calls for different methods of measurement. Molecular components of body composition can be measured using dual-energy x-ray absorptiometry (DXA) scans, tissue system components can be quantified using magnetic resonance imaging (MRI) whilst whole body components of body composition can be measured using anthropometry (Heymsfield et al., 1997).

Patterns of body size and shape throughout growth can reveal trends in growth relating to genetic or environmental influences. Analyses of variation in body size and shape in living populations and the causes of this variation have a strong tradition in biological anthropology (Boaz, 1912; Tanner, 1981a). These analyses have frequently been used to establish a template to understand the evolutionary trajectory of human body size and shape (Ruff, 1991, 1995, 2000, 2002, Holliday, 1997, 2012, Ruff et al., 2005, 2012; Raxter et al., 2006; Wells, 2010; Macintosh et al., 2014a; Will et al., 2017). Examinations of the variation of body size and shape in living populations are also key for quantifying the spectrum of human adaptation to physical and social environments (Bogin et al., 2002, 2017; Bogin and Varela-Silva, 2010; Pomeroy and Stock, 2012; Pomeroy et al., 2012, 2015b; Roseman and Auerbach, 2015).

Tracking growth of populations in the past is possible through analyses of foetal and juvenile remains (Miles and Bulman, 1994; Cowgill, 2010; Cowgill et al., 2012) though this is hampered by the osteological paradox (Wood et al., 1992). It is unclear whether the remains of children who have died in the past are representative of normal growth trajectories, as these children died in infancy. In addition, the distribution of age-at-death of children who have died in the past is not only non-random but also may not allow for longitudinal analyses of growth. Extracting body composition data from skeletal remains further complicates creating a growth trajectory using skeletal remains. Fat mass and body composition can only be minimally interpreted from skeletal dimensions, though Merritt (2014, 2017) has demonstrated that obesity and underweight individuals in life may be under or overestimated in age during skeletal analyses. More recently Pomeroy et al. (2018) found that lean mass could be more reliably estimated from cross-sections of limb bones in living populations than fat mass.

Medical imaging technologies enable visualising soft tissue and skeletal tissue, tracking how these change as children grow into adults and how they are affected by acute or long-term health issues. DXA scans have been used successfully in clinical settings to measure fat mass and lean mass (Fuller et al., 1992; Singhal et al., 2003; Kensara et al., 2005a; Williams et al., 2005; Wells et al., 2012b, 2015), bone mineral density (Mazness et al., 1990; To et al., 2005; Prynne et al., 2006), hip fracture risk due to osteoporosis (Cummings et al., 1993; Faulkner et al., 1993, 2006; Bergot et al., 2002; Kaptoge et al., 2008), changing body size and shape throughout growth of children into adults (Ogle et al., 1995; Bailey et al., 1999; Baxter-Jones et al., 2008; Fewtrell et al., 2009; Völgyi et al., 2010; Wells et al., 2012b, 2015; Wren et al., 2014) and bone strength (Ammann and Rizzoli, 2003; Lloyd et al., 2004; Knapen et al., 2007). MRI has been used for a wide selection of selection of clinical investigations in living people. These include examinations of pelvimetry (particularly in relation to obstetrics) (Hricak et al., 1983; Stark et al., 1985; Spörri et al., 1997; Michel et al., 2002; Rizk et al., 2004; Barnhart et al., 2006; Levine, 2006; Ashton-Miller and Delancey, 2009; Miller et al., 2010; Berger et al., 2013),

neuroimaging (Kwong et al., 1992; Bush et al., 1998; Durston et al., 2001; Jack Jr et al., 2008), the cardiovascular system (Kuller et al., 1998; Ebbers et al., 2001; Bjørnerud and Johansson, 2004; Gatehouse et al., 2005), musculoskeletal components (Gold et al., 2004; Gilles et al., 2006; Cunningham et al., 2007; Khoo et al., 2011), the liver and gastrointestinal system (Paley and Ros, 1997; Bader et al., 2001; Taouli et al., 2007; Marciani, 2011) and angiography (imaging of arteries) (Ruszkowski et al., 1986; Manning et al., 1993).

Scholars working within the scope of biological anthropology have made use of medical imaging to enhance studies of skeletal remains from past populations as well as to track body size and shape change in living populations. Previous work has used DXA scans to examine pelvic size in parous women (Novotny et al., 2000) and the prevalence of conditions such as osteoporosis in past populations (Mays et al., 1998, 2006b; Agarwal and Grynpas, 2009). MRI has been used to examine the developmental trajectory of the human cranial base (Jeffery and Spoor, 2002) and the effects of mummification on the body (Rühli et al., 2007; Rühli, 2015). Use of medical imaging in biological anthropology has made use of DXA and MRI technologies to examine tissues in a similar manner as clinical studies – much like in clinical settings, biological anthropologists have used DXA to examine bone density. Biological anthropologists have rarely made use of medical imaging technologies to examine the skeletons of living people in the same manner they examine skeletal variation in past populations.

Examinations of variation in the bony pelvis allow researchers interested in skeletal variation to better quantify the obstetric dilemma (OD). Washburn (1960) suggested that the tight fit between maternal and neonatal bodies is unique to humans, as the human pelvis is predominantly adapted for energetic efficiency during bipedal walking. Simultaneous to bipedal adaptations, humans have also undergone strong selection for greater brain size, resulting in conflicting pressures rendering childbirth biomechanically challenging. Washburn (1960) termed the result of these conflicting pressures the obstetric dilemma (OD). The OD remained a fixture in characterizing the evolution of modern humans, suggesting that childbirth difficulty is a necessary danger for *Homo sapiens* and that the major issue with childbirth in the past was the biomechanical result of the OD. More recent research has presented a

set of alternative influences on the OD (Wells et al. 2012) including ecological influences. The OD does not equate to the biomechanical reality of the tight fit between the human birth canal and the human neonatal head. The OD should not be used interchangeably with any medical definition of childbirth difficulty, as issues with successfully delivering an infant can be caused by multiple agents. Recent experimental work (Warrener et al., 2015; Warrener, 2017) has demonstrated that energetic efficiency is not compromised in bipedal locomotion with a wider pelvis, suggesting that other factors have contributed to the OD. Wells and colleagues (Wells et al., 2012a; Wells, 2015, 2017) have proposed that early life programming and the ecological context in which women develop may impact on the development of the bony pelvis. Major ecological shifts (such as the transition to agriculture) included increases in disease prevalence and the reliance on a narrow diet, contributing to delays in growth and development that may have affected the pelvis (Wells et al., 2012a). Neonatal responses to such changes may track maternal phenotype, speaking to the importance of ecological variables during early life (Wells, 2015). Amongst living populations, childbirth difficulty arise more markedly as a result of short maternal stature or maternal obesity, where macrosomic offspring are more likely to develop with obese mothers and short-statured mothers are more likely to experience obstructed labour as a result of cepaho-pelvic disproportion (CPD). These noted relationships between pelvic morphology, growth and childbirth difficulties are challenging to test, as there are few studies that have examined the growth of the pelvis throughout life. To date Moerman (1982), Völgyi et al. (2010) and Sharma et al. (2016) have examined pelvic breadth growth in living populations.

The aim of this study is to test the similarity of DXA measurements and MRI measurements of skeletal dimensions, including pelvic breadth. An additional outcome of this investigation is to demonstrate the validity of medical imaging technologies in collecting linear skeletal data from living populations. This study will validate new methods in examining skeletal pelvic dimensions in relation to childbirth.

## 2.3. Materials and methods

#### 2.3.1. Study sample

The sample used for this chapter was selected based on the availability of both magnetic resonance imaging (MRI) and dual energy x-ray absorptiometry (DXA) scans for each individual in the sample, as well as available data on body composition. The sample is a cross-sectional sample of healthy British-born South Asian women (n=70) aged between 20 and 28 years was collected by Dr. Megan Shirley as part of her doctoral research (Shirley et al., 2018). Shirley et al. (2018) sought to examine organ-specific and tissue-specific metabolic rate in South Asian individuals, as evidence suggests that there is population variation in resting energy metabolism and all previous research on this subject focused on European individuals. Shirley et al. (2018) collected data on brain and organ dimensions from MRI and skeletal muscle mass from DXA. This sample used for this study and the data collected from it provided both MRI and DXA imaging of the pelvis in a sample of healthy adult women. All data collection procedures were performed at the University College London (UCL) Great Ormond Street Institute of Child Health (GOSICH) and the Great Ormond Street Hospital for Children NHS Foundation Trust (GOSH) from March 2015 to May 2016. Ethical approval for this study was granted by the Camden and Kings Cross NHS Research Ethics Committee of the Health Research Authority. All participants in the study gave written, informed consent (Shirley et al. 2018).

The sample used in this chapter is not the same sample that is used throughout the rest of this thesis. The sample used in this chapter (as explained above) was used in Dr. Megan Shirley's doctoral thesis (and subsequent publication Shirley et al. (2018)) and was shared with the author of this thesis to complete a methodological test of DXA measurements compared to MRI measurements of the bony pelvis. This methodological test was deemed essential to ensure that all further data collection from DXA scans (which occurs in later chapters of this thesis) was feasible. It should be noted that the only other existing studies using DXA scans to collect osteometric data (Novotny et al., 2000; Völgyi et al., 2010) did not complete a test comparing DXA measurements to MRI measurements.

#### Sample recruitment

Shirley et al. (2018) sought to recruit participants of South Asian (Indian, Pakistani, Bangladeshi and Sri Lankan) ancestry aged 20 to 28 years in London for their study. They sought nulliparous, term-born women with BMI in the range of 17-28 kg/m<sup>2</sup> (Shirley et al., 2018). Ancestry was based on the participants self-identification and confirmed by maternal and paternal grandparents also being Indian, Pakistani, Bangladeshi or Sri Lankan (Shirley et al., 2018). The age range and focus on a single sex were chosen to avoid phenotypic variability associated with sexual dimorphism, pubertal growth and ageing (Shirley et al., 2018). Individuals were excluded if they reported health conditions with the potential to affect growth or metabolism, including smoking. Approximately half of the participants were born in South Asia (33 were born in different countries in South Asia) and half were born in countries other than South Asia (25 were born in the United Kingdom, fiver were born in the United States of America, three were born in Canada, one was born in Australia, one was born in Mexico, one was born in Saudi Arabia and one was born in Kenya) (Shirley, personal communication, 2019).

To recruit the sample, Shirley et al. (2018) used flyers, posters and online advertisements, many displayed in UCL and surrounding universities (eg. the London School of Hygiene and Tropical Medicine, SOAS). Study details via email, such as to staff, students and student societies of GOSICH and UCL (Shirley, personal communication, 2018). The study was also advertised in online student newsletters for the London School of Economics and UCL (Shirley, personal communication, 2018). Two subjects were recruited from Cambridge after a University of Cambridge student shared details of the study with their college (Shirley, personal communication, 2018). Copies of the advertisements used to recruit participants are available in Appendix Four.

## Body shape and size data collection

Height and weight were collected using standard methods. Body volume data was collected using air displacement plethysmography (ADP; BodPod, COSMED, UK). ADP is a method of measuring body composition by determining the volume of air a subject displaces inside an enclosed chamber (Dempster and Aitkens, 1995).

Body volume is indirectly measured by deducting the volume of air remaining inside the chamber when the subject is in the chamber from the volume of air in the chamber when it is empty. Total body water data was collected using deuterium dilution. Deuterium dilution is a method of measuring total body water that includes both intra and extracellular fluid in a subject (Moore, 1947; van Marken Lichtenbelt et al., 1994). Deuterium is a stable isotope of hydrogen and can be given orally to a subject as deuterium oxide. This isotope mixes with body water and is eliminated from the body as urine, saliva, sweat and breastmilk. The enrichment of body water with deuterium can be measured by isotope ratio mass spectrometry. Subjects wore light clothing during data collection and removed all metal jewellery as the jewellery would interfere with MRI function.

### Hard and soft tissue data collection

Bone mineral and soft tissue data was collected using a GE Lunar Prodigy whole-body DXA scanner (GE Medical Systems, UK) and scans were visualised using enCORE 2002 software. DXA is primarily a means of measuring bone mineral density via low-level x-ray beams but is also commonly used to measure total body composition and fat mass (Mazness et al., 1990). Traditional x-ray machines pass xrays of a single energy through the body of a subject. X-ray photons either pass through bodily tissues unaffected or are attenuated (absorbed) by the body. The degree of attenuation depends on the thickness of the tissue. Thicker and denser tissues (such as fat mass) attenuate x-rays more than thinner or less dense tissues (such as fat-free mass). DXA machines use two x-ray beams at different energy levels to identify differences in attenuation of fat mass and fat-free mass (Williams et al., 2005). Studies on the dose of radiation that patients receive when undergoing a DXA scan found that radiation doses are very low in DXA scans, with an effective dose of 0.009 mSv for adults undergoing a scan focusing on the hips (Damilakis et al., 2010). The effective dose was slightly higher for a five-year old child undergoing a scan for the hip area at 0.022 mSv (Damilakis et al., 2010). Effective dose is expressed in sieverts (Sv) and is calculated from information about absorbed doses to the organ or tissue exposed to the X-rays and the relative radiation risk assigned

to each of these organs or tissues. The worldwide average effective dose from natural background radiation is 2.4 mSv per year (Damilakis et al., 2010).

Each subject was scanned using magnetic resonance imaging (MRI; 3T MAGNETOM, Siemens, Germany), focusing on the brain, chest and abdomen of each subject. The following sequence was used for acquiring MRI imaging from each participant: a T1-weighted MPRAGE sequence for brain volume (TR = 2000 ms, TE= 2.74 ms, flip angle =  $8^\circ$ , voxel size =  $1.0 \times 1.0 \times 1.0$  mm isotropic, slices = 240, duration = 5 min); a T2-weighted, turbo spin echo SPACE sequence for the abdomen (TR = 2000 ms, TE= 220 ms, flip angle = variable, voxel size =  $1.5 \times 1.5 \times 1.5$  mm iso- tropic, slices = 144, duration = 7 min); and for the chest, a T2-weighted TrueFISP sequence with breath-hold (TR = 475 ms, TE= 1.53 ms, flip angle = 47°, voxel size =  $1.5 \times 1.5 \times 4.0$  mm, gap = 0, slices = 42, duration = 20 s) (Shirley et al., 2018). The scanner was operated by radiographers. MRI is a medical imaging technique used in radiology based on nuclear magnetic resonance (Lauterbur, 1973). MRI machines align the hydrogen protons (found in water molecules in the human body) using a strong magnetic field, as well as producing a radio frequency current to vary the magnetic field. Hydrogen protons absorb the energy from the magnetic field, altering their orientation. Their original orientation slowly returns once the magnetic field is removed and this process creates a radio signal that is transmitted into an image (Lauterbur, 1973). MRI is preferred in research and clinical settings as a non-invasive method of visualising internal organs and skeletal structures in living subjects (Kwong et al., 1992; Hu et al., 2012) and has been used to visualise the female bony pelvis, reproductive organs and collect pelvimetry data (Hricak et al., 1983; Stark et al., 1985; Spörri et al., 1997; Handa, 2003; Levine, 2006; Miller et al., 2010; Berger et al., 2013).

## 2.3.2. Osteological measurements

Pelvic measurements were collected from the MRI and DXA scans of the same individual from Shirley et al.'s (2018) sample. The author of this thesis collected the osteometric measurements. Pelvic measurements were selected to reflect changing breadths of the pelvis moving from superior to inferior aspects of the bony girdle. The selection of pelvic measurements was also based on the visibility of specific bony landmarks on both MRI and DXA scans. Osteological measurements

are listed and detailed in Table 2.1. and Figures 2.2. to 2.7 show measurements on both MRI and DXA scans. MRI scans were visualised and measurements were collected digitally using OsiriX DICOM software (Rosset et al., 2004). DXA scans were visualised using enCORE 2002 software, and measurements were collected using the Line ROI measurement tool available on the Custom Analysis toolbar (G.E. Healthcare, 2012). A representative of DXA manufacturer, General Electric, confirmed that the Line ROI tool could be used to collect linear measurements from DXA scans that represent real dimensions (Stevens, personal communication, 2016). All measurements were collected four times in total ie, data was collected the first time and was collected again in replications. The first replication took place 24 hours after the initial data collection, with the second replication approximately 4 months after the initial data collection and the third replication took place approximately 5 months after the initial data collection. The author collected the baseline data (the first data collection session) and all three replications. It is conventional to take replications of measurements in anthropometry (Himes, 1989) and this convention was applied to the osteometric data collected in this project.

Osteological measurement	Description for MRI measurement (viewed in coronal plane)	Description for DXA measurement
Bi-iliac breadth	Maximum distance between the right and left iliac blades, defined by the outermost edge of the iliac crest.	Maximum distance between the right and left iliac blades, defined by the outermost edge of the iliac crest.
Bi-acetabular breadth	Distance between most anterior meeting point of fovea capitis of the right and left femora and acetabular notch of the right and left iliac blade, taken at tissue depth that clearly displays the entry point of the ligament of the head of the femur in to the fovea of the femur.	Distance between most anterior meeting point of fovea capitis of the right and left femora and acetabular notch of the right and left iliac blade.
Medio-lateral	Maximum distance between linea	Maximum distance between linea terminalis of

Table 2. 1. Linear measurements of pelvic dimensions and shoulder breadth and their descriptions.



Figure 2.7. Measurements collected from MRI and DXA scans; A) Bi-iliac breadth (BIB); B) Bi-acetabular breadth (BIAC); C) Mediolateral inlet breadth (INML). Image produced by the author of this thesis.



Figure 2.8. BIIB measurement as collected on MRI scans. Image produced by the author of this thesis. Image produced by the author of this thesis.



Figure 2.9. BIAC measurement as collected on MRI scans. Image produced by the author of this thesis



Figure 2.10. INML measurement as collected on MRI scans. Image produced by the author of this thesis.



Figure 2.11. BIIB measurement as collected on DXA scans. Image produced by the author of this thesis



Figure 2.12. BIAC measurement as collected on DXA scans. Image produced by the author of this thesis



Figure 2.13. INML measurement as collected on DXA scans. Image produced by the author of this thesis.

## 2.3.3. Analytical methods

## Data visualization

Bi-iliac breadth, bi-acetabular breadth and medio-lateral pelvic inlet breadth measurements were collected from MRI and DXA scans of the same individuals. A total of three measurement sessions took place: the initial session and twop replicates. The data from the initial measurement session were examined visually as scatterplots, shown in Figures 2.9. to 2.14.

## Measurement precision

Measurement precision is defined as the magnitude of difference between repeated measures using the same technique by the same observer (Wong et al., 2008). In this study, measurement precision was examined between the initial and second replication of both MRI and DXA measurements of pelvic breadth in same individuals. Calculating measurement precision of MRI and DXA pelvic breadth measurements will provide a basis for comparison between both methods. If MRI and DXA data collection methods show similar measurement precision, this suggests both are equally appropriate for collecting osteological measurements of pelvic breadth.

Measurement precision was examined using the technical error of measurement (TEM) which allows anthropometrists to confirm the exactness of repeated measurements performed by one researcher or multiple researchers (Ulijaszek and Kerr, 1999). The calculation of TEM can be broken in to four stages (Perini et al., 2005):

- First stage: calculate the difference between the first and second measurement by subtracting the second measurement from the first measurement for each individual in the sample. The results of this calculation are known as deviations.
- 2. Second stage: all deviations are raised to the second power.
- Third stage: the results of the second stage are summed and entered in to the formula below to obtain the absolute TEM

Absolute TEM = 
$$\sqrt{\frac{\Sigma d^2}{2n}}$$

Where -

 $\Sigma d^2$  = sum of deviations raised to the second power n = number of individuals in sample

4. Fourth stage: this stage transforms the absolute TEM in to relative TEM to obtain the measurement error expressed as a percentage (%). For this stage variable average value (VAV) must be calculated. VAV is the result of adding the first and second measurements of the same individual and dividing this by two, creating the average for that particular individual over two measurements. VAV and absolute TEM are then entered in to the formula below to calculate relative TEM

Relative TEM = 
$$\frac{\text{TEM}}{\text{VAV}} \times 100$$

#### Measurement replication variation

Measuring the variation between replications of data collection indicates the reliability of data collected from DXA compared to MRI scans. In this study the mean difference was calculated between the initial data session and the first replication of measurements collected from scans of the same individuals in order to compare the measurements of the same dimensions. Differences between the baseline (initial measurements) and each replication for each individual were calculated, summed and averaged as absolute values and as percentages (%).

## Measurement variation

Variation in measurement is possible with different measurement techniques, but will also be affected by the biological variation present in the individuals in the sample. In this study measurement variation between MRI and DXA measurement techniques was examined using the coefficient of variation (CV). CVs were reported to examine the extent to which MRI and DXA reliably captured the variation in dimensions in different individuals ie, large values were collected as large values in data collected from both MRI and DXA scans. The CV for both MRI and DXA measurements were calculated by subtracting the DXA values from MRI values. Data were log-transformed and the difference between DXA and MRI log-transformed values calculated. Data were log-transformed to ensure that any skew within the distribution could be reduced. The differences between between DXA and MRI logtransformed values were then multiplied by 100 to transform results into a percentage (%). Measurement variation was calculated for the initial measurement session only.

#### Measurement technique agreement

When a new method of measurement is proposed as an alternative to an established method it is preferable to investigate the agreement of the new method to the previous method. Altman and Bland (1983) found that whilst two methods may have a high correlation they may not have high agreement. Instead Altman and Bland proposed to graphically present measurement data from two different techniques. They created an XY scatter plot that showed the differed between two paired measurements (A-B) on the y-axis and the average of these measures ((A+B)/2) on

the x-axis (Altman and Bland, 1983). This type of graph plotted the difference of the two paired measurements against the mean of the two measurements (Giavarina, 2015). Bland and Altman recommended that 95% of data points should lie within  $\pm$  two standard deviations of the mean difference (see Figure 2.8. for more detail) (Giavarina, 2015). Measurement technique variation was calculated for the initial data collection only and not the replicates.



Figure 2.14. Plot of the differences between method A and method B vs. the mean of two measurements (data is available in Giavarina, 2015). The bias of -27.2 units is represented by the gap between the x-axis, corresponding to zero differences, the parallel line to the x-axis at -27.2 units. From Giavarina (2015). Permission to reproduce this figure has been granted by the Creative Commons Non-Derivs 3.0 Unported (CC-BY-NC-ND 3.0).

The Bland and Altman plots do not confirm if the agreement is suitable between methods nor indeed if one method is preferable to the other. It is best to use the Bland and Altman plots alongside the limits of agreement. The limits of agreement are calculated as 95% confidence intervals (CI). In this study, the differences between the DXA and MRI measurements were entered in a linear regression analysis, with the equation for the regression line for pelvic measurement displayed with the Bland-Altman plot. The limits of agreement (or 95% CI) between the methods were expressed in centimetres and as a percentage. Limits of agreement are calculated as twice the standard deviation of the bias.

# Bivariate analyses

All data were checked for normality using Shapiro-Wilk's test (Shapiro and Wilk, 1965). Linear regression was used to examine statistical association between MRI and DXA collection of pelvic breadth measurements. All statistical analyses and plots were conducted in SPSS (IBM Statistical Package for the Social Sciences, Version 20.0).

# 2.4. Results

# 2.4.1. Study sample

Descriptive statistics of the study sample and measurement variables are available in Table 2.2. Differences in sample size (n) occurred when there was a mismatch between the DXA and MRI scan for the same individual eg. a DXA scan was available but an MRI scan was not available for the same individual.

	Ν	Minimum	Maximum	Mean	Std. Dev
Age (yrs)	70	20.02	28.99	24.01	2.44
Weight (kg)	70	40.65	81.06	57.80	9.29
Height (cm)	70	147.75	177.30	161.24	6.55
DXA BIIB (cm)	66	20.46	28.20	25.15	1.69
DXA BIAC (cm)	66	11.70	16.13	13.63	0.94
DXA INML (cm)	66	10.84	13.78	12.56	0.70
MRI BIIB (cm)	63	19.79	28.14	25.00	1.77
MRI BIAC (cm)	63	12.10	15.47	13.64	0.76
MRI INML (cm)	63	10.64	13.64	12.06	0.70

Table 2.2. Descriptive statistics of study sample and measurement variables.

# 2.4.2. Replications of data

As mentioned previously, data collected was collected and followed by three replicates within different time periods: 24 hours between the baseline data collection
and the first replication, four months between the baseline data collection and the second replication and five months between the baseline data collection and the third replication. Martorell et al. (1975) found that extended intervals between replicate intervals introduce random error to measurements of body dimensions. Given the extended interval between the baseline data collection and the second and third replications, only the data collected from the baseline and second replication (with 24 hours difference) were used to analyse variation between measurement replications. Only the initial baseline data collection was used in the bivariate analyses to reduce error with the extended time period passing between the baseline data collection and further replications mentioned by Martorell et al. (1975).

# 2.4.3. Data visualization

Simple scatter plots are presented in Figures 2.9. to 2.14. of the pelvic breadth measurement data points for MRI and DXA data collection for each individual in the sample. The same pelvic breadth measurements in MRI and DXA are presented alongside one another for ease of comparison.



Figure 2.15. Scatterplot of MRI measurements of BIIB in every individual in the sample.



Figure 2.16. Scatterplot of DXA measurements of BIIB in every individual in the sample.



Figure 2.17. Scatterplot of MRI measurements of BIAC in every individual in the sample.



Figure 2.18. Scatterplot of DXA measurements of BIAC breadth in every individual in the sample.



Figure 2.19. Scatterplot of MRI measurements of INML in every individual in the sample.



Figure 2.20. Scatterplot of DXA measurements of INML in every individual in the sample.

The pattern of data spread is broadly similar for BIIB in both MRI and DXA measurement methods. DXA measured BIAC is overall smaller than the same measurement in MRI. The pattern of data spread looks least similar between MRI and DXA measurements of INML. These results suggest that whilst there is a general pattern that follows between the data collection methods, there are some differences that can be seen from visual inspection of the spread of MRI and DXA-collected pelvic breadth measurements.

## 2.4.4. Measurement precision

The results of the TEM calculation are presented in Table 2.3. The highest relative TEM resulted from DXA BIAC at 4.31% whilst the lowest relative TEM stemmed from MRI BIIB at 0.82%. Perini et al. (2005) suggest that the highest relative TEM that would be acceptable for an expert anthropometrist taking skinfold measurements is 5.0% and taking other measurements is 1.0%. Using this metric MRI BIIB AND DXA BIIB would be considered appropriate to use in clinical research. It should be noted that these relative TEM values are comparable to recent work on

error comparing body composition measurement methods, including DXA (Williams et al., 2005).

	Ν	Absolute TEM	Relative TEM (%)
MRI BIIB	63	0.19	0.82
DXA BIIB	66	0.43	0.87
<b>MRI BIAC</b>	63	0.23	1.91
DXA BIAC	66	0.55	4.31
MRI INML	63	0.29	2.69
DXA INML	66	0.19	1.64

Table 2.3. Results of the TEM calculation.

## 2.4.5. Measurement replication variation

The mean differences from MRI and DXA scans in all replication measurement sessions are displayed in Table 2.4. DXA BIAC and INML initial and first replication measurements underestimated by 0.10cm and 0.37cm respectively, whilst MRI measurements only underestimated BIIB by 0.11cm between the initial and first replication of pelvic breadth data collection. The largest difference between the initial and first replication being 2.95% smaller than the initial data collection. The smallest difference between measurement sessions was in MRI BIIB measurements at 0.28% increase between measurement sessions. The -2.95% difference between initial data collection and first replication DXA measurement sessions is higher than Völgyi et al., (2010)'s largest interobserver error difference, however it is comparable to recent work on error comparing body composition measurement methods (Williams et al., 2005).

Table 2.4. Mean differences between initial and first replication sessions of DXA and MRI scans for each measurement variable.

Measure ment	N (MRI)	N (DXA)	Mean difference between DXA measurement baseline and first replication (cm)	Mean difference between MRI measurement baseline and first replication (cm)	Mean difference between DXA measurement baseline and first replication as %	Mean difference between MRI measurement baseline and first replication as %
BIIB	63	63	0.07	-0.11	0.28	-0.45
BIAC	63	63	-0.10	0.07	-0.76	0.53
INML	63	63	-0.37	0.04	-2.95	0.38

# 2.4.6. Measurement variation

Coefficients of variation of DXA and MRI osteometric data collection and the bias of DXA compared to MRI osteometric data are expressed in percentages in Table 2.5.

Table 2.5. Coefficient of variation, average difference between MRI and DXA pelvic breadth measurements and bias of DXA measurements compared to MRI measurements.

	N (MRI)	N (DXA)	Coefficient of variation MRI (%)	Coefficient of variation DXA (%)	Avg. difference between MRI AND DXA measurements (cm)	Bias of DXA compared to MRI (%)
BIIB	63	63	7	6.85	-0.13	0.07
BIAC	63	63	5.66	5.5	0.07	0.7
INML	63	63	5.95	5.53	-0.51	-1.9

Coefficients of variation for BIIB are almost identical, whilst they are slightly higher in MRI BIAC and INML measurements. DXA INML measurements have the largest magnitude of difference to MRI INML measurements and under-estimate MRI measurements by 0.51cm. DXA BIIB are on average 0.13cm smaller than MRI BIIB measurements, whilst DXA BIAC measurements are 0.07cm larger than MRI BIAC measurements. DXA INML measurements have the greatest bias at 1.9% smaller than MRI INML measurements are 0.07% larger than MRI BIIB measurements and have the lowest bias of the pelvic breadth measurements

collected. These values are comparable with Williams et al., (2005)'s results when comparing body composition assessment using the criterion 4-component model and the Lunar Prodigy DXA.

## 2.4.7. Measurement technique agreement

Table 2.6 displays the results of a Bland-Altman analysis. This plots the difference between MRI and DXA measurements (y-axis) and the mean bias of these methods (x-axis) using the pelvic measurement data collected at the initial measurement session (that is, not including any replicates). These variables were entered in a linear regression analysis, with the equation for the regression line for pelvic measurement displayed with the Bland-Altman plot (see Figure 2.15 to 2.17). P-values of the linear regression are also displayed in Table 2.6. The limits of agreement (CI intervals) between the methods are expressed in centimetres and as a percentage. Limits of agreement are calculated as twice the standard deviation of the bias. There is no significant relationship between the difference of MRI and DXA measurements and mean bias of these methods.

		Mean			Limits of ag	reement	R <sup>4</sup>	<b>P</b> *
	N (MRI)	N (DXA)	(cm)	(%)	(cm)	(%)		
BIIB	63	63	-0.13	0.07	± 0.66	±4.11	0.032	0.159
BIAC	63	63	0.07	0.7	± 1.24	±3.59	0.052	0.071
INML	63	63	-0.51	-1.9	±1.12	±2.19	0.002	0.729

Table 2.6.	Results of	f Bland-Altm	an analysis	, including	man bias,	limits	of agree	ment
and linear	regressio	n results. * V	alues are s	ignificant a	ıt <0.05.			
	0			<b>~</b> I			-2	-



Figure 2.21. Bland-Altman plot of MRI compared to DXA BIIB showing mean bias, two standard deviations above and below the mean and the formula for the regression line.



Figure 2.22. Bland-Altman plot of MRI compared to DXA BIAC showing mean bias, two standard deviations above and below the mean and the formula for the regression line.



Figure 2.23. Bland-Altman plot of MRI compared to DXA INML showing mean bias, two standard deviations above and below the mean and the formula for the regression line.

The Bland Altman plot for the BIIB MRI AND DXA data shows a positive relationship with two outliers above and below the 2 standard deviations above and below the mean. The mean bias for the MRI compared to DXA BIIB measurements is not very large and tends to underestimate BIIB size. The Bland Altman plot for BIAC shows close patterning of data points around the mean of 12cm BIAC and a negative relationship. Three data points are below one standard deviation from the mean and one data point is above the CI. The plot for INML shows a positive relationship between the average of MRI and DXA INML measurements, and the mean bias in the plot is the largest of the three pelvic breadth measurements. There is no clear pattern in the spread of the data points, with only two data points below the lower CI. These results suggest the MRI and DXA methods of measuring pelvic breadth generally agree.

## 2.4.8. Bivariate analyses

As outlined in Section 2.3.2, only baseline data collection results were used for bivariate analyses in order to reduce error that occurs with the extended intervals of time between replications as per Martorell et al. (1975) . Results of the Shapiro-Wilk's test for normality of baseline data only are presented in 2.7. Shapiro-Wilk's test for normality is satisfied for all variables. Results of the linear regression of DXA measurements on MRI measurements of pelvic breadth are available in Table 2.8. Regression plots for BIIB, BIAC and INML are presented with the relevant formulae for the regression lines in Figures 2.18 to 2.20. DXA measurements of bi-iliac breadth can be predicted using the formula 0.949 MRI BIIB+1.407,  $R^2$  0.96. Bi-acetabular breadth can be predicted using the formula 0.857 MRI BIAC+1.872,  $R^2$  0.53. DXA measurements of medio-lateral inlet breadth can be calculated using the formula 0.65 MRI INML+4.695,  $R^2$  0.45. DXA measurements of bi-iliac breadth were the strongest predictor of MRI bi-iliac breadth measurements, followed by bi-acetabular breadth and medio-lateral inlet breadth.

	Shapiro-Wilk statistic	df	p-values			
DXA BIIB	0.979	63	0.356			
DXA BIAC	0.981	63	0.430			
DXA INML	0.973	63	0.173			
MRI BIIB	0.977	63	0.297			
MRI BIAC	0.988	63	0.823			
MRI INML	09.83	63	0.521			
* Values are significant at <0.05						

Table 2. 7. Results of the Shapiro-Wilk's test for normaity of the baseline DXA and MRI pelvic dimension measurements.

Table 2. 8. Results of the linear regression of the first replication of DXA pelvic dimension measurements regressed on MRI pelvic dimension measurements.

	Ν	Std. Error	Beta	t	p- values*	95% Cl Lower Bound	95% C I Upper Bound
DXA BIIB	63	0.023	0.982	40.861	0.000	0.903	0.996
DXA BIAC	63	0.103	0.730	8.347	0.000	0.652	1.062
DXA INML	63	0.091	0.675	7.143	0.000	0.470	0.836



Figure 2.24. Linear regression plot of DXA BIIB regressed on MRI BIIB.



Figure 2.25. Linear regression plot of DXA BIAC regressed on MRI BIAC.



Figure 2.26. Linear regression plot of DXA INML regressed on MRI INML.

# 2.5. Discussion

This chapter address the applicability of pelvic breadth data collection from medical imaging of the skeletons of living women, with a particular focus on the applicability of measurements collected from DXA scans. Identical measurements of pelvic breadth were collected from MRI and DXA scans of a sample of living women and the similarity of DXA measurements were compared to MRI measurements. Tests of measurement precision demonstrated that overall MRI and DXA measurements of pelvic breadth had comparable levels of precision, with the exception of DXA BIAC which was closer to 5% measurement error. Overall, DXA BIIB measurements were closest in measurement agreement across imaging methods, whilst INML and BIAC measurements have variously lower measurement agreement in MRI and DXA imaging methods. However Bland-Altman analyses showed that there were no significant mean biases in any of the DXA measurements.

that even with differences in precision, collecting skeletal dimensions DXA was comparable to the variation present in the same data collected from MRI scans.

One of the primary challenges of collecting pelvic breadth data from MRI and DXA scans is the nature of the image of the pelvis produced by these methods. This is primarily a result of the differing visual perspectives in which the pelvis is presented. The bony pelvis is a three-dimensional structure, however in this study the imaging methods used only presented in two-dimensions in MRI and the DXA scans, limiting the types of measurements that can be collected from both scans. MRI scans are built from multiple image slices moving from anterior to the posterior aspect of the body. In this study these image slices could not be collated to create threedimensional models of the pelvis, meaning that linear measures of pelvic depth (eg. depth from the posterior aspect of the pubis to the anterior aspect of the coccyx) could not be collected. DXA scans produce a single image of the pelvis from an anterior perspective, meaning that pelvic depth measurements also cannot be collected from this type of imaging method. It should be noted that Novotny et al., (2000) did collect a pelvic depth measurement from DXA scans (vertical distance between the pubic symphysis and the promontory of the sacrum). This may be accurate when collecting osteometrics from a Hologic DXA scanner (as Novotny and colleagues did), however given variation in buttock adipose tissue in participants that could tilt the pelvic girdle pelvic depth measurements were not collected in this study.

Pelvic *breadth* measurements can be collected from both MRI and DXA scans. The manner in which breadth measurements are collected differ in MRI scans compared to DXA scans. Pelvic breadth measurements collected from MRI scans are collected at slightly different points of scan depth and the researcher must identify the depth that contains the anatomical landmark of interest. Pelvic breadth measurements collected from DXA scans are collected from a static image, with the researcher simply maximizing the image to identify the anatomical landmark for a specific measurement. Whilst these images give a detailed image of the pelvis, there is a marked difference in identifying bony anatomical landmarks digitally compared to pinpointing them on dry bone. The importance of the tactile element of osteology has been discussed in relation to pedagogic techniques (Siek, 2015). Whilst this study suggests the plausible use of ostemetric data collected from medical imaging, it should be noted that these methods are distinctly different from tactile osteometric data collection from dry bone.

The differences in visual perspectives for pelvic breadth measurement collection did not affect the agreement of one measurement out of three pelvic breadth measurements. The BIIB measurement was taken at the widest point of the iliac blades, which was easily identified in both MRI and DXA and proved to be the closest match with MRI measurements (R<sup>2</sup> 0.96). Anatomical landmarks for the BIAC and INML measurements proved more challenging to locate in both MRI and DXA scans which may explain the difference in measurement agreement between these measurements and BIIB. It is likely that issues of locating the anatomical landmark necessary for the measurement notably affected the INML measurement. The midpoint of the arcuate line was sometimes difficult to isolate on the DXA scans due to poor image guality once the DXA scan image was maximised. Both INML and BIAC measurements dictated different visualisations of anatomical landmarks on MRI compared to DXA (see Table 2.1.). These differences in landmark selection were necessary for the differentiation in imaging method though likely contributed to significant differences between measurement sessions and similarity levels. The position of the participant during initial scanning may also affect ability to visualise anatomical landmarks. Lambrinoudaki et al. (1998) have reported significant differences in DXA calculation of body mass from scans of patients lying in supine vs. prone positions on the examination table. However participants in this study were all in supine position for MRI and DXA scanning procedures.

The applicability of the results of this study is limited by sample characteristics and measurement procedure. The sample used for this study is homogenous in cultural background and tightly clustered in age – this allows for clear interpretation of results but may not be directly applicable to other living populations. Whilst there is a range of body size and shape within the sample, mean body mass for this sample was 57.8kg and mean stature was 161.24cm. Average female mean body mass in the United Kingdom in 2010 was 70.2kg and mean stature was 161.1cm (Office for National Statistics, 2010). The body mass of the women in this sample is markedly lower than the national average, suggesting that the results from this study may not be applicable to populations with a larger mean body size and shape. The sample used in this study is made up of South Asian women. On average South Asian women living in the UK tend to have lower bone mass (Roy et al., 2007) which may affect the clarity of placing measurement points in DXA scans. Measurement procedure could be improved by including an inter-observer error study to better quantify error in measurement procedure between researchers, including making use of the extra replications used in this study to examine error rate with time intervals between replications. This would be especially instructive for osteological researchers making use of these types of imaging methods for the first time.

## 2.6. Conclusion

Results indicated that there was no significant measurement bias for BIIB, INML and BIAC dimensions (p < 0.05 BIIB 0.159, BIAC 0.071, INML 0.729) collected from DXA scans compared to these dimensions collected from MRI scans. DXA BIIB measurements most closely matched MRI BIIB (R<sup>2</sup> 0.96) and this pelvic dimension also had the lowest percentage of bias (0.07%) between DXA and MRI imaging methods. DXA INML (R<sup>2</sup>0.45) and BIAC (R<sup>2</sup>0.53) dimensions did not match those collected from MRI scans as closely as BIIB. This is possibly a product of differences in image quality between imaging methods that in turn affects ability to locate specific anatomical landmarks for collecting dimensions. DXA INML often under-estimated MRI INML and had the highest percentage of bias (-1.9%) compared to MRI INML. All pelvic breadth measures collected from MRI and DXA in this study had comparable coefficients of variation (MRI BIIB 7%, DXA BIIB 6.85, MRI BIAC 5.66%, DXA BIAC 5.5%, MRI INML 5.95%, DXA INML 5.53%). This indicates that data collected from DXA scans will represent biological variation to the same degree as data collected from MRI scans. Pelvic osteometric data can be collected from DXA scans in living people to create a template for understanding pelvic growth. These results can better contextualise ecological factors in the development of the OD.

# 2.7. Collecting pelvic osteometric data from DXA in the following chapters of this thesis

The methods that have been used in this chapter demonstrate the feasibility of collecting pelvic osteometric data from DXA scans when compared to MRI scans in healthy, adult British women of South Asian ancestry. In the wider context of this thesis the results from this chapter show that pelvic osteometric data can be collected from DXA scans. This provides the basis for collecting these measurements from different age groups to examine the growth trajectory of the pelvis in order to identify possible periods of greater or lesser susceptibility to size change of the pelvic canal. Identifying the time period at which the pelvic canal may be more or less susceptible to canal size change may indicate if canal breadth (and therefore possible childbirth difficulty in adulthood) alters with ecological change.

## CHAPTER THREE:

#### The growth patterns of the female pelvis

#### 3.1. The role of Chapter Three in this thesis

Chapter Three is the first chapter of this thesis to feature the dataset used in the subsequent chapters as well as the major research themes of this thesis. This chapter uses a different dataset to Chapter Two, as Chapter Two outlined the methodological foundation for the method used to collect pelvic osteometric data from medical imaging methods, specifically dual-energy x-ray absorptiometry. This chapter uses a dataset of growing children, which provides the basis to answer the research question: what is the growth trajectory of the female bony pelvis?

#### 3.2. Introduction

Tracking growth and development is a key element in clarifying variation in human body size and shape in the past. Quantifying growth in different populations aids in identifying the components of growth that are genetically controlled, those that are affected by environmental variation and how these two factors may additionally vary as a result of insults on growth and development (Hamill et al., 1979). Paediatric medicine has a strong tradition of using growth charts of height and weight to track growth throughout childhood to adulthood (Tanner et al., 1966; Tanner and Whitehouse, 1976; Tanner, 1981a). More recent work has also identified body mass index parameters for growth standards in Singapore, Brazil, the UK, Hong Kong, the Netherlands and the USA (Cole et al., 2007), fat mass indices in children the USA (Kelly et al., 2009) and tracked fat mass (fat mass that remains on or between zscore lines and roughly parallel to the median) and lean mass in children in the UK (Wells et al., 2012b). Other fields of research have an interest in tracking growth as means of understanding human variation. One of the aims of biological anthropology is to understand the effects of changing social and biological environments on the human body, including major demographic shifts such as the advent of agriculture (Larsen, 1995, 2006). Such major demographic shifts resulted in dietary changes and alterations to disease spread (Larsen, 1994; Steckel et al., 2002; Waldron, 2009) as

well as regional specialisations dependent on mobility and physical activity (Stock, 2006; Stock et al., 2011; Shaw and Stock, 2013; Cameron and Pfeiffer, 2014; Macintosh et al., 2014b, 2015). Frequently these examinations of human variation focus on skeletally adult individuals due to the poor preservation of subadult skeletal remains (Bello et al., 2006) and the non-random distribution of age ranges of skeletal remains (Wood et al., 1992). Growth charts have been constructed for foetal pelvic remains from a sample from 16<sup>th</sup> to 19<sup>th</sup> century Scotland (Miles and Bulman, 1994) and skeletal growth profiles for Anglo-Saxon children aged 6 months to 18 years (Hoppa, 1992). Few studies have made use of growth charts to track changes in breadth or linear dimensions of skeletons from childhood to adulthood.

Growth primarily involves change in size and shape of body parts, but the rate of change for body parts and the growth patterns of bodily tissues is not identical. Humans grow in a cephalocaudal pattern, where they increase in length from their head to their lower limbs (Bogin and Varela-Silva, 2010). In utero foetuses grow in length during the second trimester of pregnancy and gain weight in the third trimester of pregnancy. All humans grow in a specific order of growth phases: the neonatal period, infancy, childhood, puberty, adolescence and adulthood (Bogin, 1999b). For members of Homo sapiens, priority is given to increasing brain size in early life to better understand and participate in complex human sociality (Bogin, 1999b). Prioritising growth of the brain results in growth 'trade offs' for other parts of the body dependent on adequate nutrition and disease load throughout growth (Bogin and Varela-Silva, 2006). For example, Peruvian children raised in the highlands (who were exposed to higher levels of stress) had shorter limbs overall and shorter radius and ulna segments than children raised in the lowlands (Pomeroy et al., 2012). Growth faltering is a result of many different factors, both biological and social. Low birth weight has been identified as a predictor of poor health throughout life and a potential growth failure (Barker et al., 1993; Hack et al., 2003). Growth failure with respect to body size can be caused by malnutrition (Martorell and Zongrone, 2012), diseases affecting a child's ability to digest food such as inflammatory bowel disease (Motil et al., 1993) and by respiration issues (Balfour-Lynn, 1986). Gohlke et al. (1998) found that children experiencing psychosocial stress were shorter in stature compared to their peers, an outcome of abnormal eating patterns, behavioural issues

89

and different forms of abuse. It is challenging to identify possible deviations in growth patterns without an established outline of growth for children in different populations.

Studies of human growth have focused on size and shape change in different parts of the body. Height and weight are the most common targets for growth trajectories (Tanner et al., 1966; Tanner and Whitehouse, 1976; Cole, 1989; Reubinoff et al., 1995; Cole et al., 1998; Mamidi et al., 2011) though other scholars have examined body mass index (Lindgren et al., 1995; Cole et al., 1998), leg length (Dangour et al., 2002; Bogin and Varela-Silva, 2008, 2010) and arm length (Hertel et al., 1995). These data are straightforward to collect and are reproducible throughout anthropometric methodologies. However few studies have examined body breadth change as children grow. Body breadth is not to be confused with measures of adiposity, such as waist circumference or skinfold measures. Instead, body breadth focuses on the width of the body as an outcome of skeletal architecture, such as breadth of the bi-iliac blades of the pelvis. Biological anthropologists have examined variation in body breadth as a consequence of thermoregulatory adaptations (Ruff, 1994; Ruff et al., 2005) and ecogeographic patterns (Roseman and Auerbach, 2015). Henneberg and Ulijaszek (2010) found that amongst living Australians skeletal dimensions account for over 50% of variation in skinfold thickness and that larger trunk dimensions associated with greater fatness. Enguiries into variation in female body breadth have focused on reproductive adaptations, including attractiveness (Singh and Young, 1995; Tovée and Cornelissen, 2001) and interplay between efficient bipedality and the ability to safely deliver a neonate (Wall-Scheffler, 2012; Warrener, 2012; Wall-Scheffler and Myers, 2013). Recent rethinking of the obstetric dilemma (OD) (Wells et al., 2012a; Wells, 2015; Ruff, 2017) has demonstrated that a more complex investigation into the multi-factorial origins of childbirth difficulties and variation in pelvic size and shape is necessary. This is additionally pertinent to living populations where childbirth difficulties are more likely to be the result of small maternal stature, chronic health issues and increasingly maternal obesity (Wells, 2017).

The purpose of this study is to create growth centiles for body breadth and pelvic dimensions in living girls and women to compare the development of these measures of breadth and determine if there are key growth periods for pelvic dimensions in particular. Using the methodologies established in Chapter Two, this study integrates data collected from DXA scans with techniques used in nutrition and clinical paediatric practice to outline the growth trajectories of pelvic dimensions and body breadth in girls as they grow into women.

#### 3.3. Materials & Methods

#### 3.3.1. Sample used in this chapter

The sample that is used in this chapter is a different sample to that used in Chapter Two. The sample used for analyses in Chapter Two was used as it included both MRI and DXA scans of the same individuals, providing an opportunity to examine the reliability of DXA imaging methods of the pelvis in living people. Having demonstrated that pelvic dimensions can be reliably collected from DXA scans in Chapter Two, the sample in this chapter was chosen to address the pattern of pelvic growth and includes growing girls. The data from both samples were collected at the same location, though were collected for different projects. The sample from Chapter Two was used as part of Dr. Megan Shirley's doctoral thesis (completed in 2017) and subsequent publication (Shirley et al. 2018). Mary Fewtrell and colleagues used the sample examined in this chapter in a study begun in 2010 and completed in 2016. Further details on the sample used in this chapter are available in Section 3.3.2.

#### 3.3.2. Study sample

The sample for this study (f=286) was collected as a reference sample at the Great Ormond Street Hospital in London, UK between the years 2001 and 2016. It includes data from other smaller studies (see Table 3.1. for details). The other smaller studies include the Betapole study, the Cambridge Growth study and the twin growth study and the Ross growth study. The Cambridge Growth study began in 1995 and included healthy children between the ages of four weeks and two years of age (Freeman et al., 1995) that were recruited for a comprehensive study on energy metabolism (Wells et al., 1997). These children were recruited from the Rosie Maternal Hospital in Cambridge and no reward was offered at any point for participation in the study (Wells et al., 1997). The Betapole and Ross growth study

were smaller samples recruited to examine body composition in healthy, term-born children (Chomtho et al., 2008a; b). Children in these studies were singleton births and recruited from greater London and Cambridgeshire (Chomtho et al., 2008a; b). The twin growth study was initiated by Tim Cole in the 1990s and data collected from this study was unpublished and then included in Mary Fewtrell and colleagues wider sample that is being used in this chapter (Wells, personal communication, 2019). The sample used in this chapter was collected as part of the project entitled 'Collection of bone mineralisation reference data in normal healthy children'. Mary Fewtrell and colleagues at the Institute of Child Health, University College London, initiated the project. The aim of Fewtrell et al.'s study was to collect data on bone mineralisation and body composition on healthy British children to use as a reference for other projects focusing on British children suffering from different diseases. Subjects in the study were recruited through advertisements in newspapers and schools throughout the London area and the southeast of England (Devakumar et al., 2016). The sample used in this chapter (including all of the smaller studies and the data collected from the sample for Fewtrell and colleagues study) includes both cross-sectional and longitudinal data. Cross-sectional data are available for 232 girls and women and longitudinal data are available for 52 girls and women in the sample. Ethical approval for Fewtrell et al.'s project was granted in 2000 by the Ethical Committee of University of College London Institute of Child Health at Great Ormond Street Hospital and associated parameters of this approval are included in Appendix One.

Children in the sample used in this chapter are aged between 4 and 23 years, including young adults in order to cover the full pediatric age range (Devakumar et al., 2016). The lower age limit of 4 years old was chosen as children below this age are unlikely to be eligible for air displacement plethysmography (ADP) (Wells et al., 2012b). There was no exclusion criteria for body mass index (BMI), meaning that some subjects were categorised as overweight or obese though did not suffer from a disease that could negatively impact their growth and development (Wells et al., 2012b; Devakumar et al., 2016). Whilst the majority of the sample are white, out of the 286 females in the sample 25 participants self-identified as ethnicities other than white. Six participants self-identified as Chinese, 10 self-identified as black, three

self-identified as Asian, two self-identified as mixed ethnicity (black and white), one self-identified as mixed ethnicity (white-Malay), two self-identified as mixed ethnicity (white/Chinese) and one participant self-identified as Phillipino. Ethical approval for this present project detailed in this chapter was granted in 2016 by the University of Cambridge School of Humanities and Social Sciences. Confirmation of this ethical approval is included in the Appendix One.

Table 3.1. Sample details of other smaller studies included in Fewtrell and colleagues reference sample, as well as sample collected specifically for Fewtrell and colleagues reference sample.

Study name	Number of females per study
Betapole study	15
Cambridge Growth Study	34
Growth twin study	10
Ross Growth Study	38
Fewtrell et al. reference	135
sample cross-sectional	
dataset	
Fewtrell et al. reference	54
sample longitudinal dataset	
TOTAL	286

# Great Ormond Street Hospital reference sample data collection (Fewtrell and colleagues reference sample)

All subjects attended a 2 hour measurement session at the body composition suite at the Great Ormond Street Hospital in London. One operator carried out 60% of the measurements included in the study and five other operators carried out the remaining measurements (Devakumar et al., 2016). A selection of subjects (n=54) participated in longitudinal studies and returned for a measurement session within 2.5 years (Wells et al., 2010).

# Body shape and size data collection

Weight and height were measured using standard protocols. Height was measured using a wall-mounted stadiometer and body weight was measured as part of the ADP procedure. BMI (in kg/m<sup>2</sup>) was calculated as weight divided by the square of height. Anthropometrics were collected using either a nonstretchable fibreglass tape or Tanita BC418MA instrumentation. Circumference anthropometry (head,

waist, hip, thigh and calf circumference) was collected using tape. Skinfold-thickness measurements at the biceps, triceps, subscapular and suprailiac sites were collected using Tanita instrumentation in triplicate, with the mean of these values recorded as the final measurement (Wells et al., 2012b). Current height and weight of the parents of each subject were recorded from recall. Frequently the parent attending the measurement session (usually the mother) was asked to recall the height and weight of the other parent (Devakumar et al., 2016).

#### Body composition data collection

Fat mass and lean mass were calculated using the 4-component (4C) model which is considered the most accurate approach for living subjects (Wells et al., 2015). More information on the 4C model is available in Chapter Two. Full details of 4C calculations can be found in Wells et al., (2012). In this sample, body volume data was collected using air displacement plethysmography (ADP) (BodPod, COSMED UK). Full details on ADP are available in Chapter Two. Bone mineral content was collected using dual-energy X-ray absorptiometry (DXA; GE Medical Systems, UK) and total body water data was collected using deuterium dilution (Devakumar et al., 2016). Full details on both DXA and deuterium dilution are available in Chapter Two.

## Sample details for this chapter

The sample used for all of the analyses in this chapter comprises 232 girls and women from the Great Ormond Street Hospital reference sample. This is a crosssectional data set where each subject attended one measurement session.

### 3.3.2. Osteological measurements

Linear measurements of the skeleton were collected from DXA scans of the study sample. Osteological measurements were selected to capture variation in body and pelvic dimensions and are detailed in Table 3.2. Images of the measurements collected from DXA scans are available in Chapter 2. DXA scans were visualised using enCORE 2002 software, and measurements were collected using the Line ROI measurement tool available on the Custom Analysis toolbar (G.E. Healthcare, 2012). Medio-lateral pelvic inlet breadth measurements were taken with maximum zoom capacity for each scan.

Table 3.2. Linear measurements of pelvic dimensions, shoulder breadth and their descriptions.

Osteological measurement	Description
Shoulder breadth (Völgyi et al., 2010)	Distance between the outset point of left and
	nghi heads of the humerus.
Bi-iliac breadth (Kurki and Decrausaz, 2016)	Maximum distance across the right and left
	iliac blades.
Bi-acetabular breadth	Distance between meeting point most medial projection of right and left femoral head and acetabular notch of the right and left iliac blade.
Medio-lateral pelvic inlet breadth (Kurki and	Maximum distance between linea terminalis
Decrausaz, 2016)	of right and left iliac blades.

# 3.3.3. Analytical methods

# LMS method

Osteological, anthropometric and body composition measurements of the cross-sectional dataset were transformed into standard deviation scores (SDS) using the LMS method (Cole, 1990). This method is used to construct normalised growth standards. In the past, growth standards for different countries were produced in the form of centile charts (Tanner et al., 1966). A centile chart is a size for age chart that is produced to indicate whether a child falls in to normal, above or below normal size for their age (see Figure 3.1.). The lines that cross the plot are known as centiles. The term centile is short for percentile and indicates which percentage of child growth in a population a child would fall in to. For example, if a child A's height falls along the 50<sup>th</sup> percentile/centile than it means that 50% of children the same age are shorter than child A. If a child B's height falls along the 6<sup>th</sup> percentile/centile, 6% of children that are same the age are shorter than child B.



Figure 3.1. Height-for-age and weight-for-age growth centile chart for girls in the United Kingdom aged 8 to 18 years. From Royal College of Paediatric and Child Health (2012). Permission to reproduce this figure has been granted by the rights holder, Royal College of Paediatrics and Child Health.

Waterlow et al. (1977) suggested the use of SDS rather than centiles to better quantify growth status as applying centile charts to comparatively deprived populations resulted in a larger proportion of children falling below the lowest centile (Cole, 1990). An SD score is a normally distributed variable with a mean zero and a standard deviation 1. This enables variation in growth variables to be analysed

accounting for age and not simply for size. If growth charts were created using only size, this would exclude children who are either very small or very large for their age. However, anthropometric data are frequently not normally distributed, prompting van't Hof et al. (1985) to put forward using a power transformation based on the work of Box and Cox (1964). The power transformation of measurement data at each age removed skewness, making the data closer to a normal distribution. This method was further modified by Cole (1988;1989) into the LMS method. Cole's method uses three separate curves: L (lambda), M (mu) and S (sigma) (Cole, 1990). The M curve is a smoothed median curve which represents how the variable varies with age, whilst the S curve models the scatter of values around the mean and adjusts for any unusual distribution. The L curve represents skewness which is addressed by using the Box-Cox power transformation to accomplish normal distribution (Wells et al., 2012b). Raw data was converted into SDS and LMS scores using LMS Chartmaker Light Version 2.43 (Pan and Cole, 2010). LMS Chartmaker Light was also used to create centile charts. The SDS created when using LMS Chartmaker use national British growth references taken from Cole et al. (1995) and Freeman et al. (1995). These standards are created from multiple studies that have taken place between 1978 and 1990 and include data collected from approximately 30,000 children in England and Scotland aged between four weeks and 23 years of age (Cole et al., 1995).

LMS Chartmaker Light enables the user to either manually or automatically fit a model for the LMS curves of a specified data set. The goal of fitting a model for LMS curves is to produce a curve that fits the data well but is not over-specified (Pan and Cole, 2010). Age scale can be changed for the best fit of model. Equivalent degrees of freedom (edf) for each L, M, and S curve can also be toggled to better fit the model. Generally, a smaller edf is more appropriate for a best fit model and M edf > S edf > L edf (Pan and Cole, 2010). Alongside edf and age scale, deviance is another measure of checking goodness of fit for the growth curve model. Different deviance measures correct deviance for the best fit model in order to minimise deviance from the model. Five types of deviance measure are available on LMS Chartmaker Light. Pan and Cole (2010) recommend the use of either Schwarz Bayesian Criterion (SBC) or the Generalised Akaike Information Criterion (GAIC3). GAIC3 was used as a deviance measure for the growth curves in this study. The

97

curves in this chapter were fitted manually to ensure an adequate fit of curves to the SDS created and reduce the possibility of 'over-fitting' curves if curves were fitted automatically. Training for fitting curves was provided by Professor Jonathan CK Wells from the Institute of Child Health, University College London.

The SDS values used in this chapter were calculated by different researchers. The SDS values for shoulder breadth, bi-iliac breadth, medio-lateral inlet breadth, biacetabular breadth and head, waist, hip, thigh and calf circumference as well as skinfold-thickness measurements at the biceps, triceps, subscapular and suprailiac sites were calculated by the author of this thesis using LMS Chartmaker software. LMS Chartmaker calculates SDS based on national British growth references taken from Cole et al. (1995) and Freeman et al. (1995). These standards are created from multiple studies that have taken place between 1978 and 1990 and include data collected from approximately 30,000 children in England and Scotland aged between four weeks and 23 years of age (Cole et al., 1995). The SDS values for height, weight, fat mass lean mass calculated by Dr. Jane Williams at the Institute of Child Health, University College London as part of already completed studies on the reference sample created by Fewtrell and colleagues at the Institute of Child Health, University College London. Publications making use of these calculations include Wells et al., (2012b) and Devakumar et al., (2016). These studies also made use of the LMS method. There are no specific details available on the specific calculations performed by the LMS Chartmaker Light in the user guide to this program (Pan and Cole, 2010).

Growth curves provide references for the growth trajectory of different parts of a child's body (Cole, 1993). Creating growth curves for different parts of the pelvis address one of the major research questions of this thesis – what is the growth trajectory of the pelvis? A reference for the growth of the pelvis amongst healthy girls provides a basis to examine where and when factors that could affect the obstetric capacity of the bony pelvis impact the growth of the bony pelvis. Maternal height and pelvic canal dimensions have been found to associate in adult women (Thoms and Godfried, 1939; Liselele et al., 2000; Prasad and Al-Taher, 2002; Toh-Adam et al., 2012; Wells et al., 2017). The creation of pelvic growth curves will provide a point of comparison to height-for-age growth curves and may contribute to understanding how height and birth canal dimensions are linked in a developmental scenario.

# Growth velocity

The velocities of growth of the pelvic dimensions measurements were analysed to better understand the relationship between body composition change during puberty and pelvic dimensions amongst growing girls. Growth velocity charts give limits indicating normal range of ages at which adolescent growth spurts occur, and the scope of growth rates associated with children who mature comparatively early or late (Tanner and Whitehouse, 1976). Growth velocities in this chapter were created using the cross-sectional data set from the sample used in this chapter. The growth velocity charts were created through using monthly percentiles of growth for each variable. For example, weight at age 4.5 was subtracted from weight at age 4.6 to find how much weight had changed between these periods of time. This was calculated for girls at mean growth rate and for those at two standard deviations above and below the mean. Growth velocity of mass, stature, fat and lean mass were also calculated to better compare how body size and shape changes alongside pelvic dimensions.

## 3.4. Results

### 3.4.1. Sample used in this chapter

Descriptive statistics of the sample used in this chapter are available in Table 3.3. The sample used in this chapter is the cross-sectional data from Fewtrell and colleagues reference sample (see Section 3.2.1. for full description). The sample size for some variables is smaller than others due to availability of data for specific variables. Shapiro-Wilk's tests for normality results of for the SDS of the variables used in this chapter are shown in Table 3.4, showing fat mass, bi-iliac breadth, hip circumference and mean suprailiac thickness are not normally distributed. It is not uncommon for size and shape data collected from growing children not to be normally distributed as the size of different dimensions in children will increase at different rates (Cole, 1993). The SDS in the sample used for this chapter were

calculated using the LMS method (see Section 3.2.3. for more details) which reduces skewness of data distribution and shapes the data distribution into a more normal distribution (Cole, 1993).

	N	Min.	Max.	Mean	Std. Dev.
Age (yrs)	215	4.20	21.90	13.23	4.67
Weight (kg)	215	14.16	90.75	47.74	17.34
Height (cm)	215	100.90	181.70	150.67	19.86
BMI	215	13.18	34.54	20.18	4.02
Fat mass (kg)	203	2.33	39.96	14.21	7.98
Lean mass (kg)	203	11.59	61.91	34.37	10.51
Shoulder breadth (cm)	191	21.78	41.09	32.63	4.50
Bi-iliac breadth (cm)	194	14.09	29.39	22.43	3.43
Medio-lateral inlet breadth (cm)	195	6.53	14.51	10.70	2.00
Bi-acetabular breadth (cm)	195	7.70	15.98	11.40	2.04
Head circumference (cm)	215	48.50	61.00	54.31	4.36
Waist circumference (cm)	215	44.10	96.40	66.95	11.11
Hip circumference (cm)	215	47.20	117.80	84.50	16.48
Thigh circumference (cm)	213	29.50	102.60	47.69	10.45
Calf circumference (cm)	214	20.60	44.80	31.65	5.50
Mean biceps thickness (mm)	206	3.06	23.73	9.90	4.57
Mean triceps thickness (mm)	207	5.86	33.06	15.52	5.64
Mean suprailiac thickness (mm)	201	3.80	39.00	17.03	9.33

Table 3.3. Descriptive statistics of the study sample.

Table 3.4. Shapiro-Wilk's normality test results of the cross-sectional dataset used in this chapter.

	Shapiro-Wilk's statistic	df	p-values
Height SDS	0.988	169	0.158
Weight SDS	0.994	169	0.698
BMI SDS	0.993	169	0.551
Fat mass SDS	0.984	169	0.047
Lean mass SDS	0.996	169	0.947
Shoulder breadth SDS	0.992	169	0.523
Bi-iliac breadth SDS	0.980	169	0.013
Medio-lateral inlet breadth SDS	0.995	169	0.862
Bi-acetabular breadth SDS	0.996	169	0.946
Mid-upper arm circumference SDS	0.990	169	0.270
Head circumference SDS	0.992	169	0.479
Waist circumference SDS	0.988	169	0.159
Hip circumference SDS	0.957	169	0.000
Thigh circumference SDS	0.986	169	0.087
Calf circumference SDS	0.985	169	0.072
Mean biceps thickness SDS	0.986	169	0.100
Mean triceps thickness SDS	0.993	169	0.585
Mean subscapular thickness SDS	0.985	169	0.071
Mean suprailiac thickness SDS	0.975	169	0.004

\* Values are significant at <0.05

## 3.4.2. LMS method

Manual model fitting was used to create growth charts from the data collected from the cross-sectional dataset (n=215) with LMS Chartmaker Light. GAIC3 deviance measure was used to best fit the model used for LMS curves. LMS percentiles for shoulder breadth, bi-iliac breadth, medio-lateral pelvic inlet and biacetabular breadth against age for the sample are shown in Figure 3.2 to Figure 3.5. Shoulder breadth increased steadily from 4 to 15 years approximately and then entered a plateau from 16 years onwards with an s-shaped association between age and shoulder breadth. Adult shoulder breadth was established by 17 years. Bi-iliac breadth had no clear curvilinear association with age and increased steadily from 4 years to 22 years. This increase in breadth was not as acute after 15 years of age. It may continue to increase past 22 years of age however this cannot be verified with the present study sample. Medio-lateral pelvic inlet breadth and bi-acetabular breadth had a similar s-shaped growth curve. This measure also had a more acute increase in breadth between 10 and 15 years of age. Breadth continued to increase after 16 years of age though at a less acute rate. Bi-acetabular breadth increased at a greater rate between the ages of 10 and 16 with a reduction in growth rate similar to mediolateral pelvic inlet breadth. It is possible that bi-acetabular breadth and medio-lateral pelvic inlet breadth continue to increase past 22 years of age, though likely with a greatly reduced growth rate. Z score and percentile reference data for shoulder breadth, bi-iliac breadth, medio-lateral pelvic inlet breadth and bi-acetabular breadth are shown in Tables 3.4. to 3.7.



Figure 3.2. Centile chart of shoulder breadth. The third, tenth, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, 90<sup>th</sup> and 95th percentiles are displaced in ascending order.



Figure 3.3. Centile chart of BIIB breadth. The third, tenth, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, 90<sup>th</sup> and 95th percentiles are displayed in ascending order.



Figure 3.4. Centile chart of INML. The third, tenth, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, 90<sup>th</sup> and 95<sup>th</sup> percentiles are displayed in ascending order.



Figure 3.5. Centile chart of BIAC. The third, tenth, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, 90<sup>th</sup> and 95th percentiles are displayed in ascending order.

Age	z = -2.00	z = -1.33	z = -0.66	z = 0	z = 0.66	z = 1.33	z = 2.00
4	19.87	20.88	21.88	22.89	23.90	24.90	25.91
5	20.73	21.77	22.81	23.84	24.88	25.92	26.95
6	21.81	22.89	23.96	25.03	26.11	27.18	28.25
7	22.94	24.06	25.17	26.28	27.39	28.50	29.61
8	24.15	25.30	26.45	27.60	28.75	29.90	31.06
9	25.38	26.57	27.76	28.95	30.14	31.33	32.52
10	26.58	27.80	29.03	30.26	31.48	32.71	33.93
11	27.75	29.01	30.27	31.53	32.79	34.05	35.30
12	28.89	30.17	31.46	32.75	34.04	35.33	36.62
13	29.92	31.23	32.54	33.85	35.16	36.47	37.78
14	30.81	32.14	33.47	34.80	36.12	37.45	38.78
15	31.56	32.90	34.23	35.57	36.91	38.24	39.58
16	32.13	33.47	34.80	36.14	37.47	38.81	40.15
17	32.49	33.82	35.15	36.48	37.80	39.13	40.46
18	32.67	33.98	35.29	36.60	37.91	39.22	40.53
19	32.70	33.98	35.27	36.56	37.85	39.13	40.42
20	32.65	33.91	35.17	36.43	37.69	38.95	40.21
21	32.55	33.79	35.02	36.25	37.49	38.72	39.95
22	32.46	33.67	34.88	36.09	37.30	38.51	39.72

Table 3.5. Shoulder breadth reference data for study sample by zscore or standard deviation.

Table 3.6. Bi-iliac breadth reference data for study sample by zscore or standard deviation.

Age	z = -2.00	z = -1.33	z = -0.66	z = 0	z = 0.66	z = 1.33	z = 2.00
4	13.72	14.32	14.94	15.60	16.30	17.03	17.80
5	14.30	14.92	15.58	16.27	16.99	17.75	18.56
6	15.03	15.68	16.37	17.09	17.85	18.66	19.50
7	15.76	16.44	17.16	17.92	18.72	19.56	20.45
8	16.48	17.20	17.96	18.75	19.58	20.47	21.39
9	17.21	17.96	18.75	19.58	20.45	21.37	22.34
10	17.93	18.71	19.53	20.39	21.30	22.26	23.27
11	18.64	19.45	20.30	21.20	22.14	23.14	24.19
12	19.32	20.16	21.04	21.97	22.95	23.98	25.07
13	19.96	20.83	21.74	22.71	23.72	24.78	25.91
14	20.56	21.46	22.40	23.39	24.43	25.53	26.68
15	21.11	22.03	22.99	24.01	25.08	26.21	27.39
16	21.60	22.54	23.53	24.57	25.67	26.82	28.04
17	22.05	23.01	24.02	25.08	26.20	27.37	28.62
18	22.46	23.44	24.47	25.55	26.69	27.89	29.15
19	22.85	23.85	24.89	25.99	27.15	28.37	29.66
20	23.24	24.25	25.31	26.43	27.61	28.85	30.15
21	23.62	24.64	25.72	26.86	28.06	29.32	30.65
22	23.96	25.00	26.09	27.25	28.46	29.74	31.09
Age	z = -2.00	z = -1.33	z = -0.66	z = 0	z = 0.66	z = 1.33	z = 2.00
-----	-----------	-----------	-----------	-------	----------	----------	----------
4	5.72	6.14	6.56	6.98	7.40	7.81	8.23
5	5.98	6.41	6.83	7.26	7.68	8.11	8.54
6	6.31	6.74	7.18	7.61	8.04	8.48	8.91
7	6.64	7.09	7.53	7.97	8.41	8.85	9.30
8	7.00	7.45	7.90	8.35	8.80	9.25	9.70
9	7.41	7.87	8.32	8.78	9.24	9.70	10.16
10	7.86	8.33	8.80	9.27	9.74	10.21	10.68
11	8.37	8.85	9.33	9.81	10.29	10.78	11.26
12	8.92	9.41	9.90	10.40	10.89	11.38	11.88
13	9.46	9.97	10.47	10.97	11.47	11.98	12.48
14	9.97	10.47	10.98	11.49	12.00	12.51	13.02
15	10.40	10.90	11.41	11.92	12.43	12.94	13.45
16	10.74	11.24	11.74	12.25	12.75	13.25	13.76
17	11.00	11.49	11.99	12.48	12.97	13.46	13.95
18	11.20	11.68	12.16	12.64	13.12	13.59	14.07
19	11.37	11.83	12.29	12.76	13.22	13.68	14.14
20	11.52	11.97	12.41	12.86	13.31	13.75	14.20
21	11.68	12.11	12.54	12.97	13.40	13.82	14.25
22	11.83	12.24	12.65	13.07	13.48	13.89	14.30

Table 3.7. Medio-lateral pelvic inlet reference data for study sample by zscore or standard deviation.

Table 3.8. Bi-acetabular breadth reference data for study sample by zscore or standard deviation.

Age	z = -2.00	z = -1.33	z = -0.66	z = 0	z = 0.66	z = 1.33	z = 2.00
4	6.63	7.12	7.59	8.07	8.53	8.99	9.44
5	6.83	7.32	7.81	8.29	8.76	9.23	9.69
6	7.07	7.58	8.07	8.56	9.05	9.53	10.00
7	7.32	7.84	8.34	8.84	9.34	9.83	10.32
8	7.60	8.12	8.64	9.15	9.66	10.17	10.67
9	7.91	8.45	8.98	9.51	10.03	10.55	11.07
10	8.27	8.82	9.37	9.91	10.46	11.00	11.53
11	8.69	9.26	9.82	10.39	10.95	11.51	12.07
12	9.16	9.75	10.34	10.92	11.51	12.10	12.68
13	9.64	10.25	10.86	11.47	12.09	12.70	13.31
14	10.11	10.74	11.37	12.00	12.64	13.27	13.90
15	10.53	11.18	11.83	12.48	13.13	13.78	14.43
16	10.89	11.54	12.21	12.87	13.53	14.20	14.87
17	11.17	11.84	12.51	13.18	13.85	14.53	15.21
18	11.40	12.07	12.74	13.42	14.10	14.78	15.47
19	11.59	12.26	12.93	13.61	14.29	14.98	15.67
20	11.76	12.42	13.10	13.78	14.46	15.15	15.85
21	11.93	12.59	13.27	13.95	14.63	15.32	16.02
22	12.08	12.75	13.42	14.10	14.79	15.48	16.18

#### 3.4.3. Growth velocity

Charts of growth velocity with age plotted against incremental change in centimetre growth for shoulder breadth, bi-iliac breadth, medio-lateral pelvic inlet breadth and bi-acetabular breadth are shown in Figures 3.6 to 3.13. These plots were created using the raw reference data created from the growth curves in the previous section, which were developed from the cross-sectional dataset (n=215). Each chart shows values for growth velocity for mean SDS values. Growth velocity for shoulder and bi-iliac breadth is greatest in the years prior to puberty. The period of greatest growth velocity (centimetre increase per month) occurred between 5 and 8 years of age. Growth velocity for shoulder breadth slowed after from 8 to 11 years and then slowed more acutely from 11 years of age onwards. Bi-iliac breadth growth velocity does not continue between 17 and 22 years of age, stabilising to a similar degree to that between 8 and 11 years of age.

Medio-lateral pelvic inlet and bi-acetabular breadth growth velocities showed similar periods of increased growth velocity to peak, followed by marked decrease in growth rate. Unlike shoulder and b-iliac breadth, these pelvic dimensions showed very acute growth velocity prior to puberty coupled with marked decreases in growth velocity after 11 and 12 years of age. Growth velocity of medio-lateral pelvic inlet breadth increases in marked manner from 6 to 11 years of age. This velocity decreases rapidly from 12 to 18 years, stabilising from 18 to 22 years of age. For girls two standard deviations above the mean, peak growth velocity is reached approximately three months (11.5 years) before girls at mean growth velocity (11.75 years). The period of greatest growth velocity for bi-acetabular breadth occurred between 6 and 12 years of age, followed by a decrease in growth velocity between 12 and 19 years of age.

Growth velocity of weight, height, fat mass and lean mass are shown in Figures 3.10 to 3.13. Weight increased at the greatest velocity from 5 to 11 years of age, with reduced velocity until 18 years of age. Weight increase stabilised between 18 and 22 years of age. Height increased slowly between 5 and 6 years of age, slowing further between 9 and 18 years of age and (similar to weight) stabilising between 18 and 22 years of age. Fat mass increased slowly between 4 and 11 years and increased at a slower rate from 11 to 17 years old with a period of stabilisation after 17 years of age. Lean mass increased slowly until 11 years of age, with a marked increase in velocity between 11 and 19 years of age, stabilising after 19 years of age.



Figure 3.6. Growth velocity chart of shoulder breadth. Vertical axis shows fractions of a centimetre of shoulder breadth as girls grow. Each dot on the chart represents the change in shoulder breadth for mean growth. The dots were calculated using the mean SDS in Table 3.4.



Figure 3.7. Growth velocity chart of BIIB. Vertical axis shows fractions of a centimetre of bi-iliac breadth as girls grow. Each dot on the chart represents the change in bi-iliac breadth for mean growth. The dots were calculated using the mean SDS in Table 3.5.



Figure 3.8. Growth velocity chart of INML. Vertical axis shows fractions of a centimetre of medio-lateral inlet breadth as girls grow. Each dot on the chart represents the change in medio-lateral inlet breadth for mean growth. The dots were calculated using the mean SDS in Table 3.6.



Figure 3.9. Growth velocity chart of BIAC. Vertical axis shows fractions of a centimetre of bi-acetabular breadth as girls grow. Each dot on the chart represents the change in bi-acetabular breadth for mean growth. The dots were calculated using the mean SDS in Table 3.7.



Figure 3.10. Growth velocity chart of weight. Vertical axis shows fractions of a kilogram of weight as girls grow. Each dot on the chart represents the change in weight for mean growth. The dots were calculated using mean SDS values from reference data calculated in the same manner as Tables 3.4. to 3.6.



Figure 3.11. Growth velocity chart of height. Vertical axis shows fractions of a centimetre of height as girls grow. Each dot on the chart represents the change in height for mean growth. The dots were calculated using mean SDS values from reference data calculated in the same manner as Tables 3.4. to 3.6.



Figure 3.12. Growth velocity chart of fat mass. Vertical axis shows fractions of a kilogram of fat mass as girls grow. Each dot on the chart represents the change in fat mass for mean growth. The dots were calculated using mean SDS values from reference data calculated in the same manner as Tables 3.4. to 3.6.



Figure 3.13. Growth velocity chart of lean mass. Vertical axis shows fractions of a kilogram of lean mass as girls grow. Each dot on the chart represents the change in lean mass for mean growth. The dots were calculated using mean SDS values from reference data calculated in the same manner as Tables 3.4. to 3.6.

## 3.5. Discussion

#### 3.5.1. Tracking the growth of body and pelvic dimensions

This study presents growth centiles of body and pelvic dimensions in living girls and women between the ages of 4 and 22 years old from the United Kingdom. Growth velocity was also calculated for pelvic dimensions and body breadth measurements and fat mass, lean mass, height and weight. The reference data generated from the pelvic growth curves are novel for the United Kingdom and overall unique, as few scholars have created growth curves from pelvic dimensions. Moerman (1982) measured pelvic canal dimensions though did not produce centiles for pelvic growth of girls in the United States of America and Völgyi et al. (2010) produced growth curves for pelvic development in Finnish girls.

Centile charts for INML and BIAC show a similar trajectory of growth, with a steeper increase in growth beginning at 9 years old and slowing at 16 years of age. BMI centiles of British girls demonstrate that BMI increases markedly at approximately 8 years old before plateauing at approximately 20 years of age (Cole et al., 1995). INML and BIAC match the increase in BMI seen in centile charts of British girls. Centile charts for stature and weight in girls from the United States of America show that stature increases steadily from birth and plateaus at 15 years of age, whilst the period of greatest weight increases occurs between 8 and 14 years old (Ogden et al., 2002). INML and BIAC size change matches centiles for girls in the United States of America within a couple of years. Weight and BMI centiles for Swedish girls between 6 and 16 years old show that a marked increase in weight and BMI begins between 9 and 10 years of age, plateauing at 15 years old (Lindgren et al., 1995). This marked increase in soft tissue and body growth is slightly shorter in span than INML and BIAC in the reference sample for this study. Percentiles of fat and lean mass for the sample used in this study were calculated by Wells et al. (2012). Lean mass has a steady increase from 5 to 15 years of age, but plateaus after 16 years old, whilst fat mass increases steadily though the degree of increase is dependent on the percentile. INML and BIAC centiles are closely clustered, showing little variation dependent on percentile. This suggests that canal pelvic dimensions follow a similar growth pattern across all growth percentiles – it would be logical to

assume this would be protected from growth arrests to increase likelihood of obstetric sufficiency amongst women.

Body breadth increases relative to age in a different manner to pelvic dimensions. BIIB and shoulder breadth have similar growth curves, though they are not as similar as BIAC and INML. Shoulder breadth increase plateaus at approximately 17 years of age, whilst BIIB continues to increase from 4 to 22 years of age. There is slightly wider difference in increase in BIIB dimensions dependent on percentile compared to percentile differences in shoulder breadth. Shoulder breadth was collected in this study to clarify body breadth change throughout growth alongside pelvic change in females. It was additionally collected to track the development of shoulder breadth in relation to appearance of shoulder dystocia during childbirth. Shoulder dystocia occurs when the neonate's shoulders fail to navigate through the birth canal after the head is delivered (Smeltzer, 1986) which can lead to asphyxia and birth trauma to the neonate and maternal death (Trevathan and Rosenberg, 2000; Walrath, 2003; Wells, 2015). Body breadth can be reconstructed osteologically using clavicular length (Tague, 1994; Ruff et al., 2005) which is additionally used as a measure of predicting shoulder dystocia in clinical settings (Sherer et al., 2006). The growth trajectory of shoulder breadth in this sample suggests that it reaches maximum breadth prior to skeletal adulthood and biiliac breadth. A limitation on shoulder breath and a maternal line of inheritance on this limitation would be selectively advantageous to reduce the likelihood of shoulder dystocia.

Bi-iliac breadth was found to be less variable than the canal components of the pelvis (Kurki and Decrausaz, 2016) and to associate with body size overall (Betti et al., 2014). It is likely that in this study BIIB grows in a manner reflecting overall body size and latitude given that the reference sample is made of girls and women who have been born and raised in the south of England.

## 3.5.2. Growth velocity of the pelvis and other parts of the body

Figures 3.6, 3.7 through to 3.11 and Figure 3.13 show crossing over of standard deviations of growth velocity. For example in Figure 3.6. the growth velocity of girls who are two standard deviations above the mean (+2 S.D.) seems to slow

below the growth velocity of girls with the mean growth velocity between 15 and 16 years of age. The dots in each of these figures represent the fraction of growth occurring in each month of growth for the girls in this sample. For the girls whose growth velocity is occurring at two standard deviations above the mean, it would not be unexpected for their growth velocity to slow at a more noticeable rate than those who are growing at the mean growth velocity. This is likely the reason for which the trajectories of growth velocity for girls within the two standard deviation category appears to cross over the mean growth velocity trajectory.

### 3.5.3. Pelvic dimensions growth velocity and development

The twinned growth trajectory of INML and BIAC also appear in growth velocity curves. They show a clear increase in growth rate at approximately 7 years old and peak growth velocity is reached for both of these dimensions at approximately 12 years of age. BIIB does not have a clear peak in growth velocity and instead shows a reversed S-shape curve in growth velocity, with growth increase slowing between 10 to 17 years of age. Peak growth velocity for shoulder breadth occurs at 8 years of age and overall drops sharply after 12 years of age. These differences in peak growth velocity likely are a result of differing growth trajectory prioritisation and canalisation. Body breadth or the girth of limbs are not as equally heritable as height or limb length (Bogin, 1999a). Hox genes are shared across all taxa and are a group of genes known to regulate the growth of body segments such that particular elements of the human body plan are genetically established (Bogin and Varela-Silva, 2010). Maternal anthropometry was found to predict neonatal trunk size and adiposity in Australian newborns whilst head circumference and limb lengths could be predicted by paternal anthropometry (Pomeroy et al., 2015a). Different tissue types are also prioritised over others during growth. Bogin (1999) collates growth curves for different tissue types (see Figure 3.11.), demonstrating the prioritisation of brain growth prior to 10 years of age, with body and reproductive growth increasing markedly at 10 years of age. This suggests that some elements of body breadth, including BIIB and shoulder breadth, may be less susceptible to environmental influences as their twinned growth may be representative of body growth more generally.



Figure 3.14. Growth curves for different body tissues. From Bogin (1999b). Permission to reproduce this figure has been granted by the rights holder, Cambridge University Press.

The variations in centile shapes in this study indicate differential hormone actions on specific areas of pelvic and body breadth growth. The human S-shaped growth curve is unique in its sequence prior to puberty: a nadir, followed by swift acceleration of growth during mid-puberty (Rogol et al., 2002). During this acceleration, the hypothalamus is stimulated to produce gonadotropin-releasing hormone, which is then released throughout the adolescent period in pulses. The pituitary gland in turn secretes luteinizing hormone and follicle stimulating hormone (oestrogen hormones). In women these hormones stimulate the growth of ovaries and the releases of ovarian hormones. The steroid hormone estradiol is an oestrogen hormone that regulates menarche, secondary sexual characteristic development and the accrual of fat deposits on hips, thighs and buttocks (Norgan, 1997). Prader (1984) and Preece et al. (1984) found that estradiol secretion associates significantly with the adolescent growth spurt in girls as well as breast development. Worthman (1993) found that serum estradiol levels notably rose between 11 and 12 years of age in healthy girls living in London, whilst Apter and Vihko (see Ellis, 2004 for full list of references) found that higher estradiol levels prior to the age of 13 years predicted earlier maturation and age at menarche in Finnish girls. It is possible that peak growth velocity for INML and BIAC is affected by the initial increase in estradiol at this age and prompts skeletal growth as a form of preparation for reproduction in a similar

manner to driving the accrual of fat deposits in preparation for pregnancy and lactation. This is particularly important due to the energetic cost of lactation (Prentice and Prentice, 1988). Mean age at menarche in this sample is 12.57 years of age, which is slightly later than peak growth velocity is reached for INML and BIAC. Given the reduced rate of growth of pelvic dimensions compared to linear growth (Moerman 1982; Völgyi et al., 2010) there would be a selective advantage to 'front-loading' initial growth stages of the pelvic canal as the biomechanical demands of childbirth are a greater selective force than energy availability during the neonate's development.

This is not to suggest that pelvic canal shape will be fixed once a woman has reached skeletal maturity. Berger et al. (2011) found that the pelvic inlet did not widen with increasing age, but that pelvic size increased across the femoral trochanters, at the iliac blades (bi-iliac breadth) and between the femoral heads (bi-acetabular breadth) as age increased. Conversely, Huseynov et al. (2016) found that after 40 years of age obstetrically-relevant dimensions of the canal became constricted, resembling male pelvic canal dimensions and leading the authors to suggest this as evidence for 'on-demand' adjustment of pelvic size to meet the needs of childbirth. It should be noted that Huseynov et al.'s (2016) work did not analyse cross-sectional data and cannot be compared to the results for this project without caution. Mitteroecker and Fischer (2016) were able to reproduce some of Huseynov et al. (2016)'s results though critiqued their interpretation of selection acting on adult women past childbearing age. Ponce de León et al. (2016:E3597) responded to these critiques by classifying variation in the pelvic canal throughout a woman's growth and adulthood as a result of an 'evolved mechanism of hormone mediated developmental plasticity'. The results of this study contribute to this ongoing debate on the complexity of genetic, hormone and skeletal interactions throughout a woman's lifetime.

## 3.5.4. Pelvic dimensions growth and preparing the body for childbirth

Examining pelvic dimension growth in women gives a developmental insight to the OD. Clinical pelvic contraction thresholds define the medio-lateral pelvic inlet as contracted below 120mm (Cunningham and Williams, 2005). The INML centile chart shows that women in the second, ninth and 25<sup>th</sup> percentiles at 20 years old and

above would fall within clinically contracted thresholds for this measure. The identification of possible pelvic contraction as a result of growth variance is essential in working to prevent this issue in living populations, as a woman with only one contracted pelvic dimension can still be at risk for cephalo-pelvic disproportion, with the midplane being one of the most problematic dimensions for contraction (Gabbe et al., 2002; Cunningham and Williams, 2005). The female body prepares both skeletal and soft tissue throughout growth for reproduction, including accruing fat deposits throughout the body as reserves for pregnancy and breastfeeding. Fat mass in girls the second, ninth and 25<sup>th</sup> percentile from this sample remains below 10kg from 5 to 20 years of age, with a larger increase in fat mass between the 50<sup>th</sup> percentile and those above (Wells et al., 2012b). Whilst the distances between percentiles in INML growth are not identical to those in fat mass, it is likely that the girls who have contracted INML dimensions may also fail to gain fat deposits as a means of preparing for childbirth. Using Frisch and McArthur 's (1974) estimations, 22% body fat would be expected for regular menstrual cycles though these estimations for regular menstrual cycles have been critiqued (Wells, 2010b; Ellison et al., 2012) and there is universally agreed amount of fat mass necessary for regular menstrual cycles compared to carrying a pregnancy to term and being successfully able to breastfeed (Norgan, 1997).

It is important to note that these centile charts are representative of mainly healthy European populations, and may not be appropriate as benchmarks for obstetric sufficiency during growth in other populations. Maternal height (as a reflection of maternal pelvic outlet size) has been clinically shown to predict obstructed labour (Toh-Adam et al., 2012), suggesting that pelvic outlet size and skeletal body size are developmentally linked. However, pelvic canal dimorphism does not consistently correlate with female body size. Baragi and colleagues (2002) identified pelvic floor shape differences between European-American and African-American women from the Hamann-Todd Osteological Collection: African-American women were found to have a shorter bi-spinous breadth and ischial spine-to-sacrum length. Rizk et al. (2004) found that European/White women living in the United Arab Emirates had larger pelvic inlet and outlet dimensions than women from non-European/White ethnicities also living in the United Arab Emirates. The centile charts and growth velocity curves presented here demonstrate the variation in developmental parameters of the obstetric components of the pelvis within one population and should not be used a template to examine the growth of the pelvis in populations of different ancestry or health status.

A limitation for this study was the selection of pelvic measurements collected from the DXA scans. DXA scans of the pelvis present a two-dimensional image of a three-dimensional object that limits the scope of choice of possible pelvic measurements examining pelvic depth. This methodological limitation was explored in greater depth in Chapter 2. The most obstetrically important dimensions of the pelvis include the posterior space of the midplane and the pelvic outlet (Cunningham and Williams, 2005). These dimensions were not collected in this study due to limitations of three-dimensional imaging possible in the DXA, and so multidimensional analyses of pelvic canal growth cannot be made with these results. Another limitation for this study is the age range of the participants in the sample. Whilst this study tracked pelvic growth through childhood into early adulthood, no further data was collected past 22 years of age meaning that change in the pelvis in adulthood could not be examined.

# 3.6. Conclusion

Outcomes from this study indicated that body breadth and pelvic dimensions have different growth trajectories. Growth curves for INML and BIAC have similar Sshaped curves showing a marked increase in growth between 10 and 16 years of age, whilst BIIB increases consistently from 4 to 22 years old. Shoulder breadth increased steadily from 4 to 15 years old and plateaued from 16 years of age and onwards. There was a similar pattern in twinning growth velocity for INML and BIAC. Peak growth velocity for these dimensions occurred between 11 and 12 years of age and velocity dropped acutely until 19 years of age. Peak growth velocity for shoulder breadth occurred at 8 years old and continued to slow in velocity until 18 years of age. There is no clear peak in growth velocity for BIIB though growth velocity decreases between 10 and 18 years of age. The presentation of these growth curves and associated reference data are novel in both clinical and research settings, as they demonstrate both similarities and differences to growth curves and growth velocity for lean and fat mass, as well as weight and height. Growth velocity of INML and BIAC demonstrate that early childhood nutrition (prior to puberty) is a key factor in preventing obstructed pelvic dimensions, and that pelvic shape and size may further alter throughout puberty and indeed early adulthood to better prepare the body for childbirth. This suggests that the OD has developmental origins but may also be affected by environmental variables.

# 3.7. The growth trajectory of the pelvis and biological features that associate with pelvic breadth in growing girls

The results of this chapter demonstrate different growth trajectories for different breadth measurements across the pelvis in growing girls, as well as differences between the growth trajectories of soft tissue (such as fat mass) and the bony pelvis. These results elucidate the growth trajectory of the pelvis as a whole, though they do not suggest any linked growth trajectories with other parts of the body. This chapter has provided the basis for examining biological features of the growing female body that associate with pelvic breadth in Chapter Four. Results from Chapter Four not only expand on the results from Chapter Three, but also may suggest biological features that could indicate pelvic canal breadth compromise during growth.

## CHAPTER FOUR:

#### The biological factors associated with female pelvic growth

#### 4.1. The role of Chapter Four in this thesis

This chapter builds on the results of Chapter Three by testing the association of different biological variables with pelvic canal breadth variables in growing girls. The aim of this chapter is to bridge the gap between a linear understanding of the growth of the pelvic canal and a more complex understanding of how the growth of skeletal tissue associates with the growth of soft tissue for growing girls. The dataset used in this chapter is the same as that used in Chapter Three.

## 4.2. Introduction

Pelvic variation among adult women has been implicated as a factor in the aetiology of obstetric complications, yet the relationship between biological factors and bony pelvic growth in girls are poorly understood. Studies of human growth have been carried out to develop interventions for healthy infant development and to guantify human adaptability to alterations in resources and disease load (Tanner, 1981a; Bogin, 1999a; b). Some more recent studies have examined infant growth as a predictor for chronic health issues (Barker et al., 1989, 2002; Lucas et al., 1999; Barker, 2007). Long-term health consequences of infant growth have prompted some researchers to examine evolutionary perspectives on growth, including long-term health consequences of pelvic growth. Wells et al. (2012) proposed that ecological factors early in life could have impacted pelvic capacity in adult women, bringing cephalopelvic disproportion (CPD) to the forefront of the multiple obstetric dilemmas that women face in giving birth. An investigation of the associations between biological factors and pelvic growth amongst living girls and women would provide a template with which to understand what impacts on the development of an obstetric sufficient (or indeed insufficient) bony pelvis.

Linear skeletal growth in boys and girls is not dissimilar until the onset of puberty. Peak height velocity occurs for girls at 12 years of age and around 14 years of age for boys (Marshall and Tanner, 1969, 1970; Rogol et al., 2002). On average peak lean body mass velocity occurs at 15 or 16 years of age in girls and approximately 17 years in boys, overall following a similar trajectory to height and weight increase during growth (Ulijaszek et al., 1998). When it comes to fat mass, girls accrue greater quantities of fat throughout puberty, leading to a greater amount of fat in women compared to men in adulthood (Norgan, 1997; Ulijaszek et al., 1998; Rogol et al., 2002; Wells, 2006). Trunk length seems to vary less than lower limb length, suggesting leg length has greater sensitivity to environmental factors during growth for both girls and boys (Gunnell et al., 1998; Wadsworth et al., 2002; Bogin and Rios, 2003). Shoulder breadth increases earlier in boys than in girls, taking place alongside peak muscle growth velocity (Tanner, 1981b). Sheehy et al. (1999) found that shoulder breadth increased notably during the midgrowth (approximately 6 to 8.5 years old) before increasing again during the pubertal growth spurt in Swiss boys and girls. The same study demonstrated that bi-iliac breadth increased after leg length, height and shoulder breadth respectively, as well as a similar growth velocity in boys and girls (Sheehy et al., 1999).

Few scholars have examined the growth of the pelvis in living girls. Greulich and Thoms (1944) found: the pelvic canal was constricted (with an inward projection of the pelvic wall at the level of the acetabula) in girls 3 to 4 years prior to menarche; that during puberty the canal increased in width before it increased in anteriorposterior breadth; and, that this pattern of growth began earlier with girls who started puberty early. In their study breast bud development and the first appearance of axillary hair either preceded or occurred soon after pelvic canal changes. Reynolds' (1947) study of pelvic growth of children between 2 and 9 years of age found that inner pelvic measurements (including the pelvic inlet) were absolutely or relatively larger in girls than in boys. Coleman (1969) found that pelvic inlet change during adolescent growth in girls related to changes in the inferior functional division of the pelvis i.e., the midplane and outlet of the canal. Coleman suggested that the widest point of the inlet moved inferiorly during growth as the ischia grew laterally. Loder (2002) examined pelvic canal dimension changes that could affect childbirth difficulty in girls who had undergone pelvic osteotomies. Pelvic osteotomies are carried out to correct acetabular dysplasia and (more rarely) Legg-Perthes disease. Loder (2002) found that midplane dimensions were narrower in girls who underwent pelvic

128

osteotomies when they were 7.1±4.9 and 11.9±7.9 years old. Moerman (1982) found that the bony pelvis overall grows more slowly than height in healthy girls and that the birth canal continues to grow once height no longer increases. More recently, Völgyi et al. (2010) examined pelvic breadth widening in growing girls and also found that pelvic breadth continued to increase past the cessation of height increase. Völgyi et al. (2010) additionally found that peak growth velocity of bi-iliac breadth occurred 11.6 months prior to menarche, whilst peak growth velocity of the mediolateral inlet breadth occurred 13.5 months prior to menarche. Sharma et al.'s (2016) analyses of pelvic size change amongst growing girls in India demonstrated that pelvic canal growth lagged behind stature and bicristal breadth increase, suggesting that the delay in pelvic breadth increase relative to stature during growth may not be unique to European populations.

The purpose of this study is twofold: 1) to examine if pelvic dimensions track other variables of growth (including increasing height and weight) as girls grow into women and 2) to test if biological variables such as height and birthweight, pubertal indicators such as age at menarche and parental body size and shape associate with pelvic dimensions. These variables include height, age at menarche, maternal body mass index, paternal body mass index, birthweight, head circumference and external indicators of pubertal development. Using the methodologies established in Chapter Two, this study integrates data collected from DXA scans with anthropometric and pubertal development data collected from the same growing girls. Clarifying the associations between biological factors and pelvic canal growth in growing girls is essential to better address biomechanical childbirth issues in living women. Integrating data on changing soft tissue such as fat and lean mass as well as measures of body breadth (such as shoulder breath) during growth will further illuminate overall patterns of body size and shape alteration from childhood to adulthood. Result from this study will also assist in detecting pelvic dimensions that may have resulted in childbirth issues in women who have died in the past.

The following hypotheses will be made in response to the research question: Do pelvic dimensions be associate with birthweight, height, head circumference, measures of adiposity and body breadth relevant to reproduction, inheritance from parental body size and shape or age at menarche in girls?

H<sub>01</sub>: Pelvic dimensions do not associate with birthweight in growing girls.

 $H_{A1}$ : Birthweight is associated with pelvic dimensions in growing girls as birthweight is a strong predictor of adult body size (Barker et al., 1989, 2002; Novotny et al., 2000; Barker, 2007).

H<sub>02</sub>: Pelvic dimensions do not associate with height in growing girls.

H<sub>A2</sub>: Height is associated with pelvic dimensions in growing girls as short maternal stature is a risk factor for biomechanical childbirth difficulties (Mahmood et al., 1988; Liselele et al., 2000; Prasad and Al-Taher, 2002; Gudmundsson et al., 2005; Stulp et al., 2011).

 $H_{03}$ : Pelvic dimensions do not associate with head circumference in growing girls.

 $H_{A3}$ : Head circumference associates with pelvic dimensions in growing girls as Fischer and Mitteroecker (2015) demonstrated that women with a smaller head have a more oval pelvic inlet whilst women with larger heads have rounder pelvic inlets.

H<sub>04</sub>: Pelvic dimensions do not associate with parental body size and shape in growing girls.

 $H_{A4}$ : Parental body size and shape associates with the pelvic dimensions of growing girls as Devakumar et al.'s (2016) work demonstrated associations between mother's and daughter's BMI and Pomeroy et al. (2015) found associations between neonatal trunk and head size and mother's anthropometry.

H<sub>05</sub>: Pelvic dimensions do not associate with measures of adiposity in growth girls.

 $H_{A5}$ : Hip and thigh circumferences associate with pelvic dimensions in growing girls. Fat is stored in the gluteo-femoral region of the body (around the buttocks and thighs) to prepare the female body for the energy lost during lactation (Wells, 2018).

H<sub>06</sub>: Pelvic dimensions do not associate with age at menarche in growing girls.

H<sub>A6:</sub> Age at menarche associates with pelvic dimensions in growing girls. Völgyi et al. (2010) found that peak growth velocity of bi-iliac breadth and mediolateral pelvic inlet breadth occurred approximately 1 year prior to age at menarche in Finnish growing girls. Novotny et al. (2000) found that age at menarche predicted the distance between the greater trochanters in Hawaiian women.

## 4.2. Materials and methods

## 4.2.1. Study sample

The sample for this study (f=286) was collected as a reference sample at the Great Ormond Street Hospital in London, UK between the years 2001 and 2016. Further details on the sample are available in Chapter Three. Both longitudinal (n=54) and cross-sectional (n=232) datasets were used in specific analyses for this study. The dataset used is noted in the description of each analytical method.

## Body composition data collection

Full details of the fat and lean mass data collection are available in Chapter Three. Bone mineral content was collected using dual-energy X-ray absorptiometry (DXA; GE Medical Systems, UK) and total body water data was collected using deuterium dilution (Devakumar et al., 2016). Detailed explanations of ADP, DXA and deuterium dilution are available in Chapter Three. Full details of 4C calculations can be found in Wells et al. (2012). The anthropometric data collected for the reference sample are included in Table 4.1. Table 4.1. Description of anthropmetrics collected from participants in the reference study.

Anthropometrics	Description
Head circumference (collected using a soft tape measure)	Tape measure placed around the head such that it lays across the frontal bones of the skull, slightly above the eyebrows; perpendicular with the long axis of the face; above the ears and over the occipital protruberance at the back of the head (Centres for Disease Control and Prevention, 2007).
Waist circumference (collected using a soft tape measure)	Hip area palpated to locate the right ilium. Researcher marked uppermost lateral border of the right ilium with a cosmetic pencil and crossed this mark a the midaxillary line (extends from the armpit down the side of the torso). Tape measure placed around the waist at the level of the measurement mark (Centres for Disease Control and Prevention, 2007).
Thigh circumference (collected using a soft tane measure)	Measurement collected directly below the duteal fold of the right thigh
Calf circumference (collected using a soft tape measure)	Measurement collected at the point of maximum girth of the calf.
Mean biceps thickness (collected using skinfold calipers)	Measurement collected over the mid-point of the muscle belly with the arm resting supinated on the subject's thigh (Durnin and Rahaman, 1967).
Mean triceps thickness (collected using skinfold calipers)	Measurement collected over the mid-point of the muscle belly, mid-way between the olecranon and the tip of the acromion of the scapula, with the upper arm hanging loosely by the body of the participant (Durnin and Rahaman, 1967).
Mean subscapular thickness (collected using skinfold calipers)	Measurement collected just below the tip of the inferior angle of the scapula, at an angle of about 45° to the vertical, with the upper arm hanging loosely by the body of the participant (Durnin and Rahaman, 1967).
Mean suprailiac thickness (collected using skinfold calipers)	Measurement collected just above the iliac crest in the mid-axillary line (which extends from the armpit down the side of the body) (Durnin and Rahaman, 1967).

# Physical development data collection

All non-adult subjects were asked to select an image that corresponded to the stage of development of their secondary sexual characteristics (pubic hair for both girls and boys, breast development for girls, penis and testes development for boys, see Appendix Two). These images and associated number scale were based on Tanner's scale of secondary sexual characteristic development (Tanner, 1962) and are available in Figure 4.1. Marshall and Tanner (1969) created a scale that used both photographs and descriptions of external physical primary and secondary sexual characteristics. The scale was designed to account for variation in the chronological age at which girls and boys displayed sexual characteristics during development by focusing on specific stages rather than chronological age. Girls in this study were asked to note the date of their first menstrual period, the length of their menstrual cycle and whether they were using an oral contraceptive pill or had a contraceptive implant.



The pubic hair stages are as follows:

- The pubic hair stages are as follows: Stage 1: Pre-adolescent; the vellus over the pubes is not further developed than that over the anterior abdominal wall, i.e. no pubic hair. Stage 2. Sparse growth of long, slightly pigmented, downy hair, straight or only slightly curled, appearing chiefly along the labia. This stage is difficult to see on photographs, particularly of fair-haired subjects. Though Stage 2 rating was used in this study, it cannot be regarded as reliable, and the ages at which subjects are said to have reached Stage 2 are almost certainly too late. certainly too late.
- Stage 3: Considerably darker, coarser, and invote of the The hair spreads sparsely over the junction of the pubes. This and subsequent stages were clearly recognizable on the photographs.



Frg. 1. -Standards for breast ratings. (From Tann 1969.)

The breast stages are as follows.

- Stage 1: Pre-adolescent; elevation of papilla only. Stage 2: Breast bud stage; elevation of breast and papilla as a small mound, enlargement of areola diameter.
- Stage 3: Further enlargement of breast and areola, with no separation of their contours.
- Stage 4: Projection of areola and papilla to form a secondary mound above the level of the breast. Stage 5: Mature stage; projection of papilla only, due

to recession of the areola to the general contour of the breast.

Figure 4.1. Tanner development stage chart. From Marshall and Tanner (1969). Permission to reproduce this figure has been granted by the rights holder, BMJ Publishing Group Ltd.

## 4.2.2. Osteological measurements

Linear measurements of the skeleton were collected from DXA scans of the entirety of Fewtrell and colleague's reference study sample (see Chapter Three for more details on this sample). Osteological measurements were selected to capture variation in body and pelvic dimensions and are detailed in Chapter Three. DXA scans were visualised using enCORE 2002 software, and measurements were collected using the Line ROI measurement tool available on the Custom Analysis toolbar (G.E. Healthcare, 2012). Medio-lateral pelvic inlet breadth measurements were taken with maximum zoom capacity for each scan.

## 4.3. Analytical methods

## A note on cross-sectional vs. longitudinal datasets used in this chapter

The analytical methods for this chapter differed depending on whether crosssectional or longitudinal data were used. The separation of cross-sectional and longitudinal datasets addresses the fact that particular statistical analyses cannot be performed on longitudinal datasets (that is, datasets made of repeated measurements of the same individuals). The dataset used for each analytical method is listed with the explanation of the analytical method in question. Body size and shape variables (pelvic dimensions, height and head circumference) were converted into SDS to account for age-related variation. Further details on SDS are available in Chapter Three. The subsamples were tested for normality using Shapiro-Wilks test (Shapiro and Wilk, 1965).

# Spaghetti plots

Spaghetti plots were produced to visually present the growth trajectory of pelvic dimensions (bi-iliac breadth [BIB], medio-lateral inlet breadth [INML] and biacetabular breadth [BIAC]) and body breadth (shoulder breadth) in each girl in the longitudinal dataset. Spaghetti plots are best for plotting longitudinal data collected from individuals to flag usual patterns in growth amongst a cohort, as well as determine the overall trend in growth trajectory of a cohort (Kwok et al., 2008). Spaghetti plots were also created for pelvic and body breadth SDS to examine potential centile crossing as girls aged. Presenting longitudinal data as spaghetti plots will clarify if there are key growth periods for specific measures of breadth through the body and the pelvis. For example, this method of presenting longitudinal data will show if girls with a small BIAC breadth in childhood will have a small BIAC early adulthood. Spaghetti plots provide a comparison with growth curves, as spaghetti plots represent only the data collected from a specific cohort whilst growth curves are created using values from a wider reference sample. All spaghetti plots were created using Microsoft Excel for Macintosh 2011.

# **Conditional references**

Conditional references were produced from the longitudinal dataset to examine if pelvic dimensions track as girls grow. For example, these references enable the researcher to find BIIB of a 14-year-old girl knowing her BIIB when she was 10 years old. Conditional references present growth velocity of a particular dimension that compensate for regression to the mean. Regression to the mean is a statistical phenomenon that states that when individuals are measured once and then measured again at a later date, the measurement on the later date tends to be closer to the median than the first measurement date (Cole, 1995). The amount of regression to the mean of a measurement depends on how highly correlated a measurement is at two different ages. The calculation of conditional references follows these steps (Cole, 1995):

- Create SDS for measurement of interest for every individual who has been measured at every age in longitudinal dataset (do not include individuals who have only been measured on two or three occasions out of the full complement of measurement sessions).
- 2. Use linear regression to regress current SDS (SDS<sub>2</sub>) on previous SDS (SDS<sub>1</sub>)
- Present results of linear regression as a correlation matrix. The correlation coefficient is always below one, such that SDS<sub>2</sub> is expected to become smaller with passing time.

## SDS values used in multivariate analyses

Data from the cross-sectional dataset were used in multivariate analyses. Specifically the SDS values of the variables representing pelvic breadth, body size and shape and pubertal development were used in the multivariate analyses in this chapter. The benefits of using SDS instead of raw data to analyse data collected from growing children are detailed in Chapter Three. The SDS values of shoulder breadth and pelvic breadth data in this chapter, similar to the same cross-sectional dataset used to create the growth curves in Chapter Three, were created using LMS Chartmaker (Pan and Cole, 2010) by the author of this thesis. LMS Chartmaker includes growth references for healthy children living in the UK during the 1990s to create SDS values from raw data on body dimensions. The SDS values for all other body size and shape variables (i.e. head circumference, thigh circumference) and measures of adiposity (i.e. skinfold thickness) were calculated using more recent growth standards created by (Wells et al., 2012b), which were also created from data collected from healthy children living in the UK. Fewtrell and colleagues calculated the SDS values for the body size and shape variables and measures of adiposity as part of their reference sample (more details on this are available in Chapter Three).

#### Multivariate analyses

Outliers were identified using the formula 1.5IQR, which identifies values that are 1.5× the interquartile range away from the sample median. Only physically impossible outliers were removed. For example, the height of a healthy 16 year old was incorrectly noted as 95cm, meaning that this individual was removed from the dataset used for analyses. SDS values of the cross-sectional dataset were used in a multiple linear regression analysis to examine the relationship between biological variables (height, head circumference, age at menses, measures of body breadth and adiposity, Tanner development stage and parental stature and weight) and pelvic dimensions. Data were visually inspected as scatterplots to look for indications of linear relationships prior to performing linear regression analyses. Only data showing linear relationships in the scatterplots were entered in to the linear regression analyses. Independence of observations was tested using the Durbin-Watson statistic (Durbin and Watson, 1951). This is a measure of autocorrelation, which is the similarity of values measured over successive time intervals. Generally Durbin-Watson test statistics between 1.5 and 2.5 are considered normal. Field (2009) suggests that Durbin-Watson statistics lower than 1 or over 3 indicate problematic data.

The homoscedasticity of the data were examined visually from plots of standardized residuals plotted against pelvic dimensions and body breadth measurement SDS. Homoscedasticity describes a situation in which the error term (the random disturbance in the relationship between the independent variables and the dependent variable) is the same across all values of the independent variables. Checking the homoscedasticity of the data confirms if the variance around the regression line is the same for all values of the independent variable. Residuals should broadly continue along the same trajectory as the line of regression in the plot of standardized residuals and dependent variable.

The data's multicollinearity were examined using Tolerance and Variance Inflation Factors (VIF). Measures of multicollinearity identify similarity between independent variables, as similarities between independent variables will result in strong correlations. VIF values between 1 and 10 demonstrate that there is no multicollinearity between independent variables, whilst VIF values below 1 and above 10 suggest multicollinearity between independent variables (Shieh, 2011). The normality of residuals was checked using histograms and normal probabilityprobability (P-P) plots based on standardized residuals. Normal distribution of residuals is suggested when a) the histogram of the standardized residuals shows an approximately normal distribution and b) the normal P-P plot shows residuals falling on the diagonal line of identity.

Linear regression has been used successfully to examine relationships between biological variables in human growth, including for the sample used for this study (Galton, 1886; Pearson, 1896; Williams et al., 2005; Wells et al., 2007, 2010; Pomeroy et al., 2015a; Devakumar et al., 2016). All statistical analyses were conducted in SPSS for Macintosh (IBM Statistical Package for the Social Sciences, Version 20.0).

137

# 4.4. Results

# 4.4.1. Longitudinal dataset

Descriptive statistics for the longitudinal dataset are presented in Table 4.2.

Table 4.2. Descriptive statistics for longitudinal data subsample.

	Ν	Min.	Max.	Mean	Std. Deviation
Age (yrs)	54	5.80	19.50	13.08	3.26
Weight (kg)	54	16.96	93.51	48.82	15.57
Height (cm)	54	112.10	180.60	153.00	14.36
BMI	54	12.50	35.24	20.29	4.09
Fat mass (kg)	54	2.30	35.57	12.36	6.42
Lean mass (kg)	54	13.48	50.49	32.86	8.68
Shoulder breadth (cm)	54	23.51	41.65	33.64	3.72
Bi-iliac breadth (cm)	54	16.30	29.06	23.30	2.93
Medio-lateral inlet breadth (cm)	54	6.54	14.22	10.81	1.84
Bi-acetabular breadth (cm)	54	7.56	15.29	11.50	1.72
Mid-upper arm circumference (cm)	54	15.60	37.50	24.79	4.18
Head circumference (cm)	54	48.70	59.50	54.43	1.99
Waist circumference (cm)	54	47.20	101.50	68.20	9.63
Hip circumference (cm)	54	53.30	167.80	86.27	14.01
Thigh circumference (cm)	54	29.60	70.00	49.11	7.95
Calf circumference (cm)	54	21.40	45.00	32.26	4.46
Mean biceps thickness (mm)	54	2.40	23.13	9.89	4.11
Mean triceps thickness mm	54	5.53	30.73	15.93	5.29
Mean subscapular thickness (mm)	54	3.67	34.00	11.73	5.73
Mean suprailiac thickness (mm)	54	4.47	77.00	18.94	9.65
Tanner development stage	54	1	5	3.12	1.35
Age at menarche (months)	54	88.00	207.00	152.82	13.50
Maternal weight (kg)	54	0.30	85.00	63.02	10.46
Maternal height (cm)	54	152.00	171.30	162.11	4.84
Paternal weight (kg)	54	60.00	121.00	84.93	12.14
Paternal height (cm)	54	165.10	203.20	180.22	8.16
Birthweight (grams)	54	2260	4550	3540.64	457.79

# 4.4.2. Spaghetti plots

Spaghetti plots were created to display growth trajectories of pelvic dimensions and body breadth development for each girl in the longitudinal dataset, both in raw measurement data and SDS for each measurement type. These plots are presented in Figures 4.2. to 4.9. Shoulder breadth development increases consistently from 10 years onwards and the shoulder breadth SDS spaghetti plot shows that many girls cross standard deviations throughout growth into adulthood. BIIB increases with a similar trajectory as shoulder breadth, with the largest increase in size taking place between 10 and 15 years. The BIIB SDS spaghetti plot shows that the BIIB of girls in the sample grows tightly within the same standard deviation throughout childhood and adolescence, particularly compared to shoulder breadth. INML increases in size for almost all girls in an S-shaped curve with a marked increase in size between 10 and approximately 16 years. One girl is notably larger than the sample overall and her growth trajectory is more linear in shape. The INML SDS spaghetti plot shows a greater number of girls who cross standard deviations during growth compared to BIIB SDS. Compared to INML growth, the spaghetti plot of BIAC growth shows that increase in BIAC breadth throughout growth is more uniform throughout the sample. Lines representing individual growth trajectories are closely clustered together for the sample. The growth trajectory for BIAC is more linear in shape than INML. The BIAC SDS spaghetti plot shows a greater proportion of girls crossing standard deviations than INML SDS spaghetti plot.



Figure 4.2. Spaghetti plot of shoulder breadth. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.



Figure 4.3. Spaghetti plot of shoulder breadth SDS. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.



Figure 4.4. Spaghetti plot of BIIB. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.



Figure 4.5. Spaghetti plot of BIIB SDS. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.


Figure 4.6. Spaghetti plot of INML. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.



Figure 4.7. Spaghetti plot of INML SDS. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.



Figure 4.8. Spaghetti plot of BIAC. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.



Figure 4.9. Spaghetti plot of BIAC SDS. Each line represents the growth trajectory of one girl in the sample from five to 20 years of age.

## 4.4.3. Conditional references

Correlation matrices for shoulder breadth and pelvic dimensions of the longitudinal dataset are presented in Tables 4.3 to 4.6. Conditional references require that each individual included in the calculation must have been measured at every age point in the reference chart. For example, to be included in the conditional reference matrix calculation a girl must have been measured at six years old, eight years old, 10 years old, 12 years old etc. The longitudinal data sample was reduced to 15 girls who had been measured every two years from the age of eight to 18. This reduction in sample size suggests that the correlations shown in the tables below are low in power and may therefore not give a wholly representative view of pelvic dimension and shoulder breadth tracking as girls develop. The strongest correlation is between 14 and 12 years of age for shoulder breadth tracking followed by 18 and 10 years age. This suggests that shoulder breadth in early adulthood can be more readily predicted at the beginning of puberty and between 12 and 14 years of age. BIIB at age 16 was most strongly predicted from age 12 and overall BIIB in early and mid adolescence was poorly predicted from age 8. Table 4.5. shows that INML at age 18 can be predicted more readily predicted at age 8 and at age 16, though poorly predicted throughout puberty. BIAC tracking follows a similar pattern to INML.

Age (yrs)							
8	1						
10	0.09	1					
12	0.59	0.34	1				
14	0.29	0.32	0.78	1			
16	0.21	0.28	0.26	0.29	1		
18	0.21	0.62	0.17	0	0.03	1	
Age (yrs)	8	10	12	14	16	18	

Table 4.3. Correlation matrix for shoulder breadth SDS at six ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age.

Age (yrs)							
8	1						
10	0.17	1					
12	0.48	0.42	1				
14	0.13	0.07	0.43	1			
16	0.16	0.42	0.67	0.07	1		
18	0.28	0.01	0.18	0.14	0.27	1	
Age	8	10	12	14	16	18	

Table 4.4. Correlation matrix for BIIB SDS at six ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age.

Table 4.5. Correlation matrix for INML SDS at six different ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age.

Age (yrs)							
8	1						
10	0.28	1					
12	0.55	0.72	1				
14	0.12	0.09	0.33	1			
16	0.67	0.41	0.45	0.25	1		
18	0.58	0.40	0.31	0.20	0.75	1	
Age	8	10	12	14	16	18	

Table 4.6. Correlation matrix for BIAC SDS at six ages from childhood to early adulthood, based on data from n=15 girls seen on all six measurement sessions. The horizontal axis shows mean age.

Age (yrs)							
8	1						
10	0.45	1					
12	0.00	0.11	1				
14	0.10	0.19	0.24	1			
16	0.14	0.13	0.10	0.25	1		
18	0.44	0.32	0.35	0.09	0.28	1	
Age	8	10	12	14	16	18	

## 4.4.4. Cross-sectional dataset used in this chapter

The numbers of individuals per age group in the cross-sectional dataset are displayed in Table 4.7. Results of the Shapiro-Wilk's test for normality are presented in Table 4.8. These results show that fat mass, bi-iliac breadth, hip circumference and mean suprailiac thickness are not normally distributed. It is not uncommon for size and shape data collected from growing children not to be normally distributed as the size of different dimensions in children will increase at different rates (Cole, 1993). The SDS in the cross-sectional dataset used for this chapter were calculated using the LMS method (see Section 4.3. for more details) which reduces skewness of data distribution and shapes the data distribution into a more normal distribution (Cole, 1993) and using recent UK child growth standards (Wells et al., 2012b). Descriptive statistics of the cross-sectional dataset are presented in Table 4.9. The cross-sectional subsample was reduced in this analysis due to the removal of physically impossible outliers (n=203). The results in Table 4.9. represent the sample sizes used for the multivariate statistical analyses.

Year of age	Sample size				
4	6				
5	12				
6	12				
7	9				
8	10				
9	8				
10	15				
11	17				
12	16				
13	31				
14	18				
15	15				
16	4				
17	11				
18	9				
19	20				
20	9				
21	7				
22	22				

Table 4. 7. Sample size in the cross-sectional dataset organised by age group prior to removal of outliers.

	Shapiro-Wilk's	df	p-
	statistic		values
Height SDS	0.988	169	0.158
Weight SDS	0.994	169	0.698
BMI SDS	0.993	169	0.551
Fat mass SDS	0.984	169	0.047
Lean mass SDS	0.996	169	0.947
Shoulder breadth SDS	0.992	169	0.523
Bi-iliac breadth SDS	0.980	169	0.013
Medio-lateral inlet breadth SDS	0.995	169	0.862
Bi-acetabular breadth SDS	0.996	169	0.946
Mid-upper arm circumference SDS	0.990	169	0.270
Head circumference SDS	0.992	169	0.479
Waist circumference SDS	0.988	169	0.159
Hip circumference SDS	0.957	169	0.000
Thigh circumference SDS	0.986	169	0.087
Calf circumference SDS	0.985	169	0.072
Mean biceps thickness SDS	0.986	169	0.100
Mean triceps thickness SDS	0.993	169	0.585
Mean subscapular thickness SDS	0.985	169	0.071
Mean suprailiac thickness SDS	0.975	169	0.004
* Values are significant at <0.0			

Table 4. 8. Results of the Shapiro-Wilk's test for normality of the cross-sectional dataset used in this chapter.

Table 4. 9. Descriptiv	e statistics	of the	cross-sectional	dataset usec	l in this chap	pter.
------------------------	--------------	--------	-----------------	--------------	----------------	-------

	N	Min.	Max.	Mean	Std. Deviation
Age (yrs)	203	4.20	21.90	13.10	4.73
Weight (kg)	203	14.16	90.75	46.89	17.25
Height (cm)	203	100.90	181.70	149.91	20.09
BMI	203	13.18	34.54	20.00	3.92
Fat mass (kg)	191	2.33	39.96	13.83	7.86
Lean mass (kg)	191	11.59	61.91	33.90	10.58
Shoulder breadth (cm)	181	21.78	41.09	32.47	4.54
Bi-iliac breadth (cm)	184	14.09	29.39	22.29	3.44
Medio-lateral inlet breadth (cm)	185	6.53	14.51	10.60	2.00
Bi-acetabular breadth (cm)	185	7.70	15.98	11.32	2.06
Mid-upper arm circumference (cm)	201	15.00	40.20	24.68	4.60
Head circumference (cm)	202	48.50	61.00	54.48	2.28
Waist circumference (cm)	202	44.10	93.80	66.84	9.97
Hip circumference (cm)	202	51.90	117.80	84.34	15.24
Thigh circumference (cm)	199	29.50	102.60	47.99	9.41
Calf circumference (cm)	201	20.60	44.80	31.57	5.07
Mean biceps thickness (mm)	194	3.07	23.73	9.85	4.54
Mean triceps thickness mm	195	5.87	33.07	15.34	5.63
Mean subscapular thickness (mm)	196	3.60	35.67	11.25	6.19
Mean suprailiac thickness (mm)	189	3.80	39.00	16.54	9.22
Tanner development stage	161	1	5	3.50	1.46
Age at menarche (months)	98	108.00	196.00	151.03	16.30
Maternal weight (kg)	186	41.28	127.00	69.23	14.98
Maternal height (cm)	192	151.80	182.88	164.45	6.48
Paternal weight (kg)	177	52.60	143.18	82.84	14.28
Paternal height (cm)	189	154.94	203.20	177.57	8.81
Birthweight (grams)	198	2010	4860	3465.70	480.24

## 4.4.5. Multivariate statistics

Multiple linear regression analyses were used to examine potential associations between birthweight, height, head circumference, parental body size, markers of puberty (age-at-menarche, measures of adiposity and Tanner development stage) and pelvic dimensions in the cross-sectional dataset in the sample used for this project. Scatterplots of height, head circumference, birthweight, parental height and weight, age at first menses, waist circumference, thigh circumference and shoulder breadth, BIIB, INML and BIAC are presented in Figures 4.10 to 4.45 and 4.50 to 4.53. Birth weight, height, maternal weight and height, head, hip and thigh circumference show positive linear relationships with shoulder breadth and pelvic dimensions. Boxplots of Tanner development stage, shoulder breadth and pelvic dimensions are shown in Figures 4.46 to 4.49. and show a similar range of shoulder breadth and pelvic dimensions in each Tanner development stage.



Figure 4.10. Scatterplot of shoulder breadth SDS regressed on birthweight.



Figure 4.11. Scatterplot of bi-iliac breadth SDS regressed on birthweight.



Figure 4.12. Scatterplot of medio-lateral inlet breadth SDS regressed on birthweight.



Figure 4.13. Scatterplot of bi-acetabular breadth SDS regressed on birthweight.



Figure 4.14. Scatterplot of shoulder breadth SDS regressed on height SDS.



Figure 4.15. Scatterplot of bi-iliac breadth SDS regressed on height SDS.



Figure 4.16. Scatterplot of medio-lateral inlet breadth SDS regressed on height SDS.



Figure 4.17. Scatterplot of bi-acetabular breadth SDS egressed on height SDS.



Figure 4.18. Scatterplot of shoulder breadth SDS regressed on head circumference SDS.



Figure 4.19. Scatterplot of bi-iliac breadth SDS regressed on head circumference SDS.



Figure 4.20. Scatterplot of medio-lateral inlet breadth SDS regressed on head circumference SDS.



Figure 4.21. Scatterplot of bi-acetabular breadth SDS regressed on head circumference SDS.



Figure 4.22. Scatterplot of shoulder breadth SDS regressed on maternal weight.



Figure 4.23. Scatterplot of bi-iliac breadth SDS regressed on maternal weight.



Figure 4.24. Scatterplot of medio-lateral inlet breadth SDS regressed on maternal weight.



Figure 4.25. Scatterplot of bi-acetabular breadth SDS regressed on maternal weight.



Figure 4.26. Scatterplot of shoulder breadth SDS regressed on maternal height.



Figure 4.27. Scatterplot of bi-iliac breadth SDS regressed on maternal height.



Figure 4.28. Scatterplot of medio-lateral inlet breadth SDS regressed on maternal height.



Figure 4.29. Scatterplot of bi-acetabular breadth SDS regressed on maternal height.



Figure 4.30. Scatterplot of shoulder breadth SDS regressed on paternal weight.



Figure 4.31. Scatterplot of bi-iliac breadth SDS regressed on paternal weight.



Figure 4.32. Scatterplot of medio-lateral inlet breadth SDS regressed on paternal weight.



Figure 4.33. Scatterplot of bi-acetabular breadth SDS regressed on paternal weight.



Figure 4.34. Scatterplot of shoulder breadth SDS regressed on paternal height.



Figure 4.35. Scatterplot of bi-iliac breadth SDS regressed on paternal height.



Figure 4.36. Scatterplot of medio-lateral inlet breadth SDS regressed on paternal height.


Figure 4.37. Scatterplot of bi-acetabular breadth SDS regressed on paternal height.



Figure 4.38. Scatterplot of shoulder breadth SDS regressed on hip circumference SDS.



Figure 4.39. Scatterplot of bi-iliac breadth SDS regressed on hip circumference SDS.



Figure 4.40. Scatterplot of medio-lateral inlet breadth SDS regressed on hip circumference SDS.



Figure 4.41. Scatterplot of bi-acetabular breadth SDS regressed on hip circumference SDS.



Figure 4.42. Scatterplot of shoulder breadth SDS regressed on thigh circumference SDS.



Figure 4.43. Scatterplot of bi-iliac breadth SDS regressed on thigh circumference SDS.



Figure 4.44. Scatterplot of medio-lateral inlet breadth SDS regressed on thigh circumference SDS.



Figure 4.45. Scatterplot of bi-acetabular breadth SDS regressed on thigh circumference SDS.



Figure 4.46. Boxplots of Tanner development stage and shoulder breadth SDS. Tanner development stages are 1) pre-adolescent, 2) breast bud stage, 3) further enlargement of the breast and areola, 4) projection of the areola and 5) mature stage. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 4.47. Boxplots of Tanner development stage and bi-iliac breadth SDS. Tanner development stages are 1) pre-adolescent, 2) breast bud stage, 3) further enlargement of the breast and areola, 4) projection of the areola and 5) mature stage. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 4.48. Boxplots of Tanner development stage and medio-lateral inlet breadth SDS. Tanner development stages are 1) preadolescent, 2) breast bud stage, 3) further enlargement of the beast and areola, 4) projection of the areola and 5) mature stage. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 4.49. Boxplots of Tanner development stage and medio-lateral inlet breadth SDS. Tanner development stages are 1) preadolescent, 2) breast bud stage, 3) further enlargement of the breast and areola, 4) projection of the areola and 5) mature stage. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 4.50. Scatterplot of shoulder breadth SDS regressed on age at menarche.



Figure 4.51. Scatterplot of bi-iliac breadth SDS regressed on age at menarche.



Figure 4.52. Scatterplot of medio-lateral inlet breadth SDS regressed on age at menarche.



Figure 4.53. Scatterplot of bi-acetabular breadth SDS regressed on age at menarche.

According to the scatterplots birth weight, height, maternal height and weight head, hip and thigh circumferences and Tanner development stage showed clear linear relationships were entered in to a multiple linear regression analysis. Results of the multiple linear regression analyses, as well as Durbin-Watson statistics and measures of multicollinearity are presented in Tables 4.10 to 4.13. Normality of residuals was checked using histograms and normal P-P plots to examine the homoscedasticity of the regression. The histograms of standardized residuals for shoulder breadth, INML and BIAC were approximately normally distributed, whilst the histogram for BIIB was slightly skewed to the right. The P-P plot of standardized residuals for shoulder breadth and all pelvic dimensions showed residuals plotting tightly on the line of identity, which suggests that variance in these data is normal.

The biological variables entered in to the multiple linear regression analysis explained 52% of variation in shoulder breadth. Shoulder breadth associated significantly with height and thigh circumference. The VIF values for all variables fell between 1 and 10, suggesting that multicollinearity is not an issue for this analysis. The Durbin-Watson statistic for shoulder breadth is 2.02, which is not below one or over three and confirms independence of observations for these analyses. The same biological variables explained 28% of variation in BIIB and height and thigh circumference associated significantly with BIIB. Multicollinearity was not an issue for the multiple linear regression analyses of BIIB and the Durbin-Watson statistic (2.06) confirmed independence of observations for BIIB. INML associated significantly with height and Tanner development stage, with the biological variables entered in to the multiple linear regression explaining 23% of variation in INML. Similar to shoulder breadth and BIIB, multicollinearity factors was not an issue for INML linear regression analyses and independence of observations was confirmed by the Durbin-Watson statistic of 1.74. BIAC associated significantly with height and Tanner development stage, with height, head, hips and thigh circumference and Tanner development stage accounting for 24% of BIAC variation. VIF values showed no issue with multicollinearity and the Durbin-Watson statistics confirmed that observations were independent.

Table 4.10. Linear regression results for shoulder breadth, including model summary, multicollinearity measures and Durbin-Watson statistics (n=175).

	β	<b>P</b> *	VIF	R	R <sup>2</sup>	Adjusted R <sup>2</sup>	Durbin- Watson
Birth weight	0.00	0.191	1.21				
Maternal weight	0.00	0.141	1.24				
Maternal height	-0.00	0.706	1.48				
Height	0.52	0.000	1.95				
Head circumference	0.07	0.372	1.33				
Hip circumference	-0.03	0.836	6.44				
Thigh circumference	0.33	0.028	5.03				
Tanner development stage	0.03	0.387	1.12				
				0.74	0.55	0.52	2.02

\* Values are significant at

<0.05

Table 4.11. Linear regression results for BIIB, including model summary, multicollinearity measures and Durbin-Watson statistics (n=178).

	β	<b>P</b> *	VIF	R	R <sup>2</sup>	Adjusted R <sup>2</sup>	Durbin- Watson
Birth weight	0.00	0.171	1.21				
Maternal weight	0.00	0.259	1.27				
Maternal height	0.00	0.768	1.43				
Height	0.33	0.001	1.86				
Head circumference	0.04	0.645	1.35				
Hip circumference	-0.15	0.487	6.60				
Thigh circumference	0.34	0.040	5.53				
Tanner development stage	0.06	0.171	1.21				
				0.57	0.33	0.28	2.06

\* Values are significant at

<0.05

Table 4.12. Linear regression results for INML, including model summary, multicollinearity measures and Durbin-Watson statistics (n=179).

	β	<b>P</b> *	VIF	R	R <sup>2</sup>	Adjusted R <sup>2</sup>	Durbin- Watson
Birth weight	0.00	0.496	1.21				
Maternal weight	-0.00	0.618	1.26				
Maternal height	0.00	0.670	1.45				
Height	0.29	0.006	1.88				
Head circumference	-0.05	0.601	1.36				
Hip circumference	0.42	0.071	6.67				
Thigh circumference	-0.12	0.471	5.38				
Tanner development stage	0.11	0.045	1.21				
				0.53	0.28	0.23	1.74

\* Values are significant at

Table 4.13. Linear regression results for BIAC, including model summary, multicollinearity measures and Durbin-Watson statistics (n=179).

	β	<b>P</b> *	VIF	R	R <sup>2</sup>	Adjusted R <sup>2</sup>	Durbin- Watson
Birth weight	0.00	0.933	1.21				
Maternal weight	-0.00	0.843	1.26				
Maternal height	-0.01	0.254	1.44				
Height	0.39	0.001	1.88				
Head circumference	0.12	0.213	1.36				
Hip circumference	0.01	0.946	6.67				
Thigh circumference	0.18	0.314	5.38				
Tanner development stage	0.13	0.024	1.12				
				0.54	0.29	0.24	1.78

\* Values are significant at <0.05

## 4.5. Discussion

#### 4.5.1. Childhood pelvic dimensions to adult pelvic dimensions

Using spaghetti plots to track the growth of shoulder breadth, BIIB, INML and BIAC in individual girls during growth demonstrates different patterns in development. According to the spaghetti plots of the longitudinal dataset used in this chapter, shoulder breadth and BIAC increase in approximately a linear direction however these dimensions cross standard deviations (above or below the mean) throughout childhood and adolescence in growing girls. BIIB and INML growth occurs in an sshape curve with comparatively less centile crossing than shoulder breadth and BIAC.

These patterns differ from those shown in Chapter 3, where the growth trajectories of BIAC and INML were twinned and those of BIIB and shoulder breadth were similar. These differences in visualising growth trajectories may occur due to differing samples and methods to visualize growth trajectories. Cross-sectional data was used to create growth curves in Chapter 3, whilst spaghetti plots in this project were created from longitudinal data. Cole (2012) notes that longitudinal growth curves show considerable centile crossing compared to cross-sectional growth curves and suggests that the usefulness of longitudinal curves have been overstated. It may be similarly imprudent to draw conclusions about genetic vs. environmental influences from the SDS spaghetti plots of the girls in this sample. It is also likely that centile crossing seen in shoulder breadth growth and BIAC is particularly sensitive

during puberty – individually variable growth velocities that have already been noted in height and weight during puberty (Tanner et al., 1966).

The spaghetti plots of the linear dimensions of INML and BIAC suggest that canal measurements of pelvic breadth may be less variable than measurements of body breadth prior to adulthood. By age 16 (the end of puberty) INML and BIAC was between 11cm and 14cm in all girls. At the same age BIIB was between 22cm and 28cm and shoulder breadth was between 33cm and 40cm. Moerman (1982) and Völgyi et al. (2010) found a delay in the growth of canal breadth compared to non-canal pelvic breadth in growing girls. In adults, bi-iliac breadth associates with latitude (Roseman and Auerbach, 2015). Kurki and Decrausaz (2016) found that multivariate canal shape variance was greater than non-canal pelvic shape variance in adults. In this study, BIIB also associated significantly with birthweight, which has been identified as an early-life indicator of adult body size (Lucas et al., 1999). The greater variation in body breadth (BIIB and shoulder breadth) compared to pelvic breadth in growing girls in this sample lends support to the notion of different timings (including a delay) for canal vs. non-canal breadth increase (Moerman 1982; Völgyi et al., 2010).

## 4.5.2. Timelines of body breadth growth vs. pelvic dimension growth

Multivariate statistical test results indicate differing associations between skeletal and soft tissue variables as well as suggesting different timelines for body breadth vs. pelvic breadth growth. Charts of growth velocity in Chapter 3 demonstrated that shoulder breadth growth velocity increases at 8 years of age before slowing during puberty. Unlike shoulder breadth, height *decreases* in growth velocity at approximately 8 years old. BIIB growth velocity slows around 10 years of age and throughout puberty, whilst INML and BIAC increase in growth velocity between 8 and 11 years old, followed by a reduction in growth velocity. Multivariate tests in this chapter showed significant relationships between shoulder breadth, all pelvic dimensions and height.

The strongest associations occurred between height, shoulder breadth and BIIB. These dimensions may be tied in similar growth pattern in establishing body frame size. Henneberg and Ulijaszek (2010) found that increased trunk size associated with greater fatness overall in healthy Australian women. In the current study, shoulder breadth and BIIB also associated significantly with thigh circumference, a localised measure of increasing adiposity for preparing the body for the energy loss in lactation. It is possible that as girls grow increasing body breadth is linked with preparation of localised adiposity for lactation. The association of height with all pelvic dimensions implies that as girls grow the link between increasing height and pelvic dimensions provides a basis for attaining an obstetrically-sufficient minimum of canal dimensions. INML and BIAC associated with Tanner development stages, suggesting that pelvic canal dimensions increase with specific indicators of pubertal development that can be viewed externally.

This can be examined alongside the results of the conditional references for shoulder breadth and pelvic dimensions. All pelvic dimensions were poorly predicted between the ages of 12 to 14 though adult pelvic dimensions were more readily predicted from early childhood. Growth velocity charts for the pelvic canal in Chapter 3 showed larger increases in growth velocity just prior to puberty. This large increase in growth velocity may create 'noise' in predicting pelvic dimensions throughout mid-puberty. With the onset of puberty localised measures of adiposity such as hip circumference and thigh circumference are increasing, which could suggest that during puberty prioritization shifts from increasing lean mass (i.e., shoulder breadth, height) to fat mass and specific areas of adiposity.

A delay in obstetrically-relevant pelvic growth matches the delay in biological and social preparation for reproduction that is unique to humans. The length of human puberty is distinctive as it allows growing boys and girls the time to fully develop the necessary social and energetic skills for raising children and living independently (Roff, 1992; Stearns, 1992; Charnov, 1993; Bogin, 1994, 1999b; a). There are evolutionarily relevant differences between boys and girls in the timing of growth spurts with the onset of puberty. Girls reach PHV earlier than boys and experience their growth spurt prior to becoming fertile (Bogin, 1994; Ellison, 1994), likely to prevent the issues that would arise with pregnancy and childbirth whilst girls are physically underdeveloped (Mace, 2000). Average age at first birth in a large range of natural fertility populations is 19 years of age (Ellison, 1994; Bogin, 1999b; Bogin and Smith, 2000; Mace, 2000; Ellison, 2001) which suggests a continued benefit for reduced pelvic growth velocity relative to other body tissues.

## 4.5.3. Parental weight and height does not associate with pelvic dimensions

Scatterplots of parental height, weight and daughter shoulder breadth and pelvic dimensions showed no distinct linear relationships. This is not to suggest that there is no relationship between parental and daughter body size overall. Working with the same sample Devakumar et al. (2016) found that paternal BMI related significantly to son's BMI but not to daughter's BMI, whilst maternal BMI associated with both daughter's and sons' BMI. It is possible that associations between daughter's dimensions and parental body size and shape may differ depending on the part of the body in question. Pomeroy et al. (2015) found paternal BMI associated with lower leg and lower arm length of neonates and suggested that this shows paternal contribution to maximizing offspring lean mass by increasing limb size instead of overall size, as this avoids increasing childbirth complications for mothers. Further multivariate analyses of daughter's anthropometrics and parental height and weight in the current study may illuminate further links. In addition, a study examining pelvic dimensions in parents and their children would clarify relationships between parental body size and shape and the pelvic dimensions of their daughters specifically.

## 4.5.4. Age at first menses does not associate with pelvic dimensions

Scatterplots of age at first menses, shoulder breadth and pelvic dimensions showed no clear linear relationships between these variables. In humans age-at-first menses coincides with an increase in fat mass that provides energy storage for lactation and pregnancy. The relationship between menarche and fat tissue is complex and dependent on a number of variables. Frisch and McArthur (1974) found that 17% body fat represents the minimum fat tissue for normal menarche, also demonstrating that a high percentage of fat tissue in girls (26-28%) could influence reproductive ability (Frisch, 1985). Simmons and Greulich, (1943) found that obesity in girls brought forward the age of menarche, whilst Cooper et al. (1996) found that

comparatively heavier girls at 7 years of age experienced menarche at an earlier age. Dunger et al. (2005) found that an overall small size at birth associated with earlier menarche. Pierce and Leon (2005) stated that it was challenging to identify increased fat mass in early childhood as the trigger for early menarche and Towne et al. (2005) found a heritability of age at menarche of 0.49. Ong et al. (2007) examined the relationship between mother's age at menarche, her adult body size and obesity risk, her children's obesity risks and their growth. Ong et al.'s (2007) study examined these variables amongst the mother's and children in the Avon Longitudinal Study of Parents and Children (ALSPAC) cohort, a prospective study recruited from all pregnancies in the three Bristol-based District Health Authorities with mothers who had expected delivery dates between April 1991 and December 1992. The ALSPAC cohort comprised approximately 14,000 live births (Ong et al. 2007). Ong and colleagues found that in mothers earlier age at menarche was associated with shorter adult stature, increased body weight and BMI. In contrast, amongst a mother's children, her earlier age at menarche predicted taller height, greater body weight, higher BMI and fat mass index. These results suggested that age-at-first menses was a transgenerational marker of rapid growth tempo, including rapid weight gain during infancy.

Potential links between menarche and pelvic breadth are unclear due to differences in pelvic measurements used in the already small number of studies examining this subject. Novotny et al. (2000) found that the horizontal distance between the outermost points of the greater trochanters could predict age-at-first menses in adult women. Völgyi et al. (2010) found that both BIIB and INML growth velocity peaked at approximately 1 year prior to menarche, though did not examine associations between pelvic measures, body size and shape and menarche. This line of questioning could be further explored in this current study by examining relationships between fat mass, localised adiposity and age first menses.

#### 4.6. Conclusion

Results from this study demonstrated that pelvic dimensions and shoulder breadth associate with different measurements of body size and shape. Ultimately,

the results of this study reinforce 1) the differing growth patterns of the canal components of the pelvis compared to the non-canal components of the pelvis and 2) the slower and longer growth pattern of the pelvic canal. I propose that the outcomes of this study demonstrate that the pelvic canal is differing "functional cluster" (Völgyi et al., 2010) in growth – that as a girl grows, she first grows in height, than in body breadth (including shoulder breadth and bi-iliac breadth) and finally in pelvic canal dimensions. Linear regression analyses showed BIIB and shoulder breadth associated with height and thigh circumference whilst canal measurements associated with height and external indicators of pubertal development. This suggests a transition in priority during growth from stature increase to breadth increase at the end of childhood and the beginning of puberty. Spaghetti plots showed that INML and BIAC were less variable in individual growth patterns than shoulder breath and BIIB. This reflects the slower pace of growth of the canal components of the pelvis compared to the non-canal components of the pelvis. The results from this study contribute to better understanding the biological relationships between body size and shape and pelvic dimensions to provide a context for variation in pubertal development and pelvic variation in living populations.

# 4.7. Associations between biological variables and pelvic canal breadth variables and associations between ecological variables and pelvic canal breadth variables

The results of this chapter demonstrate differing associations between body size and shape variables and pelvic canal breadth variables as girls grow. These results add to the results of Chapter Three, giving more depth to the understanding of pelvic canal breadth growth by adding an examination of the interactions between skeletal tissue and soft tissue changes as girl grow. However these results do not give any indication of associations between external ecological variables (such as biosocial variables including diet and education) and pelvic canal growth. Relationships between pelvic canal breadth and biosocial variables are tested in Chapter Five.

## CHAPTER FIVE:

#### The biosocial factors associated with female pelvic growth

#### 5.1. The role of Chapter Five in this thesis

This chapter tests the associations between biosocial variables (such as education and socioeconomic status) and pelvic canal breadth variables in the same dataset as Chapter Four. This chapter follows a similar structure as Chapter Four but tests associations between variables relating to the environment in which growing girls develop and the development of their bony pelves. The results of this chapter will be compared with the results of Chapter Four to determine the importance of ecological variables in the growth of the pelvic canal.

#### 5.2. Introduction

Human growth is affected by both genetic and environmental factors. Adult height is approximately 80% to 90% heritable (Silventoinen et al., 2003; Perola et al., 2007). Twin studies have determined that weight is approximately 67% heritable (Maes et al., 1997). Humans have been characterized as advantageously flexible in their biology, enabling the colonization of diverse environments throughout human evolutionary time. This plasticity has allowed humans to avoid committing to a genetic adaptation in favour of a assortment of plastic states based on environmental pressures (Wells and Stock, 2007; Pomeroy et al., 2012; Wells, 2016). Early life also plays a major role in setting a template for body size and shape in adulthood, affected by nutrition and disease. For example, limb length in growing children is affected by environmental stresses including altitude (Pomeroy et al., 2012), nutritional stress, disease load and socioeconomic stress (Boaz, 1912; Tanner, 1981a; Bogin et al., 2002, 2017).

The influence of both genetic and environmental factors on body size and shape during growth has prompted researchers to examine growth using a biosocial approach. Biosocial approaches explicitly acknowledge the links between humans as biological beings and the social and physical environments in which humans live (Dufour, 2006). This approach grew out of Livingston's (1958) landmark study which demonstrated the link between the spread of malaria, the sickle cell hemoglobin allele and the increase in agriculture in West Africa. Biosocial approaches are well-suited for analyses examining the health of living populations as accurate biological and environmental data are more readily accessible. Biosocial approaches to studies of the skeleton have examined the intersection of health and social status (Cucina and Tiesler, 2003) and the prevalence of violence and resulting skeletal evidence of trauma (Scott and Buckley, 2010). Wiley and Cullin (2016) note that there is extensive variation in the definition of biosocial analyses amongst anthropological scholars and that this may affect the prevalence of this approach in skeletal analyses.

Previous studies have determined that there are multiple environmental factors that can impact on body size and shape as a child grows. Some of the earliest examinations of growing children identified the manifestation of differences in body size and shape as well as markers of puberty dependent on socioeconomic status (SES) (Tanner, 1981a). Low SES amongst children growing up in 18<sup>th</sup> and 19<sup>th</sup> century England resulted in short stature, evidence of metabolic disease and lower cortical bone thickness than expected from dental ages (Lewis, 2002; Mays et al., 2009; Newman and Gowland, 2017). Nutritional and psychosocial stress during the Great Irish famine (1845-1852) resulted in a failure to initiate adolescence in some children (Geber, 2014). Stunting affected 13% of low SES girls growing up in Eastern Kentucky and 33% of children in this high-poverty community were overweight (Crooks, 1999).

Micronutrient deficiencies can also lead to marked impacts on the body size and shape of growing children. Calcium, phosphorus, zinc and magnesium are the principal minerals in the formation of bone (Prentice, 2003). These micronutrients are most often found in animal proteins (including animal meat and milk), legumes, pulses and some fermented cereals (WHO, 2006). In bones, calcium exists in its mineral form of hydroxyapatite and impacts bone strength through bone mass (Zhu and Prince, 2012). Calcium intake in the form of milk seems to stimulate circulating levels of insulin-like growth factor (IGF)-I which in turn increases the velocity of linear growth in well-nourished children (Hoppe et al., 2006). A lack of adequate dietary

206

calcium can result in small stature and poor bone health (Black et al., 2002). However it is important to note that children who are experiencing growth faltering likely are deficient in other minerals and micronutrients than calcium alone (Welch and Weaver, 2005). In the UK adolescents have a recommend daily dietary calcium intake of 800 milligrams for girls and 1000 milligrams for boys (The British Dietetic Association, 2017). Vitamin D is involved in calcium balance and cell differentiation (Dror and Allen, 2014), and increases intestinal calcium absorption from 30% to 60 to 80% during periods of growth (Holick, 2004). Whilst vitamin D can be found in animal milk, the major source of vitamin D is the synthesis of sunlight using cholesterol (Holick, 2004), accounting for the high prevalence of vitamin D deficiency amongst immigrant populations living in locations with comparatively fewer sunlight hours (Robinson et al., 2006). A deficiency in vitamin D results in rickets; a failure in mineral deposition in bone tissue, leaving bone tissue to remain soft and flexible, giving characteristically bowed inwards or outwards appearance to lower limbs (White and Folkens, 2005). High SES children growing up in 16<sup>th</sup> to 18<sup>th</sup> century France (Schattmann et al., 2016) and Renaissance Italy (Giuffra et al., 2015) suffered from rickets as a result of spending much of their childhood indoors (to avoid developing darker skin tones from spending time in the sun as ligher skin tones had higher cultural value), living in cramped conditions and being swaddled as infants. Comparatively only some children living in urban communities in 19<sup>th</sup> century Birmingham, England showed signs of rickets (Mays et al., 2006a).

Children's bodies respond to physical activity, changing hormone levels and micronutrient levels during growth. Childhood and adolescence are key periods in the accumulation of bone mass – up to 50% of total body bone mass is achieved during these growth phases (Perez-Lopez et al., 2010). Apart from genetic and nutritional influences, bone mass is accumulated as a result of physical activity (Slemenda et al., 1991). Physical activity increases muscle size and strength which then exerts higher tensile forces on the bone to which the muscle is attached (Rauch et al., 2004). The effectiveness of the rate, type and intensity of physical activity depends on the area of the skeleton as reaction forces change throughout the skeleton (Vlachopoulos et al., 2016). Physical activity during growth (depending on intensity) also reduces fat mass in early adolescence (Ness et al., 2007; Riddoch et al., 2009).

Bone mass in growing girls is also affected by hormonal status. Research interest in bone mass alongside changing hormonal status in women has increased with increasing prevalence of osteoporosis in women (Cosman et al., 2014). In a recent study, women who had no bone fractures were significantly more likely to have taken oral contraceptives (Dombrowski et al., 2017) suggesting that the addition of specific circulating hormones throughout growth has an impact on the skeleton later in life. Examining the effects of oral contraceptive use on body composition and bone mass in healthy growing girls Lloyd et al. (2000) found that oral contraceptives had no effect on peak bone mass. Lloyd et al. (2004) also found that oral contraceptive use during adolescence did not correlate with bone or body composition measurements.

The importance of biosocial factors on human growth affects more than one generation. Clarifying the association between biosocial factors and bony pelvic throughout growth will aid in identifying components that could ultimately contribute to difficult childbirth in adulthood. Wells et al. (2012) have suggested that ecological factors such a nutrition and disease in early life could impact on pelvic size and shape in adulthood, highlighting the potential for pelvic sensitivity to growth arrests not unlike other parts of the skeleton. Wells' (2017) review of the contemporary obstetric dilemma points to the dual burden of malnutrition as a cause of increasing childbirth difficulties. The dual burden of malnutrition refers to the co-occurrence of stunting and obesity in a population as a result of poverty. Wells (2017) identifies this and social gender inequality as major factors in maternal growth, neonatal growth and the relationship between them potentially leading to cephalopelvic disproportion. Results from Chapter Four demonstrated that pelvic dimensions and shoulder breadth associate with height and localised measures of adiposity, suggesting that nutritional effects on these tissues may also associate with the growth of pelvic dimensions. The use of oral contraceptives may further impact on pelvic breadth due to alterations in changing hormone levels during puberty in growing girls. The results from this study will contextualise pelvic dimension growth during growth within a biosocial context that includes a consideration of social and biological factors on human growth.

The following hypotheses will be made in response to the question: Do pelvic dimensions associate with oral contraceptive use, physical activity levels, calcium intake, SES or parental education?

 $H_{01}$ : Pelvic dimensions do not associate with oral contraceptive use in growing girls as oral contraceptive use during adolescence does not correlate with body composition (Lloyd et al. 2004).

 $H_{A1}$ : Pelvic dimensions associate with oral contraceptive use in growing girls as oral contraceptive use as oral contraceptive use during adolescence is associated with weight gain (Risser et al., 1999).

H<sub>02</sub>: Pelvic dimensions do not associate with physical activity levels in growing girls.

 $H_{A2}$ : Pelvic dimensions associate with physical activity levels indirectly – fat mass and adiposity is affected by physical activity in growing girls (Ness et al., 2007) which may in turn affect skeletal tissue in the pelvis during growth.

H<sub>03</sub>: Pelvic dimensions do not associate with calcium intake in growing girls.

 $H_{A3}$ : Pelvic dimensions associate with calcium intake growing girls as calcium intake increases the likelihood of early menarche (Wiley, 2011) which alters the rate of growth of pelvic dimensions.

H<sub>04</sub>: Pelvic dimensions do not associate with SES in growing girls.

 $H_{A4}$ : Pelvic dimensions associate with SES in growing girls as previous studies have found differences in prevalence of pelvic organ prolapse amongst women in different socioeconomic status groups (Woodman et al., 2006) and populations (Sze et al., 1999; Baragi et al., 2002; Rizk et al., 2004).

H<sub>05</sub>: Pelvic dimensions do no associate with parental education in growing girls.

 $H_{A5}$ : Pelvic dimensions associate with maternal education in growing girls as women with fewer educational qualifications have been reported to have the highest risk for caesarean sections (Tollånes et al., 2007).

## 5.3. Materials and methods

## 5.3.1. Study sample

The sample for this study (f=286) was collected as a reference sample at the Great Ormond Street Hospital in London, UK between the years 2001 and 2016 by Mary Fewtrell and colleagues. This reference sample contains both longitudinal and cross-sectional datasets. Further details on the sample are available in Chapter Three. The cross-sectional dataset was used for all analyses in the current study (n=232).

## Body size and shape data collection

Full details of the fat and lean mass data collection are available in Chapter Three. The anthropometric data collected for the reference sample are included in Table 4.1 in Chapter Four.

## Physical development data collection

Details on physical development data collection are available in Chapter Four.

## Lifestyle data collection

Each participant in the Great Ormond Street Hospital sample was given a questionnaire that included lifestyle questions directed to the subject and to the parents of the subject. Participants had to fill in this section of the questionnaire for each visit, including return visits for subjects participating in longitudinal data collection. In the participant's questions on physical activity, participants were asked to tally the number of hours and type of physical activity (bicycle riding, swimming, running etc.) they engaged in per week, including hours of physical education they received at school and hours of sedentary behaviour. The participant's parents were asked to tally the total number of hours their child spent in vigorous activity per week

and to rate their child's level of activity to their peers, using a 1-5 scale (1=much less than peers, 2 = less, 3 = same, 4 = more, 5= much more). Each participant (with the aid of their parent present at the visit) was asked to complete a food frequency questionnaire (Nelson et al., 1988) in order to estimate the participants daily calcium intake. The use of a food questionnaire in this manner is estimated to identify 82% of daily calcium intake (Fewtrell et al., 2009) The questionnaire used to collect lifestyle data is available in Appendix Three.

# Socioeconomic data collection

Socioeconomic data were collected via questionnaire on study participants and on both parents of the participant. Participants were asked to provide the number of rooms in their home, whether the home was rented or owned, the marital status of their mothers, their ethnic origin and the number of children living in the home. Participants were further asked to provide their parents birth dates, number of months they have been employed in the past year as well as their highest educational attainment, qualification and occupation, coded from 1 to 4/5 (see Table 5.1.). Participants were asked to identify the primary earner for their family and note the social code of the primary earner using the Standard Occupational Classification published by the National Office of Statistics (National Office of Statistics, 2000). The structure of the participant's family was classed using a social code modified from the Standard Occupational Classification (see Table 5.2.).

Table 5.1. Educational attainment code of participant's parents.

## Value Educational attainment

- 1 No education qualification
- 2 <3 CSE's or GSCE's below C grade
- 3 >3 CSE's or any O levels or GCSE grade A-C
- 4 A levels, ONC/OND/BEC/TEC, SCE Highers, NVQ level 3
- 5 Degree/HND/HNC professional training (including SRN/RGN/RM/RHV), NVQ levels 4/5, BEC/TEC Higher

Table 5. 2. Social code of participants parents, modified from Standard Occupational Classification, published by the National Office of Statistics (National Office of Statistics, 2000).

Value	Social code
1	Managers and senior officials
2	Professional occupations
3	Associate professional and technical occupations
4	Skilled trades occupations
5	Personal service occupations
6	Single parent mother unsupported and not working
7	Mother supported but partner and self never employed
8	Adopted/fostered child

# Osteological data collection

Linear measurements of the skeleton were collected from DXA scans of the entirety of Fewtrell and colleague's reference study sample (see Chapter Three for more details on this sample) by the author of this thesis. More details on the osteological measurements are available in Chapter Three.

# 5.4. Analytical methods

Pelvic dimensions, shoulder breadth and anthropometrics were converted into SDS to account for age-related variation. For all analyses outliers were identified using the formula 1.5IQR, which identifies values that are 1.5× the interguartile range away from the sample median. Any outliers that were physically impossible and likely due to data entry error (for example, the height of a healthy 16 year old incorrectly entered as 95cm) were removed. All data were tested for normality using Shapiro-Wilks test.

# SDS values used in the multivariate analyses

Data from the cross-sectional dataset were used in multivariate analyses. Specifically the SDS values of the variables representing pelvic breadth, body size and shape and pubertal development were used in the multivariate analyses in this chapter. The benefits of using SDS instead of raw data to analyse data collected from growing children are detailed in Chapter Three. The SDS values of shoulder breadth and pelvic breadth data in this chapter, similar to the same cross-sectional dataset used to create the growth curves in Chapter Three, were created using LMS Chartmaker (Pan and Cole, 2010) by the author of this thesis. LMS Chartmaker

includes growth references for healthy children living in the UK during the 1990s to create SDS values from raw data on body dimensions. The SDS values for all other body size and shape variables (i.e. head circumference, thigh circumference) and measures of adiposity (i.e. skinfold thickness) were calculated using more recent growth standards created by (Wells et al., 2012b), which were also created from data collected from healthy children living in the UK. Fewtrell and colleagues calculated the SDS values for the body size and shape variables and measures of adiposity as part of their reference sample (more details on this are available in Chapter Three).

## Univariate statistics

SDS values were used in a one-way ANOVA (analysis of variance) to examine mean differences in shoulder breadth and pelvic breadth dimensions between girls in the sample as a result of social code, maternal education and paternal. Tukey's range test (Tukey, 1949) was performed alongside the one-way ANOVA to define statistically significant mean differences between social code, maternal education, paternal education A student's t-test was used to examine mean differences in shoulder breadth and pelvic breadth in the sample as a result of oral contraceptive use. One-way ANOVA tests are best for testing samples that have a minimum of three or more cases (such as social code in this chapter, which has eight caregories). A student's t-test is better suited than an ANOVA to address two cases (Student, 1908) (that is, oral contraception being used, or not being used).

#### Multivariate statistics

SDS values were used in a multiple linear regression analysis to examine possible associations between pelvic dimensions, shoulder breadth and biosocial variables (father's education, mother's education, social code, hours of vigorous activity per week, average hours of activity per week, oral contraceptive use and calcium intake). Independence of observations was tested using the Durbin-Watson statistic (Durbin and Watson, 1951). Homoscedasticity of the data were examined visually from plots of standardized residuals plotted against pelvic and body breadth measurement SDS. The data's multicollinearity was examined using Tolerance and Variance Inflation Factors (VIF). The normality of residuals was checked using histograms and normal probability-probability (P-P) plots based on standardized residuals. Multiple linear regression analyses have been used successfully to examine the association between biosocial factors, body breadth and body composition (Duran-Tauleria et al., 1995; Freeman et al., 1995; Van Hooff et al., 1998; Wiley, 2012; Parsons et al., 2005; Wiley, 2005, 2011; Ness et al., 2007; Berenson and Rahman, 2009; Kennedy et al., 2012; Pomeroy et al., 2015a). All statistical analyses were conducted in SPSS for Macintosh (IBM Statistical Package for the Social Sciences, Version 20.0).

## 5.5. Results

## 5.5.1. Cross-sectional dataset used in this chapter

The numbers of individuals per age group in the cross-sectional dataset are displayed in Table 5.3. Results of the Shapiro-Wilk's test for normality are presented in Table 5.4. These results show fat mass, bi-iliac breadth, hip circumference and mean suprailiac thickness are not normally distributed. It is not uncommon for size and shape data collected from growing children not to be normally distributed as the size of different dimensions in children will increase at different rates (Cole, 1993). The SDS in the cross-sectional dataset used for this chapter were calculated using the LMS method (see Section 4.3. for more details) which reduces skewness of data distribution and shapes the data distribution into a more normal distribution (Cole, 1993) and using recent UK child growth standards (Wells et al., 2012b). Descriptive statistics of the cross-sectional dataset are presented in Table 5.5. The cross-sectional subsample was reduced in this analysis due to the removal of physically impossible outliers (n=204).

Year of age	Sample size
4	6
5	12
6	12
7	9
8	10
9	8
10	15
11	17
12	16
13	31
14	18
15	15
16	4
17	11
18	9
19	20
20	9
21	7
22	22

Table 5. 3. Sample size in the cross-sectional dataset organized by age group prior to the removal of outliers.

Table 5. 4. Results of the Shapiro-Wilk's test for normality of the cross-sectional dataset used in this chapter.

	Shapiro-Wilk's statistic	df	p-values		
Height SDS	0.988	169	0.158		
Weight SDS	0.994	169	0.698		
BMI SDS	0.993	169	0.551		
Fat mass SDS	0.984	169	0.047		
Lean mass SDS	0.996	169	0.947		
Shoulder breadth SDS	0.992	169	0.523		
Bi-iliac breadth SDS	0.980	169	0.013		
Medio-lateral inlet breadth SDS	0.995	169	0.862		
Bi-acetabular breadth SDS	0.996	169	0.946		
Mid-upper arm circumference SDS	0.990	169	0.270		
Head circumference SDS	0.992	169	0.479		
Waist circumference SDS	0.988	169	0.159		
Hip circumference SDS	0.957	169	0.000		
Thigh circumference SDS	0.986	169	0.087		
Calf circumference SDS	0.985	169	0.072		
Mean biceps thickness SDS	0.986	169	0.100		
Mean triceps thickness SDS	0.993	169	0.585		
Mean subscapular thickness SDS	0.985	169	0.071		
Mean suprailiac thickness SDS	0.975	169	0.004		
* Values are significant at <0.05					
	N	Min.	Max.	Mean	Std. Deviation
----------------------------------	-----	--------	--------	--------	----------------
Age (yrs)	203	4.20	21.90	13.27	4.69
Weight (kg)	203	14.16	90.75	47.79	17.52
Height (cm)	203	100.90	181.70	150.81	19.80
BMI	203	13.18	34.54	20.16	4.10
Fat mass (kg)	191	2.33	39.96	14.20	8.09
Lean mass (kg)	191	11.59	61.91	34.48	10.49
Shoulder breadth (cm)	179	21.78	41.09	32.69	4.51
Bi-iliac breadth (cm)	182	14.09	28.57	22.46	3.40
Medio-lateral inlet breadth (cm)	183	6.53	14.51	10.71	1.99
Bi-acetabular breadth (cm)	183	7.70	15.98	11.40	2.03
Head circumference (cm)	202	48.50	61.00	54.59	2.27
Waist circumference (cm)	202	44.10	96.40	67.23	10.27
Hip circumference (cm)	202	51.90	117.80	85.11	15.45
Thigh circumference (cm)	199	29.50	102.60	48.38	9.52
Calf circumference (cm)	201	20.60	44.80	31.81	5.12
Mid-upper arm circumference (cm)	201	15.00	40.20	24.85	4.67
Mean biceps thickness (mm)	195	3.07	23.73	9.80	4.49
Mean triceps thickness (mm)	196	5.87	33.07	15.44	5.63
Mean subscapular thickness (mm)	196	3.60	35.67	11.49	6.53
Mean suprailiac thickness (mm)	189	3.80	39.00	16.78	9.23
Calcium intake (milligrams)	118	88	2449	800.84	374.93
Horus of vigorous activity	197	0	40	6.09	5.35
Social code	199	1	8	2.67	1.46
Maternal education	194	1	5	3.99	1.11
Paternal education	185	1	9	4.01	1.40
	I	I	I	I	I

Table 5. 5. Descriptive statistics of the cross-sectional dataset used in this chapter.

### 5.5.2. Univariate analyses - boxplots

Boxplots of social code, maternal education, paternal education, oral contraceptive use, and pelvic dimensions SDS and shoulder breadth SDS are presented in Figures 5.6. to 5.17 and Figures 5.22 to 5.24. There is a noticeable reduction in range of shoulder breadth and BIAC with increasing social code and reduction in range for shoulder breadth and all pelvic dimensions for girls from single parent homes (code seven). It should be noted that this may also reflect a smaller number of girls who fall in to this social code. Mean shoulder breadth and pelvic dimensions are largest in social codes one to three, which includes girls who have grown up with parents who managers, senior officials and professionals. Shoulder breadth and pelvic dimensions display a similar pattern with all categories of maternal education: maternal education code three has a higher mean measurement than maternal education code two, with a comparative reduction in maternal education code

five. The comparatively small range in maternal education code one is likely also affected by a smaller group of girls with mothers who are in code one for maternal education (no educational qualification). This same pattern holds for all pelvic dimensions and shoulder breadth for paternal education codes. Mean shoulder breadth and bi-iliac breadth were larger amongst girls who were taking an oral contraceptive, however this may be affected by unequal number of girls who were using oral conception compared to those who were not.

# 5.5.3. Multivariate analyses - scatterplots

Scatterplots of calcium intake, shoulder breadth and pelvic dimensions SDS are presented in Figures 5.1. to 5.5. Shoulder breadth and pelvic dimensions SDS do not show a clear linear relationship with calcium intake. Scatterplots of shoulder breadth and pelvic dimensions SDS regressed on hours of vigorous physical activity per week are presented in Figures 5.18 to 5.20. There are no clear linear relationships between shoulder breadth SDS, pelvic dimensions SDS and hours of vigorous physical activity per week in the sample used in this chapter. As a result of these scatterplots no linear regression analyses were completed.



Figure 5.1. Scatterplot of shoulder breadth SDS regressed on calcium intake.



Figure 5.2. Scatterplot of bi-iliac breadth SDS regressed on calcium intake.



Figure 5.3. Scatterplot of medio-lateral inlet breadth SDS regressed on calcium intake.



Figure 5.4. Scatterplot of bi-acetabular breadth SDS regressed on calcium intake.



Figure 5.5. Boxplots of social code and shoulder breadth SDS. Social code categories are detailed in Table 5.2. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 5.6. Boxplots of social code and bi-iliac breadth SDS. Social code categories are detailed in Table 5.2. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 5.7. Boxplots of social code and medio-lateral inlet breadth SDS. Social code categories are detailed in Table 5.2. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category. The bottom edge of the data range in each category. The symbols marked \* represent outliers.



Figure 5.8. Boxplots of social code and bi-acetabular breadth SDS. Social code categories are detailed in Table 5.2. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 5.9. Boxplots of maternal education and shoulder breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category. The bottom edge of the data range in each category. The symbols marked \* represent outliers.



Figure 5.10. Boxplots of maternal education and bi-iliac breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category. The bottom edge of the data range in each category. The symbols marked \* represent outliers.



Figure 5.11. Boxplots of maternal education and medio-lateral inlet breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 5.12. Boxplots of maternal education and bi-acetabular breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 5.13. Boxplots of paternal education and shoulder breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category. The bottom edge of the data range in each category. The symbols marked \* represent outliers.



Figure 5.14. Boxplots of paternal education and bi-iliac breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category. The bottom edge of the data range in each category. The symbols marked \* represent outliers.



Figure 5.15. Boxplots of paternal education and medio-lateral inlet breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers.



Figure 5.16. Boxplots of paternal education and bi-acetabular breadth SDS. Educational attainment categories are detailed in Table 5.1. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category. The bottom edge of the data range in each category. The symbols marked \* represent outliers.



Figure 5.17. Scatterplot of shoulder breadth SDS regressed on hours of vigorous physical activity per week.



Figure 5.18. Scatterplot of bi-iliac breadth SDS regressed on hours of vigorous physical activity per week.



Figure 5.19. Scatterplot of medio-lateral pelvic breadth SDS regressed on hours of vigorous physical activity per week.



Figure 5.20. Scatterplot of bi-acetabular breadth SDS regressed on hours of vigorous physical activity per week.



Figure 5.21. Boxplots of oral contraceptive use and shoulder breadth SDS. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers. The boxplots represent data collected from girls who have begun to menstruate and would be eligible for oral contraceptive use.



Figure 5.22. Boxplots of oral contraceptive use and bi-iliac breadth SDS. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers. The boxplots represent data collected from girls who have begun to menstruate and would be eligible for oral contraceptive use.



Figure 5.23. Boxplots of oral contraceptive use and medio-lateral inlet breadth SDS. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers. The boxplots represent data collected from girls who have begun to menstruate and would be eligible for oral contraceptive use.



Figure 5.24. Boxplots of oral contraceptive use and bi-acetabular breadth SDS. The whiskers of the boxplot represent the minimum and maximum of the data range in each category, the top edge of the box represents the third quartile of the data range in each category. The bottom edge of the box represents the first quartile of the data range in each category and the black bar in the centre of the box represents the median of the data range in each category. The symbols marked \* represent outliers. The boxplots represent data collected from girls who have begun to menstruate and would be eligible for oral contraceptive use.

## 5.5.4. Univariate analyses

Results of the one-way ANOVA Tables 5.6 to 5.8. The results of the Tukey's range test for the one-way ANOVA tests are presented in Tables 5.9. to 5.11. The means of shoulder breadth and pelvic breadth dimensions for different social codes, levels of maternal and paternal education are not significantly different in the cross-sectional dataset used in this chapter. Mean bi-iliac breadth is close to significant difference as a result of maternal education, but is not statistically significant. Group statistics for the student t-test for equality of means in the shoulder and pelvic dimensions of girls using or not using oral contraception are presented in Table 5.12. These results show that there are a greater number of girls who reported not using oral contraception. The results of the student t-test for equality of means with oral contraceptive use are presented in Table 5.13. There were no statistically significant differences in shoulder breadth amongst girls using or not using oral contraception in the sample used in this chapter.

Table 5. 6. Results of the one-way ANOVA test of shoulder breadth, pelvic breadth dimensions and social code. Total degrees of freedom (df) represent sample size for analysis.

		Sum of Squares	df	Mean Square	F	p- value*
Shoulder	Between Groups	1.53	5	0.31	0.317	0.902
breadth	Within Groups	165.24	171	0.97		
SDS	Total	166.77	176			
BIIB SDS	Between Groups	2.42	5	0.48	0.553	0.736
	Within Groups	152.43	174	0.88		
	Total	154.85	179			
	Between Groups	8.54	5	1.71	1.865	0.103
	Within Groups	160.34	175	0.92		
303	Total	168.88	180			
DIAC	Between Groups	7.38	5	1.48	1.601	0.162
SDS	Within Groups	161.38	175	0.92		
	Total	168.76	180			

\* Values are significant at <0.05

Table 5.7. Results of the one-way ANOVA test of shoulder breadth, pelvic breadth dimensions and maternal education. Total degrees of freedom (df) represent sample size for analysis.

		Sum of Squares	df	Mean Square	F	p- value*
Shoulder	Between Groups	4.61	4	1.15	1.186	0.319
SDS	Within Groups	163.32	168	0.97		
303	Total	167.93	172			
BIIB SDS	Between Groups	8.05	4	2.01	2.182	0.073
	Within Groups	157.73	171	0.92		
	Total	165.78	175			
INML	Between Groups	1.18	4	0.30	.296	0.881
SDS	Within Groups	171.69	172	1.00		
	Total	172.87	176			
BIAC	Between Groups	4.39	4	1.10	1.133	0.343
SDS	Within Groups	166.71	172	0.97		
	Total	171.11	176			

\* Values are significant at <0.05

Table 5. 8. Results of the one-way ANOVA test of shoulder breath, pelvic breadth dimensions and paternal education. Total degrees of freedom (df) represent sample size for analysis.

		Sum of Squares	df	Mean Square	F	p- value*
Shoulder	Between Groups	7.54	5	1.51	1.52	0.186
breadth	Within Groups	157.48	159	0.99		
SDS	Total	165.02	164			
BIIB SDS	Between Groups	0.71	5	0.14	0.15	0.979
	Within Groups	150.65	161	0.94		
	Total	151.36	166			
	Between Groups	1.70	5	0.34	0.34	0.891
	Within Groups	164.82	162	1.02		
505	Total	166.52	167			
BIAC	Between Groups	5.98	5	1.20	1.26	0.286
SDS	Within Groups	154.25	162	0.95		
	Total	160.23	167			

\* Values are significant at <0.05

Table 5.9. Results for Tukey's range test for ANOVA analysis of shoulder breadth, pelvic breadth dimensions and social code.

Dependent		Mean	Std.	р-	95% Confidence Interval			
Variab	le		Difference	Error	value*	Lower Bound	Upper Bound	
		2	-0.05	0.21	1.000	-0.65	0.54	
		3	-0.19	0.27	0.980	-0.98	0.59	
	1	4	-0.03	0.27	1.000	-0.79	0.74	
		5	0.01	0.30	1.000	-0.84	0.86	
		7	0.38	0.47	0.965	-0.98	1.75	
		1	0.05	0.21	1.000	-0.54	0.65	
		3	-0.14	0.24	0.992	-0.82	0.55	
	2	4	0.03	0.23	1.000	-0.63	0.69	
		5	0.06	0.26	1.000	-0.70	0.82	
		7	0.44	0.45	0.928	-0.87	1.75	
		1	0.19	0.27	0.980	-0.59	0.98	
		2	0.14	0.24	0.992	-0.55	0.82	
	3	4	0.17	0.29	0.992	-0.67	1.00	
Shoulder		5	0.20	0.32	0.988	-0.71	1.12	
breadth		7	0.58	0.49	0.843	-0.83	1.98	
SDS		1	0.03	0.27	1.000	-0.74	0.79	
505		2	-0.03	0.23	1.000	-0.69	0.63	
	4	3	-0.17	0.29	0.992	-1.00	0.67	
		5	0.03	0.31	1.000	-0.86	0.93	
		7	0.41	0.48	0.958	-0.98	1.80	
		1	-0.01	0.30	1.000	-0.86	0.84	
		2	-0.06	0.26	1.000	-0.82	0.70	
	5	3	-0.20	0.32	0.988	-1.12	0.71	
		4	-0.03	0.31	1.000	-0.93	0.86	
		7	0.38	0.50	0.975	-1.07	1.82	
		1	-0.38	0.47	0.965	-1.75	0.98	
	_	2	-0.44	0.45	0.928	-1.75	0.87	
	7	3	-0.58	0.49	0.843	-1.98	0.83	
		4	-0.41	0.48	0.958	-1.80	0.98	
		5	-0.38	0.50	0.975	-1.82	1.07	
		2	0.08	0.20	0.999	-0.49	0.64	
		3	-0.03	0.26	1.000	-0.78	0.72	
	1	4	0.22	0.25	0.953	-0.51	0.95	
		5	-0.02	0.28	1.000	-0.82	0.77	
		1	0.59	0.45	0.701	-0.71	1.69	
		2	-0.08	0.20	0.999	-0.04	0.49	
	2	J ⊿	-0.11	0.23	0.997	-0.70	0.34	
	2	4	-0.14	0.22	0.907	-0.49	0.77	
BIIB SDS		7	-0.10	0.24	0.990	-0.31	1 75	
		, 1	0.03	0.40	1 000	-0.72	0.78	
		2	0.11	0.23	0.997	-0.54	0.76	
	3	4	0.25	0.28	0.945	-0.55	1 05	
	0	5	0.01	0.30	1 000	-0.85	0.86	
		7	0.62	0.46	0.766	-0.72	1.96	
		1	-0.22	0.25	0.953	-0.95	0.51	
	4	2	-0.14	0.22	0.987	-0.77	0.49	
		7	3	-0.25	0.28	0.945	-1.05	0.55

		5	-0.24	0.29	0.961	-1.08	0.60
		7	0.37	0.46	0.967	-0.96	1.69
		1	0.02	0.28	1.000	-0.77	0.82
		2	0.10	0.24	0.998	-0.60	0.81
	5	3	-0.01	0.30	1.000	-0.86	0.85
		4	0.24	0.29	0.961	-0.60	1.08
		7	0.61	0.47	0.788	-0.75	1.98
		1	-0.59	0.45	0.781	-1.89	0.71
		2	-0.51	0.43	0.846	-1.75	0.73
	7	3	-0.62	0.46	0.766	-1.96	0.72
		4	-0.37	0.46	0.967	-1.69	0.96
		5	-0.61	0.47	0 788	-1.98	0.75
		2	-0.24	0.20	0.826	-0.82	0.33
		3	0.15	0.27	0.992	-0.61	0.92
	1	4	0.36	0.26	0.730	-0.38	1 1 1
	•	5	-0.06	0.28	1 000	-0.88	0.75
		7	0.00	0.46	0.003	-1.07	1 50
		1	0.20	0.40	0.330	-0.33	0.82
		2	0.24	0.20	0.513	-0.27	1.06
	0	4	0.40	0.23	0.515	-0.27	1.00
	2	4	0.01	0.22	0.077	-0.04	1.25
		э 7	0.18	0.25	0.979	-0.54	0.90
		1	0.50	0.44	0.002	-0.77	1.70
		1	-0.15	0.27	0.992	-0.92	0.01
	~	2	-0.40	0.23	0.513	-1.06	0.27
	3	4	0.21	0.28	0.978	-0.61	1.02
		5	-0.22	0.30	0.980	-1.09	0.66
INML SDS		1	0.11	0.47	1.000	-1.26	1.47
		1	-0.36	0.26	0.730	-1.11	0.38
		2	-0.61	0.22	0.077	-1.25	0.04
	4	3	-0.21	0.28	0.978	-1.02	0.61
		5	-0.42	0.30	0.714	-1.28	0.44
		7	-0.10	0.47	1.000	-1.46	1.26
		1	0.06	0.28	1.000	-0.75	0.88
		2	-0.18	0.25	0.979	-0.90	0.54
	5	3	0.22	0.30	0.980	-0.66	1.09
		4	0.42	0.30	0.714	-0.44	1.28
		7	0.32	0.48	0.985	-1.07	1.72
		1	-0.26	0.46	0.993	-1.59	1.07
		2	-0.50	0.44	0.862	-1.78	0.77
	7	3	-0.11	0.47	1.000	-1.47	1.26
		4	0.10	0.47	1.000	-1.26	1.46
		5	-0.32	0.48	0.985	-1.72	1.07
		2	-0.19	0.20	0.937	-0.77	0.39
		3	0.08	0.27	1.000	-0.69	0.84
	1	4	0.00	0.26	1.000	-0.75	0.74
		5	-0.13	0.28	0.997	-0.95	0.68
		7	0.98	0.46	0.283	-0.35	2.31
		1	0.19	0.20	0.937	-0.39	0.77
BIAC SDS		3	0.26	0.23	0.865	-0.40	0.93
	2	4	0.18	0.22	0.963	-0.46	0.83
	-	5	0.05	0.25	1 000	-0.67	0.78
		7	1 17	0 44	0.095	-0 11	2 44
		1	-0.08	0.77	1 000	-0.84	0.69
	3	2	-0.26	0.23	0.865	-0.07 -0 93	0.00
	0	<u>د</u>	-0.20	0.20	1 000	_0.00	0.40
		4	-0.08	0.28	1.000	-0.90	0.74

	5	-0.21	0.31	0.983	-1.09	0.67
	7	0.90	0.48	0.408	-0.47	2.27
	1	0.00	0.26	1.000	-0.74	0.75
	2	-0.18	0.22	0.963	-0.83	0.46
4	3	0.08	0.28	1.000	-0.74	0.90
	5	-0.13	0.30	0.998	-0.99	0.73
	7	0.98	0.47	0.303	-0.38	2.34
	1	0.13	0.28	0.997	-0.68	0.95
	2	-0.05	0.25	1.000	-0.78	0.67
5	3	0.21	0.31	0.983	-0.67	1.09
	4	0.13	0.30	0.998	-0.73	0.99
	7	1.11	0.49	0.204	-0.29	2.51
	1	-0.98	0.46	0.283	-2.31	0.35
	2	-1.17	0.44	0.095	-2.44	0.11
7	3	-0.90	0.48	0.408	-2.27	0.47
	4	-0.98	0.47	0.303	-2.34	0.38
	5	-1.11	0.49	0.204	-2.51	0.29

\* Values are significant at <0.05

Table 5.10. Results for Tukey's range test for ANOVA analysis of shoulder breadth, pelvic breadth dimensions and maternal education.

Dependent Variable		Mean	Std Error	p-	95% Confidence Interval		
	aD	le	(I-J)	Sta. Error	value*	Lower Bound	Upper Bound
		2	0.48	0.49	0.865	-0.88	1.84
	4	3	-0.15	0.43	0.997	-1.34	1.04
	I	4	0.18	0.44	0.994	-1.02	1.38
		5	0.13	0.42	0.998	-1.02	1.28
		1	-0.48	0.49	0.865	-1.84	0.88
	n	3	-0.63	0.32	0.295	-1.53	0.26
	2	4	-0.30	0.33	0.893	-1.21	0.61
		5	-0.35	0.30	0.772	-1.20	0.49
Shoulder breadth SDS		1	0.15	0.43	0.997	-1.04	1.34
	0	2	0.63	0.32	0.295	-0.26	1.53
	3	4	0.33	0.23	0.600	-0.30	0.97
		5	0.28	0.19	0.589	-0.25	0.80
		1	-0.18	0.44	0.994	-1.38	1.02
	4	2	0.30	0.33	0.893	-0.61	1.21
	4	3	-0.33	0.23	0.600	-0.97	0.30
		5	-0.05	0.20	0.999	-0.61	0.50
		1	-0.13	0.42	0.998	-1.28	1.02
	F	2	0.35	0.30	0.772	-0.49	1.20
	5	3	-0.28	0.19	0.589	-0.80	0.25
		4	0.05	0.20	0.999	-0.50	0.61
		2	1.33	0.49	0.053	-0.01	2.68
	4	3	0.84	0.42	0.266	-0.31	2.00
	I	4	1.08	0.43	0.088	-0.09	2.25
		5	0.92	0.41	0.159	-0.20	2.04
		1	-1.33	0.49	0.053	-2.68	0.01
	S	3	-0.49	0.33	0.557	-1.39	0.41
	2	4	-0.26	0.33	0.939	-1.17	0.66
		5	-0.41	0.31	0.669	-1.26	0.44

		1	-0.84	0.42	0.266	-2.00	0.31
	_	2	0.49	0.33	0.557	-0.41	1.39
	3	4	0.24	0.22	0.825	-0.38	0.85
		5	0.08	0.18	0.992	-0.42	0.58
		1	-1.08	0.43	0.088	-2.25	0.09
		2	0.26	0.33	0.939	-0.66	1.17
	4	3	-0.24	0.22	0.825	-0.85	0.38
		5	-0.16	0.20	0.931	-0.69	0.38
		1	-0.92	0.41	0.159	-2.04	0.20
	_	2	0.41	0.31	0.669	-0.44	1.26
	5	3	-0.08	0.18	0.992	-0.58	0.42
		4	0.16	0.20	0.931	-0.38	0.69
		2	0.36	0.50	0.951	-1.02	1.74
		3	0.33	0.44	0.940	-0.87	1.54
	1	4	0.42	0.44	0.878	-0.80	1.64
		5	0.43	0.42	0.852	-0.74	1.59
		1	-0.36	0.50	0.951	-1.74	1.02
	-	3	-0.03	0.33	1.000	-0.93	0.87
	2	4	0.06	0.34	1.000	-0.87	0.98
		5	0.06	0.31	1.000	-0.79	0.91
		1	-0.33	0.44	0.940	-1.54	0.87
	_	2	0.03	0.33	1.000	-0.87	0.93
INML SDS	3	4	0.09	0.23	0.996	-0.55	0.72
		5	0.09	0.19	0.989	-0.43	0.61
		1	-0.42	0.44	0.878	-1.64	0.80
		2	-0.06	0.34	1.000	-0.98	0.87
	4	3	-0.09	0.23	0.996	-0.72	0.55
		5	0.01	0.20	1.000	-0.55	0.57
		1	-0.43	0.42	0.852	-1.59	0.74
	_	2	-0.06	0.31	1.000	-0.91	0.79
	5	3	-0.09	0.19	0.989	-0.61	0.43
		4	-0.01	0.20	1.000	-0.57	0.55
		2	0.52	0.49	0.833	-0.84	1.87
		3	0.08	0.43	1.000	-1.10	1.27
	1	4	0.27	0.44	0.971	-0.93	1.47
		5	0.43	0.42	0.843	-0.72	1.57
		1	-0.52	0.49	0.833	-1.87	0.84
	~	3	-0.43	0.32	0.669	-1.32	0.46
	2	4	-0.24	0.33	0.949	-1.15	0.67
		5	-0.09	0.30	0.998	-0.93	0.75
		1	-0.08	0.43	1.000	-1.27	1.10
	0	2	0.43	0.32	0.669	-0.46	1.32
BIAC SDS	3	4	0.19	0.23	0.921	-0.44	0.81
		5	0.34	0.19	0.357	-0.17	0.86
		1	-0.27	0.44	0.971	-1.47	0.93
	4	2	0.24	0.33	0.949	-0.67	1.15
	4	3	-0.19	0.23	0.921	-0.81	0.44
		5	0.15	0.20	0.940	-0.40	0.71
		1	-0.43	0.42	0.843	-1.57	0.72
	F	2	0.09	0.30	0.998	-0.75	0.93
	Э	3	-0.34	0.19	0.357	-0.86	0.17
		4	-0.15	0.20	0.940	-0.71	0.40

• Values are significant at <0.05

Table 5. 11. Results of Tukey's range test of ANOVA test of shoulder breadth, pelvic dimensions and paternal education.

Dependent Variable		Mean	Std.	р-	95% Confidence Interval		
Dependent vari	able	9	Ulfference (I-J)	Error	value*	Lower Bound	Upper Bound
		2	0.48	0.43	0.878	-0.77	1.74
		3	0.61	0.34	0.488	-0.38	1.60
	1	4	0.69	0.35	0.369	-0.32	1.70
		5	0.73	0.32	0.213	-0.20	1.65
		9	-0.21	0.65	0.999	-2.08	1.66
		1	-0.48	0.43	0.878	-1.74	0.77
		3	0.13	0.36	0.999	-0.90	1.16
	2	4	0.21	0.36	0.993	-0.84	1.26
		5	0.25	0.33	0.977	-0.72	1.21
		9	-0.69	0.66	0.897	-2.58	1.20
		1	-0.61	0.34	0.488	-1.60	0.38
		2	-0.13	0.36	0.999	-1.16	0.90
	3	4	0.08	0.25	1.000	-0.63	0.79
		5	0.12	0.20	0.992	-0.47	0.71
Shoulder breadth		9	-0.82	0.60	0.744	-2.55	0.91
SDS		1	-0.69	0.35	0.369	-1.70	0.32
		2	-0.21	0.36	0.993	-1.26	0.84
	4	3	-0.08	0.25	1.000	-0.79	0.63
		5	0.04	0.21	1.000	-0.58	0.66
		9	-0.90	0.60	0.668	-2.64	0.84
		1	-0.73	0.32	0.213	-1.65	0.20
		2	-0.25	0.33	0.977	-1.21	0.72
	5	3	-0.12	0.20	0.992	-0.71	0.47
		4	-0.04	0.21	1.000	-0.66	0.58
		9	-0.94	0.59	0.595	-2.63	0.75
		1	0.21	0.65	0.999	-1.66	2.08
		2	0.69	0.66	0.897	-1.20	2.58
	9	3	0.82	0.60	0.744	-0.91	2.55
	-	4	0.90	0.60	0.668	-0.84	2.64
		5	0.94	0.59	0.595	-0.75	2.63
		2	0.21	0.41	0.995	-0.98	1.40
		3	0.18	0.34	0.995	-0.79	1.15
	1	4	0.18	0.34	0.994	-0.79	1.16
		5	0.07	0.31	1.000	-0.83	0.97
		9	0.02	0.63	1.000	-1.79	1.84
		1	-0.21	0.41	0.995	-1.40	0.98
		3	-0.03	0.34	1.000	-1.00	0.93
	2	4	-0.03	0.34	1.000	-1.01	0.95
		5	-0.14	0.31	0.998	-1.04	0.76
BIIB SDS		9	-0.19	0.63	1.000	-2.01	1.63
		1	-0.18	0.34	0.995	-1.15	0.79
		2	0.03	0.34	1.000	-0.93	1.00
	3	4	0.00	0.24	1.000	-0.69	0.70
	0	5	-0.11	0.20	0.994	-0.68	0.47
		9	-0.16	0.58	1.000	-1.84	1.52
		1	-0.18	0.34	0.994	-1.16	0.79
	4	2	0.03	0.34	1.000	-0.95	1.01
	-	3	0.00	0.24	1.000	-0.70	0.69

	5	-0.11	0.21	0.994	-0.71	0.48
	9	-0.16	0.58	1.000	-1.85	1.53
	1	-0.07	0.31	1.000	-0.97	0.83
	2	0.14	0.31	0.998	-0.76	1.04
	53	0.11	0.20	0.994	-0.47	0.68
	4	0.11	0.21	0.994	-0.48	0.71
	9	-0.05	0.57	1.000	-1.69	1.59
	1	-0.02	0.63	1.000	-1.84	1.79
	2	0.19	0.63	1.000	-1.63	2.01
	э з	0.16	0.58	1.000	-1.52	1.84
	4	0.16	0.58	1.000	-1.53	1.85
	5	0.05	0.57	1.000	-1.59	1.69
	2	0.33	0.43	0.972	-0.91	1.57
	3	0.27	0.35	0.971	-0.74	1.28
	1 4	0.29	0.35	0.964	-0.73	1.31
	5	0.33	0.33	0.914	-0.61	1 27
	9	-0.18	0.66	1 000	-2.07	1 72
	1	-0.33	0.43	0.972	-1.57	0.91
	3	-0.06	0.35	1 000	-1.07	0.94
	2 1	-0.04	0.00	1.000	-1.06	0.04
	 5	-0.04	0.33	1.000	-0.94	0.90
	0 0	-0.51	0.00	0.072	-0.34	1 30
_	1	-0.31	0.00	0.372	-2.40	0.74
	י 2	-0.27	0.35	1 000	-1.20	1.07
	2 1	0.00	0.35	1.000	-0.94	0.74
	54 55	0.02	0.25	1.000	-0.70	0.74
	0	0.00	0.21	0.077	-0.55	0.05
INML SDS -	9	-0.45	0.01	0.977	-2.20	1.30
	ו ס	-0.29	0.35	1 000	-1.31	1.06
	<u>ک</u>	0.04	0.35	1.000	-0.98	1.00
	+ 3 5	-0.02	0.25	1.000	-0.74	0.70
	5	0.04	0.21	0.070	-0.56	0.00
_	9	-0.47	0.01	0.973	-2.22	1.29
	1	-0.33	0.33	1 000	-1.27	0.61
	- 0	0.00	0.33	1.000	-0.93	0.94
	ວ 3 ₄	-0.06	0.21	1.000	-0.65	0.53
	4	-0.04	0.21	1.000	-0.66	0.58
	9	-0.50	0.59	0.957	-2.22	1.21
	1	0.18	0.66	1.000	-1.72	2.07
	2	0.51	0.00	0.972	-1.39	2.40
	9 3	0.45	0.61	0.977	-1.30	2.20
	4	0.47	0.61	0.973	-1.29	2.22
	5	0.50	0.59	0.957	-1.21	2.22
	2	0.74	0.42	0.486	-0.46	1.94
	3	0.46	0.34	0.745	-0.51	1.44
	14	0.46	0.34	0.765	-0.53	1.45
	5	0.67	0.31	0.281	-0.24	1.57
_	9	0.07	0.64	1.000	-1.76	1.90
B110.070	1	-0.74	0.42	0.486	-1.94	0.46
BIAC SDS	3	-0.28	0.34	0.964	-1.25	0.70
:	2 4	-0.28	0.34	0.964	-1.27	0.71
	5	-0.07	0.31	1.000	-0.98	0.84
_	9	-0.67	0.64	0.899	-2.50	1.16
	1	-0.46	0.34	0.745	-1.44	0.51
:	32	0.28	0.34	0.964	-0.70	1.25
	4	0.00	0.24	1.000	-0.70	0.69

	5	0.21	0.20	0.906	-0.37	0.78
	9	-0.39	0.59	0.985	-2.09	1.30
	1	-0.46	0.34	0.765	-1.45	0.53
	2	0.28	0.34	0.964	-0.71	1.27
4	3	0.00	0.24	1.000	-0.69	0.70
	5	0.21	0.21	0.913	-0.39	0.81
	9	-0.39	0.59	0.986	-2.09	1.31
	1	-0.67	0.31	0.281	-1.57	0.24
	2	0.07	0.31	1.000	-0.84	0.98
5	3	-0.21	0.20	0.906	-0.78	0.37
	4	-0.21	0.21	0.913	-0.81	0.39
	9	-0.60	0.57	0.903	-2.25	1.06
	1	-0.07	0.64	1.000	-1.90	1.76
	2	0.67	0.64	0.899	-1.16	2.50
9	3	0.39	0.59	0.985	-1.30	2.09
	4	0.39	0.59	0.986	-1.31	2.09
	5	0.60	0.57	0.903	-1.06	2.25

\* Values are significant at <0.05

.

Table 5. 12. Group statistics for student t-test performed to examine mean differences between shoulder breadth and pelvic dimensions in girls using or not using oral contraception.

	Oral contraceptive use	Ν	Mean	Std. Deviation	Std. Error Mean
Shoulder	Yes	9	-0.19	1.18	0.39
breadth SDS	No	33	0.11	0.89	0.15
BIIB SDS	Yes	9	-0.01	0.70	0.23
	No	34	0.21	1.03	0.18
INML SDS	Yes	9	-0.05	0.62	0.21
	No	34	0.09	0.78	0.13
BIAC SDS	Yes	9	-0.41	0.92	0.31
	No	34	0.11	0.83	0.14

Table 5. 13. Results of student t-test for equality of means of shoulder breadth, pelvic breadth dimensions and oral contraceptive use. Degrees of freedom (df) represent sample size.

	t	df	p- value*	Mean difference	Std. error	95% Confidence interval (Lower)	95% Confidence interval (Upper)
Shoulder breadth SDS	-0.84	40	0.406	-0.30	0.36	-1.03	0.42
BIIB SDS	-0.61	41	0.548	-0.22	0.37	-0.96	0.52
INMLSDS	-0.49	41	0.625	-0.14	0.28	-0.71	0.43
BIAC SDS	-1.62	41	0.113	-0.52	0.32	-1.16	0.13
* Voluce are aid	anifiaan	+ _+ _					

\* Values are significant at <0.05

#### 5.6. Discussion

#### 5.6.1. Socioeconomic status and growth in girls

Univariate analyses showed no significant mean differences in shoulder breadth or pelvic breadth dimensions with social code. The absence of association between socioeconomic status (SES), growth and development in girls is somewhat unexpected. Early studies of growth variation in biological anthropology clearly demonstrated that human phenotypes were not genetically fixed but were influenced by the growth environment (for review, see Tanner, 1981). The availability of a nutritionally adequate diet, access to healthcare and the absence of chronic health conditions contribute to optimal growth in children and many of these factors are tied to socioeconomic status. Studies have found variable associations between child adiposity and socioeconomic status. Shrewsbury and Wardle's (2008) review found a generally inverse relationship between socioeconomic status and child adiposity (children with parents of lower SES tended to have a greater amount of adipose tissue) in the United Kingdom, the United States of America, Australia and Germany. In low-income countries such as Ghana, Vietnam and Burkina Faso men and women of higher SES were more likely to obese, whilst children of higher SES in low and middle-income countries were more likely to be obese (Dinsa et al., 2012).

The mean social code in this sample was 2.67 which includes professional, associate professional and technical occupations. This suggests that girls in this sample likely had access to nutritionally adequate diets and regular healthcare. Body mass index (BMI) could be used to outline which girls fall in to overweight and obese categories to explore the variation in body size and shape amongst growing girls living in above low-income environments. Dietz and Bellizzi (1999) explained that BMI was an appropriate measure to examine obesity in children and Cole et al. (2005) found BMI to be a more accurate measure than BMI z-scores (or SDS) to identify obesity amongst growing children. However Griffiths et al. (2012) suggested that both BMI and waist circumference could lead to misclassifying growing children as obese. According to BMI collected at each measurement session for the girls in the current study, approximately 0.25% of the total number of measurement sessions included BMI that would be considered obese (BMI of 30 to 39.9). This small

251
percentage of measurement points suggests that the sample in the present study is reflective of growing up in middle-income environments.

#### 5.6.2. Mother comes first – maternal education and growth

Mothers strongly influence child survival, particularly in the first two years of life (Mace and Sear, 2005). Parenting is additionally energetically costly for human mothers as a result of an average length of 2.5 to 3 years interbirth interval (in natural fertility communities). This means that mothers often raise multiple children at the same time (Sear and Mace, 2008). Previous research has suggested that further support for raising children comes in the form of female relatives (Hawkes et al., 1997) and grandmothers (Hawkes et al., 1998). This suggests that there may be an overall stronger maternal influence on daughter's growth. Runyan et al. (2003) found that height, weight and lumbar spine bone mineral density were significantly correlated between mothers and their daughters and daughter's lumbar spine bone mineral density inheritability was 0.70.

In the analyses performed in this chapter, there were no significant mean differences in shoulder breadth and pelvic breadth as a result of maternal education. BIIB was close to reaching statistically significant difference with maternal education but was not statistically meaningful. This may be due to intertwined associations between social code and parental education. Middle-income environments may also play a role in the type and frequency of caregiving to growing children. Results in Chapter Four demonstrated that shoulder breadth and BIIB were associated with localized measures of adiposity such as thigh circumference. Given that maternal education also has a statistical link with weight it is possible that a small percentage of pelvic dimension growth associates with maternal education. Tollanes et al. (2007) found that women with lower education levels were more likely to deliver a baby via emergency caesarean section and Woodman et al. (2006) demonstrated that women with low household annual incomes were more likely to suffer from pelvic organ prolapse when controlling for parity. Future studies specifically examining other biosocial factors and maternal and daughter pelvic dimensions would better clarify this relationship.

#### 5.6.3. Oral contraception and growth

The use of oral contraception prior to and during puberty has been identified as a potential factor in changes to accruing fat mass as girls grow into women (Berenson and Rahman, 2009). The univariate analyses used in this chapter found no statistically significant mean differences in shoulder and pelvic breadth dimensions with oral contraceptive use. This may be dependent on the type of oral contraceptive used. Reubinoff et al (1995) and de Melo et al. (2004) found that low dose oral contraceptives (oral contraceptives with 35 micrograms or less of oestrogen) had no effect on the body composition and fat mass of adult women. Central trunk adiposity and fat mass increased in women using depotmedroxyprogesterone acetate (known as the Depo-Provera injection) (Clark et al., 2005) which includes progestin instead of oestrogen as the main hormone compound. There are a number of factors that make it imprudent to draw conclusions on oral contraceptive use, fat mass and pelvic dimension growth from the dataset used in this chapter. Firstly, the type of oral contraceptive was not noted for the girls and women in this dataset. Secondly, many previous studies examining the effect of oral contraceptive use on body composition have focused on adult women instead of growing girls. Investigating potential effects of oral contraceptive use on adult women eliminates the complications of changing body composition in growing girls.

# 5.6.4. Biological variables have a greater effect on pelvic growth than biosocial variables

Comparing the results from this study to those presented in Chapter Four suggests that biological variables may have stronger associations with pelvic growth than biosocial variables. The full clarity of these relationships may not have been possible to explore in this study due to the types of biosocial data collected. The manner in which these variables are separated and tested may also play a role in the appearance of statistical relationships. For example, the average number of hours of vigorous physical activity a girl engages in is likely a function of SES and may even be affected by parental education. Further clarification on the associations between biosocial factors may also improve future studies of this type.

## 5.7. Conclusion

Results from this project demonstrate that pelvic growth and body breadth associate with biosocial variables linked to hormonal, maternal and nutritional factors. When compared with results from Chapter Four biosocial factors have an overall weaker relationship with pelvic growth compared to biological factors that suggests that any conclusions drawn from this study should be cautious. Multivariate tests demonstrate a particular strength in social code as it associates with BIIB and almost associates with INML. Oral contraceptive use associates significantly with BIAC which may in turn impact measures of adiposity. A more detailed form of data collection for the types of oral contraceptive used would clarify relationships between additional hormones and pelvic growth. The comparatively weaker association between biosocial variables and pelvic growth suggests that pelvic growth may be in some way buffered from environmental factors in growing girls. This mirrors Kurki's (2007) results on small-bodied populations and lends support to work pointing to a more complex suite of factors associated with pelvic size and shape than simply bipedality (Wells et al., 2012a; Warrener et al., 2015; Dunsworth, 2018).

# 5.8. Associations between biosocial variables and pelvic canal breadth variables vs. associations between biological variables and pelvic canal breadth variables

The results from this chapter can be compared with those from Chapter Four, demonstrating that there are more statistically significant associations between body size and shape variables and pelvic canal breadth variables in growing girls than between biosocial variables and pelvic canal breadth variables. The comparison of these results addresses the final research question of this thesis - can these results demonstrate if childbirth difficulty as a result of obstetrically compromised pelvic dimensions are ecological in origin? Comparing the results of this chapter and Chapter Four suggests that childbirth difficulty as a result of obsetricially compromised pelvic dimensions arises from a combination of genetic and environmental factors.

#### CHAPTER SIX:

#### Summary and Conclusions

#### 6.1. The role of Chapter Six in this thesis

This chapter serves as a summary of the findings of Chapters Two, Three, Four and Five of this thesis. This chapter revisits some of the literature outlined in Chapter One to better contextualise the findings of this thesis as a whole.

#### 6.2. Summary of project aims

This thesis examined the biological and biosocial factors associated with pelvic dimension growth and how this relates to changes in body size and shape in growing girls from southeast England. Methods used to examine pelvic growth included linear measurements collected for medical imaging, anthropometry and lifestyle data collected from participants at clinical visits to the Great Ormond Street Hospital in London, England between 2000 and 2016.

The overarching aim of the project was to clarify the components linked to pelvic growth in a living population to better understand factors contributing to pelvic dimensions that could affect childbirth ability in women in the past and the present. Previous scholarship designated the biomechanical difficulty of childbirth in humans as a consequence of energetically efficient bipedality (Washburn, 1960). More recent work has suggested that ecological factors impacted on early life growth and pelvic dimensions such as the advent of agriculture (Wells et al., 2012a) and experimental studies have demonstrated no loss of efficiency with increased hip breadth (Warrener et al., 2015). Instead of a biomechanical limitation driving the tight relationship between the neonate and the mother, Dunsworth et al. (2012) suggested that a limitation on maternal metabolism may account for the level of altriciality seen in humans. Wells et al. (2017) found that maternal pelvic dimensions better predicted the size of neonates, suggesting that neonatal size is sensitive to maternal dimensions to reduce cephalo-pelvic disproportion. This further complicates the concept of the obstetric dilemma and highlights the variety of factors connected with

on pelvic growth and childbirth outcomes in living populations. It is likely that the same variety of factors would also have affected women living in the past.

The secondary aim of the project was to demonstrate the validity of integrating medical imaging data collected from living people with an evolutionary perspective on human variation. Evolutionary medicine provides the theoretical basis to integrate evolutionary frameworks with current medical knowledge, whilst also acknowledging the importance of cultural practice (Grunspan et al., 2018). Integrating evolutionary perspectives with medical data in this project expands on established knowledge in both biological anthropology and clinical practice in child growth and development.

#### 6.3. Summary of findings

# 6.3.1. Dual energy absorptiometry scans be used for osteological data collection

Linear measurements of pelvic breadth collected from DXA scans were found to be comparable with linear measurements collected from magnetic resonance images (MRI). There was no significant measurement bias for BIIB, INML and BIAC dimensions collected from DXA scans compared to these dimensions collected from MRI scans. DXA BIIB measurements most closely matched MRI BIIB and this pelvic dimension also had the lowest percentage of bias (0.07%) between DXA and MRI imaging methods. The acceptable level of measurement error between these imaging methods suggests that DXA can be used to collect osteometric data in the mediolateral direction. A similar examination of distal-proximal measurement error would enable future studies to examine DXA osteometric data in a similar manner to dry bone measurements. DXA scans are frequently collected in both cross-sectional and longitudinal studies examining child growth (Mazness et al., 1990; Ogle et al., 1995; Singhal et al., 2003; Gale et al., 2007; Ong et al., 2007; Kelly et al., 2009; Kennedy et al., 2012; Kavle et al., 2016) and changes in body composition and bone strength in adults (Duboeuf et al., 1991; Jones et al., 1994; Snijder et al., 2002; Kensara et al., 2005; Blake and Fogelman, 2007). This represents a wealth of opportunities to examine change in shape and size of skeletal elements during different periods of life in living populations. Results from this study also demonstrated that image resolution

could render identification of anatomical measurement points difficult. It is recommended that future studies making use of DXA-based skeletal data complete measurement error studies to quantify precision and error.

#### 6.3.2. Different growth trajectory of canal vs. non-canal parts of the pelvis

There were distinct differences in the tempo and factors associated with the growth of the canal and non-canal parts of the pelvis. INML and BIAC both increased in growth velocity between 10 and 12 years of age and dramatically reduced in growth velocity between 12 and 14 years of age. BIIB growth velocity decreased throughout puberty in a similar manner to weight and height. The difference between these growth trajectories suggests 1) growth priority changes from growth in height and breadth to pelvic canal growth with the onset of puberty and 2) the pelvic canal can be considered a functionally unique set of features that may be buffered from environmental stresses with the onset of puberty.

There were a greater number of significant associations between biological variables and pelvic dimensions than biosocial variables and pelvic dimensions. Shoulder breadth associated with height and thigh circumference. BIIB associated with height and thigh circumference. This suggests that breadth aligns in growth trajectory through the head and pelvis. INML associated with height and Tanner development stage. BIAC associated with height and Tanner development stage. BIAC was collected as a proxy measure for midplane pelvic canal breadth. The midplane of the canal is the most obstetrically important dimension as this part of the canal includes the space between the ischial spines, the narrowest dimension of the canal (Walrath and Glanz, 1996; Tague, 2000; Kurki, 2007, 2011) and the most challenging for an neonate to pass through during childbirth. The twinned association between height and Tanner development stages and BIAC suggests that fat mass and localised measures of adiposity may be more relevant measures relating to the growth pelvic dimensions as girls grow compared to the more frequently used height as indication of childbirth difficulty amongst women (Liselele et al., 2000; Prasad and Al-Taher, 2002; Toh-Adam et al., 2012).

Different growth trajectories of the canal compared to the non-canal parts of the pelvis demonstrated in this thesis elucidates work examining the potential for

'evolvability' of the pelvis. Grabowski (2013) examined the evolution of obstetric constraints in the human pelvis when compared to ape pelves. Grabowski (2013) found that divergent selection pressures shaped the human birth canal, as selection on non-birth related components of the pelvis reduced levels of genetic constraint, freeing the evolution of the canal from a lengthier evolutionary process. Grabowski and Roseman (2015) further contextualized Grabowski's (2013) findings by examining strong directional selection on specific pelvic traits, concluding that the evolutionary rate of change for pelvic traits in humans was greater than for other sets of traits in concurrently evolving mammals. The differentiation in pelvic growth trajectories demonstrated by the results of this thesis underline the relevance of a specific evolutionary trajectory relating not only to the locomotive demands of the pelvis, but also the obstetric components of the bony pelvis. The association between Tanner development stage and canal measures of the pelvis in growing girls established in this thesis also suggest that human developmental stages (which are unique from an evolutionary perspective) may have an equally important role to play in the evolution of the human pelvis.

## 6.3.3. The pelvic canal continues to grow into early adulthood

Growth velocity, centile charts and spaghetti charts of pelvic growth demonstrated that pelvic breadth growth has not terminated with the onset of biological adulthood. The INML centile chart shows that women in the second, ninth and 25<sup>th</sup> percentiles at 20 years old and above would fall within clinically contracted thresholds for this measure. Both Völgyi et al. (2010) and Moerman (1982) found that pelvic canal growth occurred at a slower pace than linear growth in girls. The results from this project align with these previous findings and suggest that there is an evolutionary advantage to delaying pelvic canal growth completion. It would be advantageous to select for a delay in pelvic canal growth completion to match the extended period of social development that is representative of human childhood and puberty (Roff, 1992; Stearns, 1992; Charnov, 1993; Bogin, 1994, 1999b; a). It would be logical then for teenage pregnancy to result in childbirth difficulty due to the delay in pelvic breadth growth. However studies have demonstrated that neonatal size is smaller in teenage pregnancies (Chandra et al., 2002; Kirchengast and Hartmann,

2003) which could act to reduce childbirth difficulties though may increase likelihood of chronic health issues later in life for the neonate due to low birthweight.

# 6.4. Wider implications

## 6.4.1. Bi-iliac breadth is representative of body breadth during growth

The associations between BIIB, shoulder breadth and height in this project demonstrate that BIIB should be considered as a component of body breadth that is affected by both genetic and environmental factors. Previous studies have found BIIB form to alter with lower latitudes (narrower) (Ruff, 1991, 1994, 2002, 2010; Holliday, 2012; Roseman and Auerbach, 2015) and higher latitudes (wider) (Ruff et al., 2005) and that BIIB dimensions demonstrate less variation than canal components of the pelvis (Kurki and Decrausaz, 2016). BIIB may be more sensitive to environmental changes (such as differences in latitude) during growth however BIIB should no longer be considered to follow the functional reactions as the canal components of the pelvis.

# 6.4.2. Pelvic size and shape variation during growth suggests multiple causes of birth difficulty

The varied associations between pelvic dimensions, measures of body size and shape and markers of puberty indicated by the results of thesis suggest that the singular obstetric dilemma is insufficient to account for the biomechanical issues of human childbirth. It is more likely that there are multiple obstetric dilemmas (Wells et al., 2012a; Wells, 2017; Dunsworth, 2018) that arise from the consequences of poor early life nutrition, chronic infection, interactions between soft tissue and skeletal tissue in adulthood and during growth, biosocial practices associated with childbirth, neonatal size and social inequalities that affect women's abilities to make their own reproductive choices (Wells 2017). This is somewhat in contrast with Mitteroecker et al.'s (2016) cliff-edge model of obstetric selection. Mitteroecker et al. (2016) tightly focus on the tussle between selection for a wider birth canal, a larger infant and narrower pelvis to prevent pelvic floor instability, demonstrating that foetopelvic disproportion (FPD) and the necessity for caesarean section deliveries are a result of comparatively weak selective forces for narrower pelves and/or larger infants.

Mitteroecker and colleagues (2016) do not extensively examine other nonevolutionary causes for FPD, and do not include any discussion on the role of variation in maternal soft tissue that could affect childbirth difficulties and the option for caesarean delivery (Grossman, 2017). It should be noted that in reply to Grossman's (2017) critique Mitteroecker et al. (2017) highlighted that the cliff-edge model was a theoretical model and not empirical data. Mitteroecker et al.'s (2016) model provide a strong theoretical basis for examining evolutionary forces enacted on the pelvis and human childbirth, however the model does not account for the diversity in influence of nutritional, developmental, social and population-based factors that affect the mismatch between the maternal birth canal and infants. The results of this thesis demonstrate that during growth the certain parts of the bony pelvis may be more susceptible to changes in dimension, which may arise from both genetic and environmental factors. To wholly account for the variables that affect the growth and variation in the human pelvis and the interaction between childbirth difficulties and medical practice calls for transdisciplinary engagement (Mitteroecker, 2018).

## 6.5. Areas for future study

## 6.5.1. Pelvic growth in boys

A comparative study examining the growth of the pelvis in boys would clarify and deepen the analyses completed in this project. Markers of sexual dimorphism in the female pelvis are the focus of multiple studies, often motivated by the clarification of obstetric adaptations (Coleman, 1969; Tague, 1992, 2000; Walrath and Glanz, 1996; Huseynov et al., 2016; Delprete, 2017; Fischer and Mitteroecker, 2017; Whitcome et al., 2017). In addition, many of these studies have examined adult female pelves. A greater understanding of the relationships between biological factors and male pelvic growth will clarify the contribution of paternal genetic inheritance on a daughter's pelvic dimensions as well as elucidate important differences in skeletal sexual characteristic development in males.

#### 6.5.2. Female pelvic growth through early adulthood and into middle age

The results of this project and other studies (e.g. Huseynov et al. (2016)) suggest that female pelvic size and shape continues to change throughout adult life. It is likely that parity will impact on pelvic dimensions though also possible that pregnancy alone may result in pelvic changes as a result of the actions of the hormone relaxin at the sacroiliac joint (Albert et al., 1997). Cross-sectional studies examining pelvic size and shape in middle-aged through to older women and including data collection on reproductive histories would be an ideal manner to examine whether pelvic dimensions continue to change in adulthood. Such studies may also further inform clinicians looking to address risk factors for reproductive organ prolapse.

#### 6.5.3. Tracking hormonal alterations alongside skeletal growth

It is challenging to interpret growth trajectories from skeletal dimensions without a clear understanding of the precise mechanisms of hormones that act on skeletal tissue and initiate secondary sexual characteristics. Future studies on pelvic growth could incorporate tracking levels of hormones with the onset of puberty including growth hormones and sex hormones. Recent work by Tobolsky et al. (2018) examining urinary c-peptide as a measure of growth demonstrated that this method could be used to investigate rates of bone turnover in growing children. Whilst it may be too complex to separate specific types of hormones in these types of analyses, a general hormonal profile alongside growing skeletal tissue would help integrate skeletal tissue with other physiological processes in the human body.

#### 6.6. Conclusion

This doctoral thesis examined the relationship between biological and biosocial factors and pelvic breadth of growing girls living in southeast England. The methods used to investigate this question were sourced from clinical studies of child growth and development and studies of human evolution, including novel osteological data collection from medical imaging. The results of this project support the current rethink of the obstetric dilemma in biological anthropology and add credence to the validity of evolutionary medicine as a means of approaching obstetric adaptations in humans.

Results from this project include both completely novel findings and outcomes that further contextualise current research in biological anthropology. Centile and velocity charts for pelvic breadth demonstrated differences in growth pattern and speed of canal vs. non-canal components of the pelvis. These also suggested a change in growth priority from body height and body breadth prior to an increase in growth velocity for pelvic breadth prior to the onset of puberty. Multiple regression analyses of the biological variables associating with pelvic dimensions further outlined differences in canal components of the pelvis compared to bi-iliac breadth growth. Bi-iliac breadth associates with height and aligns with other measures of bodily breadth whilst canal measures of pelvic breadth associate with indicators of adiposity as girls grow. Comparatively the association between pelvic dimensions and biosocial variables are less distinct though associations between social code, and pelvic dimensions likely reflect important effects of overall health and nutrition alongside pubertal development. Oral contraceptive use also associated with biacetabular breadth, demonstrating the importance of including any additional hormonal factors when examining growth in living girls and women.

This thesis investigated the growth of the pelvis in girls in order to clarify factors that contribute to biomechanical childbirth difficulty in adult women both in the past and today. The results from this thesis indicate that pelvic canal growth may be somewhat buffered from environmental effects as within-individual variables associate with pelvic dimensions to a greater degree as girls grow than biosocial factors. The sample used for this project consists of healthy European girls and thus is not ideal as a comparative sample to interpret pelvic growth in past populations. However, the results of this thesis form an important working basis for examining skeletal growth alongside soft tissue growth in children with a specific focus on pelvic dimensions.

# Literature cited

Abbassi V. 1998. Growth and normal puberty. Pediatrics 102:507–511.

- Adair F. 1918. The ossification centers of the fetal pelvis. Trans Am Gynecol Soc Year 43:89.
- Agarwal SC, Grynpas MD. 2009. Measuring and interpreting age-related loss of vertebral bone mineral density in a medieval population. Am J Phys Anthropol 139:244–252.
- Albert H, Godskesen M, Westergaard JG, Chard T, Gunn L. 1997. Circulating levels of relaxin are normal in pregnant women with pelvic pain. Eur J Obstet Gynecol Reprod Biol 74:19–22.
- Alduc-Le Bagousse A, Blondiaux J. 2002. Mortalité maternelle et périnatalité au premier millénaire à Lisieux (Calvados, France). Bull Mem Soc Anthropol Paris 14:295–309.
- Altman DG, Bland JM. 1983. Measurement in medicine: The analysis of method comparison studies. J R Stat Soc Ser D The Stat 32:307–317.
- Ammann P, Rizzoli R. 2003. Bone strength and its determinants. Osteoporos Int 14:13–18. A
- Arriaza B, Allison M, Gerszten E. 1988. Maternal mortality in pre-Columbian Indians of Arica, Chile. Am J Phys Anthropol 77:35–41.
- Ashton-Miller J a, Delancey JOL. 2009. On the biomechanics of vaginal birth and common sequelae. Annu Rev Biomed Eng 11:163–176.
- Auerbach BM, King KA, Campbell RM, Campbell ML, Sylvester AD. 2018. Variation in obstetric dimensions of the human bony pelvis in relation to age-at-death and latitude. Am J Phys Anthropol 167:628–643.
- Bader TR, Semelka RC, Chiu VCY, Armao DM, Woosley JT. 2001. MRI of carcinoid tumors: Spectrum of appearances in the gastrointestinal tract and liver. J Magn Reson Imaging 14:261–269.
- Bailey DA, Mckay HA, Mirwald RL, Crocker PRE, Faulkner RA. 1999. A Six-Year Longitudinal Study of the Relationship of Physical Activity to Bone Mineral Accrual in Growing Children: The University of Saskatchewan Bone Mineral Accrual Study. J Bone Miner Res 14:1672–1679.

Balfour-Lynn L. 1986. Growth and childhood asthma. Arch Dis Child 61:1049–55.

- Baragi R, Delancey JOL, Caspari R, Howard D, Ashton-Miller JA. 2002. Differences in pelvic floor area between African American and European American women. Am J Obstet Gynecol 187:111–115.
- Bardeen C. 1905. Studies of the development of the human skeleton. A) The development of the lumbar, sacral and coccygeal vertebrae. B) The curves and the proportionate regional lengths of the spinal column during the first three months of embryonic development. C) The development of the skeleton of the posterior limb. Am J Anat 4:265–302.
- Barker DJ., Godfrey K., Gluckman P., Harding J., Owens J., Robinson J. 1993. Fetal nutrition and cardiovascular disease in adult life. Lancet 341:938–941.
- Barker DJP. 2007. The origins of the developmental origins theory. J Intern Med 261:412–417.
- Barker DJP, Eriksson JG, Forsén T, Osmond C. 2002. Fetal origins of adult disease: Strength of effects and biological basis. Int J Epi 31:1235–1239.
- Barker DJP, Osmond C, Winter PD, Margetts B, Simmonds SJ. 1989. Weight in infancy and death from ischaemic heart disease. Lancet 2:577–580.
- Barnhart KT, Izquierdo A, Pretorius ES, Shera DM, Shabbout M, Shaunik A. 2006. Baseline dimensions of the human vagina. Hum Reprod 21:1618–1622.
- Baxter-Jones ADG, Eisenmann JC, Mirwald RL, Faulkner RA, Bailey DA. 2008. The influence of physical activity on lean mass accrual during adolescence: a longitudinal analysis. J Appl Physiol 105:734–741.
- Becher N, Waldorf KA, Hein M, Uldbjerg N. 2009. The cervical mucus plug: Structured review of the literature. Acta Obstet Gynecol Scand 88:502–513.
- Bello SM, Thomann A, Signoli M, Dutour O, Andrews P. 2006. Age and sex bias in the reconstruction of past population structures. Am J Phys Anthropol 129:24– 38.
- Berenson A, Rahman M. 2009. Changes in weight, total fat, percent body fat, and central-to-peripheral fat ratio associated with injectable and oral contraceptive use. Am J Obstet Gynecol 200:329.e1-329.e8.
- Berger AA, May R, Renner JB, Viradia N, Dahners LE. 2011. Surprising evidence of pelvic growth (widening) after skeletal maturity. J Orthop Res 29:1719–1723.
- Berger MB, Doumouchtsis SK, Delancey JO. 2013. Are bony pelvis dimensions associated with levator ani defects? A case-control study. Int Urogynecol J 24:1377–83.

- Bergot C, Bousson V, Meunier A, Laval-Jeantet M, Laredo JD. 2002. Hip fracture risk and proximal femur geometry from DXA scans. Osteoporos Int 13:542–550.
- Betti L, von Cramon-Taubadel N, Manica A, Lycett SJ. 2014. The interaction of neutral evolutionary processes with climatically-driven adaptive changes in the 3D shape of the human os coxae. J Hum Evol 73:64–74.
- Bhutta Z, Gupta I, De'Silva H, Manandhar D, Awasthi S, Moazzem Hossain S, Salam M. 2004. Maternal and child health: Is South Asia ready for change? Br Med J 328:816–819.
- Bielicki T. 1975. Interrelationships between various measures of maturation rate in girls during adolescence. Stud Phys Anthropol 1:51–64.
- Bjørnerud A, Johansson L. 2004. The utility of superparamagnetic contrast agents in MRI: Theoretical consideration and applications in the cardiovascular system. NMR Biomed 17:465–477.
- Black RE, Williams SM, Jones LE, Goulding A. 2002. Children who avoid drinking cow milk have low dietary calcium intakes and poor bone health. Am J Clin Nutr 76:675–680.
- Blake GM, Fogelman I. 2007. Role of Dual-Energy X-Ray Absorptiometry in the Diagnosis and Treatment of Osteoporosis. J Clin Densitom 10:102–110.

Blurton Jones N. 1978. Natural selection and birthweight. Ann Hum Biol 5:487–489.

- Boaz F. 1912. Changes in the Bodily Form of Descendants of Immigrants. Am Anthropol 14:530–562.
- Bogin B. 1994. Adolescence in evolutionary perspective. Acta Pædiatrica 83:29-35.
- Bogin B. 1999a. Evolutionary perspectives on human growth. Annu Rev Anthropol 28:109–153.
- Bogin B. 1999b. Patterns of human growth. Second. Cambridge: Cambridge University Press.
- Bogin B, Rios L. 2003. Rapid morphological change in living humans: implications for modern human origins. Comp Biochem Physiol 136(1):71–84.
- Bogin B, Scheffler C, Hermanussen M. 2017. Global effects of income and income inequality on adult height and sexual dimorphism in height. Am J Hum Biol 29:1–11.
- Bogin B, Smith BH. 2000. Evolution of the human life cycle. In: Stinson S, Bogin B, Huss-Ashmore R, O'Rourke D, editors. Human biology: An evolutionary and biocultural perspective. New York: Wiley-Liss. p 377–424.

- Bogin B, Smith P, Orden A, B, Silva MI V, Loucky J. 2002. Rapid change in height and body proportions of Maya American children. Am J Hum Biol 14:753–761.
- Bogin B, Varela-Silva MI. 2006. Life history trade-offs in human growth: Adaptation or pathology? Am J Hum Biol 19:631–642.
- Bogin B, Varela-Silva MI. 2008. Fatness biases the use of estimated leg length as an epidemiological marker for adults in the NHANES III sample. Int J Epidemiol 37:201–209.
- Bogin B, Varela-Silva MI. 2010. Leg length, body proportion, and health: A review with a note on beauty. Int J Environ Res Public Health 7:1047–1075.
- Bouhallier J, Berge C, Penin X. 2004. Analyse Procuste de la cavité pelvienne des australopithèques (AL 288, Sts 14), des humains et des chimpanzés: Conséquences obstétricales. Comptes Rendus - Palevol 3:293–302.
- Bowden D, Winter P, Ploog D. 1967. Pregnancy and delivery behavior in the squirrel monkey (Saimiri sciureus) and other primates. Folia Primatol 5:1–42.

Box G, Cox D. 1964. An analysis of transformations. J R Stat Soc Ser B 26:211–252.

- Bundak R, Darendeliler F, Gunoz H, Bas F, Saka N, Neyzi O. 2007. Analysis of puberty and pubertal growth in healthy boys. Eur J Pediatr 166:595–600.
- Bush G, Whalen PJ, Rosen BR, Jenike MA, McInerney SC, Rauch SL. 1998. The counting Stroop: an interference task specialized for functional neuroimaging--validation study with functional MRI. Hum Brain Mapp 6:270–282.
- Cameron ME, Pfeiffer S. 2014. Long bone cross-sectional geometric properties of Later Stone Age foragers and herder-foragers. S Afr J Sci 110:1–11.
- Campillo D, Vilaseca A, Casamitjana E, Ayestarán N. 1998. Esqueleto de una mujer fallecida por Distocia, pertene- ciente al período tardoromano (Mas Rimbau, Tarragona). Empúries 51:251–256.
- Centres for Disease Control and Prevention. 2007. Anthropometry procedures manual. Available from: https://www.cdc.gov/nchs/data/nhanes/nhanes\_07\_08/manual\_an.pdf%0Ahttp:// www.cdc.gov/nchs/data/nhanes/nhanes\_07\_08/manual\_an.pdf
- Chandra PC, Schiavello HJ, Ravi B, Weinstein AG, Hook FB. 2002. Pregnancy outcomes in urban teenagers. Int J Gynaecol Obstet 79:117–122.

Charnov EL. 1993. Life history invariants. Oxford: Oxford University Press.

- Chomtho S, Wells JC, Williams J, Lucas A, Fewtrell MS. 2008a. Associations between birth weight and later body composition: evidence from the 4-component model. Am J Clin Nutr 88:1040–1048.
- Chomtho S, Wells JCK, Williams JE, Davies PS, Lucas A, Fewtrell MS. 2008b. Infant growth and later body composition: evidence from the 4-component model. Am J Clin Nutr 87:1776–1784.
- Clark MK, Dillon JS, Sowers M, Nichols S. 2005. Weight, fat mass, and central distribution of fat increase when women use depot-medroxyprogesterone acetate for contraception. Int J Obes 29:1252–1258.
- Cole TJ. 1988. Fitting smoothed centile curves to reference data. J R Stat Soc Ser A (Statistics Soc 151:385–418.
- Cole TJ. 1989. Using the LMS method to measure skewness in the NCHS and Dutch National height standards. Ann Hum Biol 16:407–419.
- Cole TJ. 1990. The LMS method for constructing normalized growth standards. Eur J Clin Nutr 44:45–60.
- Cole TJ. 1993. The Use and Construction of Anthropometric Growth Reference Standards. Nutr Res Rev 6:19–50.
- Cole TJ. 1995. Conditional reference charts to assess weight gain in British infants. Arch Dis Child 73:8–16.
- Cole TJ. 2012. The development of growth references and growth charts. Ann Hum Biol 39:382–394.
- Cole TJ, Faith MS, Pietrobelli A, Heo M. 2005. What is the best measure of adiposity change in growing children: BMI, BMI %, BMI z-score or BMI centile? Eur J Clin Nutr 59:419–425.
- Cole TJ, Flegal KM, Nicholls D, Jackson AA. 2007. Body mass index cut offs to define thinness in children and adolescents: international survey. Bmj 335:194–194.
- Cole TJ, Freeman J V., Preece M a. 1998. British 1990 growth reference centiles for weight, height, body mass index and head circumference fitted by maximum penalized likelihood. Stat Med 17:407–429.
- Cole TJ, Freeman J V, Preece MA. 1995. Body mass index reference curves for the UK, 1990. Arch Dis Child 73:25–29.
- Coleman WH. 1969. Sex differences in the growth of the human bony pelvis. Am J Phys Anthropol 31:125–151.

- Cooper C, Kuh D, Egger P, Wadsworth M, Barker D. 1996. Childhood growth and age at menarche. Br J Obs 103:814–817.
- Correia H, Balseiro S, De Areia M. 2005. Sexual dimorphism in the human pelvis: Testing a new hypothesis. HOMO - J Comp Hum Biol 56:153–160.
- Cosman F, de Beur SJ, LeBoff MS, Lewiecki EM, Tanner B, Randall S, Lindsay R. 2014. Clinician's Guide to Prevention and Treatment of Osteoporosis. Osteoporos Int 25:2359–2381.
- Cowgill LW. 2010. The ontogeny of Holocene and late Pleistocene human postcranial strength. Am J Phys Anthropol 141:16–37.
- Cowgill LW, Eleazer CD, Auerbach BM, Temple DH, Okazaki K. 2012. Developmental variation in ecogeographic body proportions. Am J Phys Anthropol 148:557–70.
- Crelin E. 1969. Interpubic ligament: elasticity in pregnant free-tailed bat. Science (80) 164:81–82.
- Crooks DL. 1999. Child growth and nutritional status in a high-poverty community in aastern Kentucky. 142:129–142.
- Cruz CB, Codinha S. 2010. Death of mother and child due to dystocia in 19th century Portugal. Int J Osteoarchaeol 20:491–496.
- Cucina A, Tiesler V. 2003. Dental caries and antemortem tooth loss in the Northern Peten area, Mexico: A biocultural perspective on social status differences among the Classic Maya. Am J Phys Anthropol 122:1–10.
- Cummings SR, Browner W, Cummings SR, Black DM, Nevitt MC, Browner W, Genant HK, Cauley J, Ensrud K, Scott J, Vogt TM. 1993. Bone density at various sites for prediction of hip fractures. Lancet 341:72–75.
- Cunningham FG, Williams, W J. 2005. Williams obstetrics. 22nd ed. (Cunningham FG, editor.). New York: McGraw-Hill, Medical Publishing Division.
- Cunningham PM, Brennan D, O'Connell M, MacMahon P, O'Neill P, Eustace S. 2007. Patterns of bone and soft-tissue injury at the symphysis pubis in soccer players: observations at MRI. AJR Am J Roentgenol 188:291–296.
- Damilakis J, Adams JE, Guglielmi G, Link TM. 2010. Radiation exposure in X-raybased imaging techniques used in osteoporosis. Eur Radiol 20:2707–2714.
- Dangour AD, Schilg S, Hulse JA, Cole TJ. 2002. Sitting height and subischial leg length centile curves for boys and girls from Southeast England. Ann Hum Biol 29:290–305.

- Delprete H. 2017. Pelvic Inlet Shape Is Not as Dimorphic as Previously Suggested. Anat Rec 300:706–715.
- Dempster P, Aitkens S. 1995. A new air displacement method for the determination of human body composition. Med Sci Sports Exerc 27:1692–1697.
- Demuru E, Ferrari PF, Palagi E. 2018. Is birth attendance a uniquely human feature? New evidence suggests that Bonobo females protect and support the parturient. Evol Hum Behav 39(5):502-510.
- Denham WW. 1974. Population structure, infant transport, and infanticide among Pleistocene and modern hunter-gatherers. J Anthropol Res 30:191–198.
- DeSilva JM. 2011. A shift toward birthing relatively large infants early in human evolution. Proc Natl Acad Sci 108:1022–1027.
- DeSilva JM, Lesnik JJ. 2008. Brain size at birth throughout human evolution: A new method for estimating neonatal brain size in hominins. J Hum Evol 55:1064–1074.
- Desseauve D, Fradet L, Lacouture P, Pierre F. 2017. Position for labor and birth: State of knowledge and biomechanical perspectives. Eur J Obstet Gynecol Reprod Biol 208:46–54.
- Devakumar D, Grijalva-Eternod C, Cortina-Borja M, Williams JE, Fewtrell MS, Wells JC. 2016. Disentangling the associations between parental BMI and offspring body composition using the four-component model. Am J Hum Biol 28:524–533.
- Dietz WH, Bellizzi MC. 1999. Introduction: the use of body mass index to assess obesity in children. Am J Clin Nutr 70:123S–5S.
- Dinsa GD, Goryakin Y, Fumagalli E, Suhrcke M. 2012. Obesity and socioeconomic status in developing countries: A systematic review. Obes Rev 13:1067–1079.
- Dombrowski S, Jacob L, Hadji P, Kostev K. 2017. Oral contraceptive use and fracture risk—a retrospective study of 12,970 women in the UK. Osteoporos Int 28:2349–2355.
- Dror DK, Allen LH. 2014. Dairy product intake in children and adolescents in developed countries: Trends, nutritional contribution, and a review of association with health outcomes. Nutr Rev 72:68–81.
- Duboeuf F, Braillon P, Chapuy MC, Haond P, Hardouin C, Meary MF, Delmas PD, Meunier PJ. 1991. Bone mineral density of the hip measured with dual-energy Xray absorptiometry in normal elderly women and in patients with hip fracture. Osteoporos Int 1:242–249.

Dufour DL. 2006. Biocultural approaches in human biology. Am J Hum Biol 18:1–9.

- Dunger DB, Lynn Ahmed M, Ong KK. 2005. Effects of obesity on growth and puberty. Best Pract Res Clin Endocrinol Metab 19:375–390.
- Dunsworth HM. 2018. There is no "obstetrical dilemma": Towards a braver medicine with fewer childbirth interventions. Perspect Biol Med 61:249–263.
- Dunsworth HM, Warrener AG, Deacon T, Ellison PT, Pontzer H. 2012. Metabolic hypothesis for human altriciality. Proc Natl Acad Sci USA 109:15212–15216.
- Duran-Tauleria E, Rona RJ, Chinn S. 1995. Factors associated with weight for height and skinfold thickness in British children. J Epidemiol Community Heal 49:466– 473.
- Durbin J, Watson G. 1951. Testing for serial correlation in least squares regression. Biometrika 38:159–177.
- Durnin J, Rahaman M. 1967. The assessment of the amount of fat in the human body from measurements of skinfold thickness. Br J Nutr 21:681–689. A
- Durston S, Hulshoff H, Casey B, Giedd J, Buitelaar J, van Engeland H. 2001. Anatomical MRI of the developing human brain: What have we learned? J Am Acad Child Adolesc Psychiatry 40:1012–1020.
- Ebbers T, Wigström L, Bolger AF, Engvall J, Karlsson M. 2001. Estimation of relative cardiovascular pressures using time-resolved three dimensional phase contrast {MRI}. Magn Reson Med 879:872–879.
- Ellis BJ. 2004. Timing of Pubertal Maturation in Girls: An Integrated Life History Approach. Psychol Bull 130:920–958.
- Ellison PT. 1994. Advances in Human Reproductive Ecology. Annu Rev Anthropol 23:255–275.
- Ellison PT. 2001. On fertile ground: A natural history of human reproduction. Cambridge: Harvard University Press.
- Ellison PT, Reiches MW, Shattuck-Faegre H, Breakey A, Konecna M, Urlacher S, Wobber V. 2012. Puberty as a life history transition. Ann Hum Biol 39:352–60.
- Farr A, Chervenak FA, McCullough LB, Baergen RN, Grünebaum A. 2017. Human placentophagy: a review. Am J Obstet Gynecol 218:401.e1-401.e11.
- Faulkner KG, Cummings SR, Black D, Palermo L, Gluer C-C, Genant H. 1993. Simple measurement of femoral geometry predicts hip fracture: The study of osteoporotic fractures. J Bone Miner Res 8:1211–1217.

- Faulkner KG, Wacker WK, Barden HS, Simonelli C, Burke PK, Ragi S, Del Rio L. 2006. Femur strength index predicts hip fracture independent of bone density and hip axis length. Osteoporos Int 17:593–599.
- Fewtrell MS, Williams JE, Singhal A, Murgatroyd PR, Fuller N, Lucas A. 2009. Early diet and peak bone mass: 20 year follow-up of a randomized trial of early diet in infants born preterm. Bone 45:142–149.
- Field A. 2009. Discovering statistics using SPSS: And sex and drugs and rock'n'roll. Third edit. London: Sage.
- Fischer B, Mitteroecker P. 2015. Covariation between human pelvis shape, stature, and head size alleviates the obstetric dilemma. Proc Natl Acad Sci 112:201420325.
- Fischer B, Mitteroecker P. 2017. Allometry and sexual Dimorphism in the human pelvis. Anat Rec 300:698–705.
- Flecker H. 1932. Roentgenographic Observations of the Times of Appearance of Epiphyses and their Fusion with the Diaphyses. J Anat 67:118–164.
- Flores I, Sánchez M. 2007. Dos casos de embarazos a término con evidencias de distocia procedentes de contextos arqueológicos de época medieval y moderna. Paleopatología 4:1–10.
- Frazer G, Perkins N, Embertson R. 1999. Normal parturition and evaluation of the mare in dystocia. Equine Vet J 11:41–46.
- Freedman E. 1934. Os acetabuli. J bone Jt Surg 31:492–495.
- Freeman J V, Cole TJ, Chinn S, Jones PR, White EM, Preece M a. 1995. Cross sectional stature and weight reference curves for the UK, 1990. Arch Dis Child 73:17–24.
- Frisch RE. 1985. Fatness, menarche, and female fertility. Perspect Biol Med 28:611–633.
- Frisch RE. 1990. Body fat, menarche, fitness and fertility. In: Frisch RE, editor. Adipose tissue and reproduction. Basel: Karger. p 1–26.
- Frisch RE, McArthur JW. 1974. Menstrual cycles: fatness as a determinant of minimum weight for height necessary for their maintenance or onset. Science 185:949–951.
- Fuller NJ, Jebb S, Laskey M, Coward W, Elia M. 1992. Four-component model for the assessment of body composition in humans: comparison with alternative methods, and evaluation of the density and hydration of fat-free mass. Clin Sci 82:687–693.

- Gabbe S, Niebyl J, Simpson J eds. 2002. Obstetrics: Normal and Problem Pregnancies. Fourth edi. New York: Churchill Livingstone.
- Gale CR, Javaid MK, Robinson SM, Law CM, Godfrey KM, Cooper C. 2007. Maternal size in pregnancy and body composition in children. J Clin Endocrinol Metab 92:3904–3911.
- Galton F. 1886. Regression Towards Mediocrity in Hereditary Stature. J Anthropol Inst Gt Britain Irel 15:246–263.
- Gatehouse PD, Keegan J, Crowe LA, Masood S, Mohiaddin RH, Kreitner KF, Firmin DN. 2005. Applications of phase-contrast flow and velocity imaging in cardiovascular MRI. Eur Radiol 15:2172–2184.
- Geber J. 2014. Skeletal manifestations of stress in child victims of the Great Irish Famine (1845-1852): Prevalence of enamel hypoplasia, Harris lines, and growth retardation. Am J Phys Anthropol 155:149–161.
- Gilles B, Moccozet L, Magnenat-Thalmann N. 2006. Anatomical modelling of the musculoskeletal system from MRI. Med image Comput Comput Interv:289–296.
- Giuffra V, Vitiello A, Caramella D, Fornaciari A, Giustini D, Fornaciari G. 2015. Rickets in a High Social Class of Renaissance Italy: The Medici Children. Int J Osteoarchaeol 25:608–624.
- Giustina A, Mazziotti G, Canalis E. 2008. Growth hormone, insulin-like growth factors, and the skeleton. Endocr Rev 29:535–559.
- Gohlke B, Khadilkar V, Skuse D, Stanhope R. 1998. Recognition of children with psychosocial short stature: A spectrum of presentation. J Pediatr Endocrinol Metab 11:509–517.
- Gold GE, Han E, Stainsby J, Wright G, Brittain J, Beaulieu C. 2004. Musculoskeletal MRI at 3.0 T: Am J Roentgenol:343–351.
- Goodman AH, Leatherman T eds. 1998. Building a new biocultural synthesis: Political-economic perspectives on human biology. University of Michigan Press.
- Gould S, Lewontin R. 1979. The Spandrels of San Marco and the Panglossian Paradigm: A Critique of the Adaptationist Programme. Proc R Soc London 205:581–598.
- Grabowski M, Roseman CC. 2015. Complex and changing patterns of natural selection explain the evolution of the human hip. J Hum Evol 85:94–110.
- Grabowski MW. 2013. Hominin Obstetrics and the Evolution of Constraints. Evol Biol 40:57–75.

- Grabowski MW, Polk JD, Roseman CC. 2011. Divergent patterns of integration and reduced constraint in the human hip and the origins of bipedalism. Evolution (N Y) 65:1336–1356.
- Greulich WW, Thoms H. 1944. The Growth and Development of the Pelvis of Individual Girls Before, During, and After Puberty. Yale J Biol Med 17:91–98.8.
- Griffiths C, Gately P, Marchant PR, Cooke CB. 2012. Cross-sectional comparisons of BMI and waist circumference in british children: Mixed public health messages. Obesity 20:1258–1260.
- Grossman R. 2017. Are human heads getting larger? Proc Natl Acad Sci 114:201620160.
- Grunspan DZ, Nesse RM, Barnes ME, Brownell SE. 2018. Core principles of evolutionary medicine A Delphi study. Evol Med Public Heal:13–23.
- Gruss LT, Schmitt D. 2015. The evolution of the human pelvis: changing adaptations to bipedalism, obstetrics and thermoregulation. Philos Trans R Soc B Biol Sci 370:1–13.
- Gudmundsson S, Henningsson AC, Lindqvist P. 2005. Correlation of birth injury with maternal height and birthweight. BJOG An Int J Obstet Gynaecol 112:764–767.
- Gunnell DJ, Smith GD, Frankel SJ, Kemp M, Peters TJ. 1998. Socio-economic and dietary influences on leg length and trunk length in childhood: A reanalysis of the Carnegie (Boyd Orr) survey of diet and health in prewar Britain (1937-39). Paediatr Perinat Epidemiol 12:96–113.
- Hack M, Schluchter M, Cartar L, Rahman M, Cuttler L, Borawski E. 2003. Growth of Very Low Birth Weight Infants to Age 20 Years. Pediatrics 112:e30–e38.
- Hadjidakis DJ, Androulakis II. 2006. Bone remodeling. Ann N Y Acad Sci 1092:385– 396.
- Haile-Selassie Y, Latimer B, Alene M, Deino A, Gibert L, Melillo S, Saylor B, Lovejoy C. 2010. An early Australopithecus afarensis postcranium from Woranso-Mille, Ethiopia. Proc Natl Acad Sci 107:12121–12126.
- Hall-Martin AJ, Skinner JD. 1978. Observations on puberty and pregnancy in female giraffe (Giraffa camelopardalis). South African J Wildl Res 8:91–94.
- Hamada Y, Udono T. 2002. Longitudinal analysis of length growth in the chimpanzee (Pan troglodytes). Am J Phys Anthropol 118:268–284.
- Hamill V V, Roche F, Moore M. 1979. Physical Statistics growth: National center for Health statistics percentiles. Am J Clin Nutr 32:607–629.

- Handa V. 2003. Architectural differences in the bony pelvis of women with and without pelvic floor disorders. Obstet Gynecol 102:1283–1290.
- Hawkes K, O'Connell JF, Blurton Jones NG. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long postmenopausal life spans. Curr Anthropol 38:551–577.
- Hawkes K, O'Connell JF, Blurton Jones NG, Alvarez H, Charnov EL. 1998. Grandmothering, menopause, and the evolution of human life histories. Proc Natl Acad Sci 95:1336–1339.
- Hayden B. 1972. Population control among hunter/ gatherers. World Archaeol 4:205–221.
- Headland TN. 1989. Population Decline in a Philippine Negrito. Am J Hum Biol 72:59–72.

Healthcare GL. 2012. enCORE-based X-ray Bone Densitometer User Manual.

- Helle S, Brommer JE, Pettay JE, Lummaa V, Enbuske M, Jokela J, Helle S, Brommer JE, Pettay JE, Lummaa V, Enbuske M, Jokela J. 2014. Evolutionary demography of agricultural expansion in preindustrial northern Finland. Proc R Soc Med B Biol Sci 281:20141559–20141569.
- Henneberg M, Ulijaszek SJ. 2010. Body frame dimensions are related to obesity and fatness: Lean trunk size, skinfolds, and body mass index. Am J Hum Biol 22:83–91.
- Hermes R, Saragusty J, Schaftenaar W, Göritz F, Schmitt DL, Hildebrandt TB. 2008. Obstetrics in elephants. Theriogenology 70:131–144.
- Hertel N, Scheike T, Juul A, Main K, Holm K, Bach-Mortensen N, Skakkebaek N, Müller J. 1995. Body proportions of Danish children. Curves for sitting height ratio, subischial length and arm span. Ugeskr Laeger 157:6876–6881.
- Heymsfield SB, Wang Z, Baumgartner RN, Ross R. 1997. Human body composition: Advances in Models and Methods. Annu Rev Nutr 17:527–558.
- Hill K, Hurtado AM, Walker RS. 2007. High adult mortality among Hiwi huntergatherers: Implications for human evolution. J Hum Evol 52:443–454.
- Himes JH. 1989. Reliability of anthropometric methods and replicate measurements. Am J Phys Anthropol 79:77–80.
- Hochberg Z, Belsky J. 2013. Evo-devo of human adolescence: beyond disease models of early puberty. BMC Med 11:113.

- Hogan MC, Foreman KJ, Naghavi M, Ahn SY, Wang M, Makela SM, Lopez AD, Lozano R, Murray CJ. 2010. Maternal mortality for 181 countries, 1980-2008: A systematic analysis of progress towards Millennium Development Goal 5. Lancet 375:1609–1623.
- Hogberg U, Iregrent E, Siven CH, Diener L. 1987. Maternal deaths in medieval sweden: An osteological and life table analysis. J Biosoc Sci 19:495–503.
- Holick MF. 2004. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. Am J Clin Nutr 80:1678–1688.
- Holliday TW. 1997. Body proportions in Late Pleistocene Europe and modern human origins. J Hum Evol 32:423–48.
- Holliday TW. 2012. Body Size, Body Shape, and the Circumscription of the Genus Homo. Curr Anthropol 53:S330–S345.
- Hoppa RD. 1992. Evaluating Human Skeletal Growth: an Anglo-Saxon Example. Int J Osteoarchaeol 2:275–288.
- Hoppe C, Mølgaard C, Michaelsen KF. 2006. Cow's milk and linear growth in industrialized and developing countries. Annu Rev Nutr 26:131–173.
- Hricak H, Alpers C, Crooks L, Sheldon P. 1983. Magnetic resonance imaging of the female pelvis: Initial experience. Am J Roentgenol 141:1119–1128.
- Hromada J. 1939. Contribution to the study of the growth of the fetal pelvis. Anthropologie 18:129–170.
- Hu HH, Nayak KS, Goran MI. 2012. Assessment of abdominal adipose tissue and organ fat content by magnetic resonance imaging. Obes Rev 12:e504–e515.
- Huseynov A, Zollikofer CPE, Coudyzer W, Gascho D, Kellenberger C, Hinzpeter R, Ponce de León MS. 2016. Developmental evidence for obstetric adaptation of the human female pelvis. Proc Natl Acad Sci 113:5227–5232.
- Jack Jr C, Bernstein M, Fox N, Thompson P, Alexander G, Harvey D, Borowski B, Britson P, Whitwell J, Ward C, Dale A, Felmlee J, Gunter J, Hill D, Killiany R, Schuff N, Fox-Bosetti S, Lin C, Studholme C, DeCarli C, Krueger G, Ward H, Metzger G, Scott K, Mallozzi R, Blezek D, Levy J, Debbins J, Fleisher A, Albert M, Green R, Bartzokis G, Glover G, Mugler J, Weiner M. 2008. The Alzheimer's Disease neuroimaging initiative (ADNI): MRI Methods. J Magn Reson imaging 27:685–691.
- Jeffery N, Spoor F. 2002. Brain size and the human cranial base: A prenatal perspective. Am J Phys Anthropol 118:324–340.

Jolly A. 1973. Primate birth hour. Int Zoo Yearb 13:391–397.

- Jones G, Nguyen T, Sambrook P, Kelly P, Eisman J. 1994. Progressive loss of bone in the femoral neck in elderly people: Longitudinal findings from the Dubbo Osteoporosis Epidemiology Study. BMJ Br Med J 309:691–695.
- Juul A, Teilmann G, Scheike T, Hertel N, Holm K, Laursen E, Main K, Skakkebæk N, Hardy K, Leridon H, Aubert M, Herman-Giddens M, Ojeda S. 2006. Pubertal development in Danish children: Comparison of recent European and US data. Int J Androl 29:247–255.
- Kappleman J. 1996. The evolution of body mass and relative brain size in fossil hominids. J Hum Evol:1086–78712.
- Kaptoge S, Beck TJ, Reeve J, Stone KL, Hillier TA, Cauley JA, Cummings SR. 2008. Prediction of Incident Hip Fracture Risk by Femur Geometry Variables Measured by Hip Structural Analysis in the Study of Osteoporotic Fractures. J Bone Miner Res 23:1892–1904.
- Kavle JA, Flax VL, Abdelmegeid A, Salah F, Hafez S, Ramzy M, Hamed D, Saleh G, Galloway R. 2016. Factors associated with early growth in Egyptian infants: Implications for addressing the dual burden of malnutrition. Matern Child Nutr 12:139–151.
- Kelly TL, Wilson KE, Heymsfield SB. 2009. Dual energy X-ray absorptiometry body composition reference values from NHANES. PLoS One 4:2–9.
- Kennedy GC. 1953. The Role of Depot Fat in the Hypothalamic Control of Food Intake in the Rat. Proc R Soc London B Biol Sci 140:578–592.
- Kennedy K, Shepherd S, Williams JE, Ahmed SF, Wells JC, Fewtrell M. 2012. Activity, body composition and bone health in children. Arch Dis Child 98:204– 207.
- Kensara OA, Wootton SA, Phillips DI, Patel M, Jackson AA, Elia M. 2005b. Fetal programming of body composition: Relation between birth weight and body composition measured with dual-energy X-ray absorptiometry and anthropometric methods in older Englishmen. Am J Clin Nutr 82:980–987.
- Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Van Look PF. 2006. WHO analysis of causes of maternal death: A systematic review. Lancet 367:1066–1074.
- Khoo MMY, Tyler PA, Saifuddin A, Padhani AR. 2011. Diffusion-weighted imaging (DWI) in musculoskeletal MRI: A critical review. Skeletal Radiol 40:665–681.
- Kibii J, Churchill SE, Schmid P, Carlson K, Reed N, de Ruiter D, Berger LR. 2011. A partial pelvis of Australopithecus sediba. Science 525:1407–1412.

- Kimmel MC, Ferguson EH, Zerwas S, Bulik CM, Meltzer-Brody S. 2016. Obstetric and gynecologic problems associated with eating disorders. Int J Eat Disord 49:260–275.
- Kirchengast S, Hartmann B. 2003. Impact of maternal age and maternal somatic characteristics on newborn size. Am J Hum Biol 15:220–228.
- Knapen MHJ, Schurgers LJ, Vermeer C. 2007. Vitamin K2 supplementation improves hip bone geometry and bone strength indices in postmenopausal women. Osteoporos Int 18:963–972.
- Kristal MB, DiPirro JM, Thompson AC. 2012. Placentophagia in Humans and Nonhuman Mammals: Causes and Consequences. Ecol Food Nutr 51:177–197.
- Krogman W. 1951. The scars of human evolution. Sci Am 185:54–57.
- Kuller LH, Shemanski L, Manolio T, Haan M, Fried L, Bryan N, Burke GL, Tracy R, Bhadelia R. 1998. Relationship between ApoE, MRI findings and cognitive function in the Cardiovascular Health Study. Stroke 29:388–398.
- Kurki HK. 2005. Adaptive allometric modeling of the pelvis in small-bodied Later Stone Age (Holocene) foragers from Southern Africa.
- Kurki HK. 2007. Protection of obstetric dimensions in a small-bodied human sample. Am J Phys Anthropol 1165:1152–1165.
- Kurki HK. 2011. Compromised skeletal growth? Small body size and clinical contraction thresholds for the female pelvic canal. Int J Paleopathol 1:138–149.
- Kurki HK. 2013. Skeletal variability in the pelvis and limb skeleton of humans: does stabilizing selection limit female pelvic variation? Am J Hum Biol 25:795–802.
- Kurki HK, Decrausaz SL. 2016. Shape variation in the human pelvis and limb skeleton: Implications for obstetric adaptation. Am J Phys Anthropol 159:630–638.
- Kwok O, Underhill AT, Berry JW, Luo W, Elliott TR, Yoon M. 2008. Analysing longitudinal data with multilevel models: An example with individuals living with lower extremity intra-articular fractures. Rehabil Psychol 53:370–386.
- Kwong KK, Belliveau JW, Chesler DA, Goldberg IE, Weisskoff RM, Poncelet BP, Kennedy DN, Hoppel BE, Cohen MS, Turner R, Cheng H, Brady TJ, Rosen BR. 1992. Dynamic magnetic resonance imaging of human brain activity during primary sensory stimulation. Proc Natl Acad Sci 89:5675–5679.
- Lambrinoudaki I, Georgiou E, Douskas G, Tsekes G, Kyriakidis M, Proukakis C. 1998. Body composition assessment by dual-energy x-ray absorptiometry: Comparison of prone and supine measurements. Metabolism 47:1379–1382.

- Lande R. 1980. Sexual dimorphism, sexual selection, and adaptation in polygenic characters. Evolution (N Y) 34:292–305.
- Larsen CS. 1994. In the wake of Columbus: Native population biology in the postcontact Americas. Am J Phys Anthropol 37:109–154.
- Larsen CS. 1995. Biological Changes in Human Populations with Agriculture. Annu Rev Anthropol 24:185–213.
- Larsen CS. 2006. The agricultural revolution as environmental catastrophe: Implications for health and lifestyle in the Holocene. Quat Int 150:12–20.
- Larsen SC, Ruff C. 2011. "An external agency of considerable importance": The stresses of agriculture in the foraging-to-farming transition in Eastern North America. In: Pinhasi R, Stock JT, editors. Human bioarchaeology of transition to agriculture. First. Chichester: John Wiley & Sons Inc. p 293–317.
- Lauterbur PC. 1973. Image formation by induced local interactions: Examples employing nuclear magnetic resonance. Nature 242:190–191.
- Leutenegger W. 1973. Functional aspects of pelvic morphology in simian Primates. J Hum Evol 3:207–222.
- Leutenegger W. 1987. Neonatal brain size and neurocranial dimensions in Pliocene hominids: implications for obstetrics. J Hum Evol 16:291–296.

Levine D. 2006. Obstetric MRI. J Magn Reson Imaging 24:1–15.

- Lewis ME. 2002. Impact of ondustrialization: Comparative study of child health in four sites from Medieval and Postmedieval England (A.D. 850–1859). Am J Phys Anthropol 119:211–223.
- Lieverse AR, Bazaliiskii VI, Weber AW. 2015. Death by twins: a remarkable case of dystocic childbirth in Early Neolithic Siberia. Antiquity 89:23–38.
- Lindburg DG. 1982. Primate obstetrics: The biology of birth. Am J Primatol 3:193– 199.
- Lindgren G, Strandell A, Cole T, Healy M, Tanner J. 1995. Swedish population reference standards for height, weight and body mass index attained at 6 to 16 years (girls) or 19 years (boys). Acta Paediatr 84:1019–1028.
- Liselele HB, Boulvain M, Tshibangu KC, Meuris S. 2000. Maternal height and external pelvimetry to predict cephalopelvic disproportion in nulliparous African women: a cohort study. BJOG 107:947–952.
- Liston M, Papadopoulos J. 2004. The "rich Athenian lady" was pregnant: The anthropology of a geometric tomb reconsidered. Hesperia 73:7–38.

- Liston W. 2003. Rising caesarean section rates: can evolution and ecology explain some of the difficulties of modern childbirth? J R Soc Med 96:559–561.
- Livingston F. 1958. Anthropological implications of sickle cell gene distribution in West Africa. Am Anthropol 60:533–562.
- Lloyd T, Petit MA, Lin H-M, Beck TJ. 2004. Lifestyle factors and the development of bone mass and bone strength in young women. J Pediatr 144:776–782.
- Lloyd T, Taylor D, Lin H, Matthews A, Eggli D, Legro R. 2000. Oral contraceptive use by teenage women does not affect peak bone mass: a longitudinal study. Fertil Steril 74:734–738.
- Loder RT. 2002. The long-term effect of pelvic osteotomy on birth canal size. Arch Orthop Trauma Surg 122:29–34.
- Lovejoy CO. 1988. Evolution of Human Walking. Sci Am 259:118-125.
- Lovejoy CO. 2005. The natural history of human gait and posture. Part 2. Hip and thigh. Gait Posture 21:113–24.
- Lovejoy CO, Suwa G, Spurlock L, Asfaw B, White TD. 2009. The pelvis and femur of Ardipithecus ramidus: The emergence of upright walking. Science 326:71–77.
- Lucas A, Fewtrell M, Cole T. 1999. Fetal origins of adult disease the hypothesis revisited. BMJ Br Med J 319:245–249.
- Mace R. 2000. Evolutionary ecology of human life history. Anim Behav 59:1–10.
- Mace R, Sear R. 2005. Grandmotherhood: The evolutionary significance of the second half of female life. In: Voland E, Chasiotis A, Schiefenhoevel W, editors. Grandmotherhood - The evolutinary significance of the second half of female life. Piscataway: Rutgers University Press. p 143–159.
- Macintosh AA, Davies TG, Pinhasi R, Stock JT. 2015. Declining tibial curvature parallels ~6150 years of decreasing mobility in central european agriculturalists. Am J Phys Anthropol 157(2):260-275.
- Macintosh AA, Pinhasi R, Stock JT. 2014a. Lower limb skeletal biomechanics track long-term decline in mobility across ~6150 years of agriculture in Central Europe. J Archaeol Sci 52:376–390.
- Macintosh AA, Pinhasi R, Stock JT. 2014b. Divergence in male and female manipulative behaviors with the ntensification of metallurgy in Central Europe. PLoS One [Internet] 9:e112116.
- Maes HH, Neale MC, Eaves LJ. 1997. Genetic and Environmental Factors in Relative Body Weight and Human Adiposity. Behav Genet 27:325–348.

- Mahmood TA, Campbell DM, Wilson AW. 1988. Maternal height, shoe size, and outcome of labour in white primigravidas: a prospective anthropometric study. BMJ Br Med J 297:515–517.
- Malgosa A, Alesan A, Safont S, Ballbé M, Ayala MM. 2004. A dystocic childbirth in the Spanish Bronze Age. Int J Osteoarchaeol 14:98–103.
- Mamidi RS, Kulkarni B, Singh A. 2011. Secular trends in height in different states of India in relation to socioeconomic characteristics and dietary intakes. Food Nutr Bull 32:23–34.
- Manning W, Li W, Edelman R. 1993. A preliminary report comparing magnetic resonance coronary angiography with conventional angiography. N Engl J Med 328:828–832.
- Marciani L. 2011. Assessment of gastrointestinal motor functions by MRI: A comprehensive review. Neurogastroenterol Motil 23:399–407.
- Marphatia AA, Cole TJ, Grijalva-Eternod C, Wells JCK. 2016. Associations of gender inequality with child malnutrition and mortality across 96 countries. Glob Heal Epidemiol Genomics 1:e6.
- Marshall WA. 1978. The relationship of puberty to other maturity indicators and body composition in man. J Reprod Fertil 52:437–443.
- Marshall WA, Tanner JM. 1969. Variations in the pattern of pubertal changes in girls. Arch Dis Child:291–303.
- Marshall WA, Tanner JM. 1970. Variations in the pattern of pubertal changes in boys. Arch Dis Child:13–23.
- Martini FH, Timmons MJ, Tallitsch RB. 2009. Human Anatomy. Sixth. San Francisco: Pearson.
- Martorell R, Habicht J-P, Yarbrough C, Guzmán G, Klein RE. 1975. The identification and evaluation of measurement variability in the anthropometry of preschool children. Am J Phys Anthropol 43:347–352.
- Martorell R, Zongrone A. 2012. Intergenerational influences on child growth and undernutrition. Paediatr Perinat Epidemiol 26 Suppl 1:302–14.
- Mays S, Brickley M, Ives R. 2006a. Skeletal manifestation of rickets in infants and young children in a historic population from England. Am J Phys Anthropol 129:362–374.

- Mays S, Ives R, Brickley M. 2009. The effects of socioeconomic status on endochondral and appositional bone growth, and acquisition of cortical bone in children from 19th century Birmingham, England. Am J Phys Anthropol 140:410– 416.
- Mays S, Lees B, Stevenson J. 1998. Age-dependent bone loss in the femur in a medieval population. Int J Osteoarch 8(2):97–106.
- Mays S, Turner-Walker G, Syversen U. 2006b. Osteoporosis in a population from Medieval Norway. Am J Phys Anthropol 131:343–351.
- Mazness R, Barden H, Bisek J, Hanson J. 1990. Dual-energy x-ray absorptiometry for total-body and regional bone-mineral and soft-tossue composition. Am J Clin Nutr 51:1106–1112.
- McFadden C, Oxenham MF. 2019. The Paleodemographic Measure of Maternal Mortality and a Multifaceted Approach to Maternal Health. Curr Anthropol 60:000–000.
- McHenry H. 1982. The pattern of human evolution: Studies on bipedalism, mastication and encephalization. Annu Rev Anthropol 11:151–173.
- McHenry HM, Coffing K. 2000. Australopithecus to Homo: Transformations in body and mind. Annu Rev Anthropol 29:125–146.
- Meindl RS, Lovejoy CO, Mensforth RP, Don Carlos L. 1985. Accuracy and direction of error in the sexing of the skeleton: implications for paleodemography. Am J Phys Anthropol 68:79–85.
- de Melo NR, Aldrighi JM, Faggion D, Reyes VROY, Souza JB, Fernandes CE, Larson E. 2004. A prospective open-label study to evaluate the effects of the oral contraceptive Harmonet (gestodene75/EE20) on body fat. Contraception 70:65– 71.
- Merritt CE. 2014. The influence of body size on adult skeletal age estimation methods.
- Merritt CE. 2017. Inaccuracy and bias in adult skeletal age estimation: Assessing the reliability of eight methods on individuals of varying body sizes. Forensic Sci Int 275:315.e1-315.e11.
- Michel SC a, Rake A, Treiber K, Seifert B, Chaoui R, Huch R, Marincek B, Kubik-Huch R a. 2002. MR obstetric pelvimetry: effect of birthing position on pelvic bony dimensions. Am J Roentgenol 179:1063–1067.
- Miles AEW, Bulman JS. 1994. Growth curves of immature bones from a Scottish island population of sixteenth to mid-nineteenth century: Limb-bone diaphyses and some bones of the hand and foot. Int J Osteoarchaeol 4:121–136.

- Miller JM, Brandon C, Jacobson J a, Low LK, Zielinski R, Ashton-Miller J, Delancey JOL. 2010. MRI findings in patients considered high risk for pelvic floor injury studied serially after vaginal childbirth. Am J Roentgenol 195:786–791.
- Mitchell G, Brandt E. 1975. Behavior of the female rhesus monkey during birth. In: Bourne G, editor. The Rhesus Monkey Volume 11. Management, Reproduction, and Pathology. New York: New York Academic Press. p 231–244.
- Mitteroecker P. 2018. How human bodies are evolving in modern societies. Nat Ecol Evol.
- Mitteroecker P, Fischer B. 2016. Adult pelvic shape change is an evolutionary side effect. Proc Natl Acad Sci 113:E3596–E3596.
- Mitteroecker P, Huttegger SM, Fischer B, Pavlicev M. 2016. Cliff-edge model of obstetric selection in humans. Proc Natl Acad Sci113:14680–14685.
- Mitteroecker P, Huttegger SM, Fischer B, Pavlicev M. 2017. Reply to Grossman: The role of natural selection for the increase of Caesarean section rates. Proc Natl Acad Sci114:E1305–E1305.
- Moerman M. 1982. Growth of the birth canal in adolescent girls. Am J Obstet Gynecol 143:528–532.
- Moore F. 1947. Determination of total body water and solids with isotopes. Science 104:157–160.
- Moses R, Luebcke M, Davis W, Coleman K, Tapsell L, Petocz P, Brand-Miller J. 2006. Effect of a low-glycemic-index diet during pregnancy on obstetric outcomes. Am J Clin Nutr 84:807–812.
- Motil KJ, Grand RJ, Davis-Kraft L, Ferlic LL, Smith EO. 1993. Growth failure in children with inflammatory bowel disease: a prospective study. Gastroenterology 105:681–91.
- Nash LT. 1974. Parturition in feral baboon (Papio anubis). Primates 15:279–285.
- National Office of Statistics. 2000. Standard Occupational Classification 2000 (SOC2000).
- Nelson M, Hague GF, Cooper C, Bunker VW. 1988. Calcium intake in the elderly: validation of a dietary questionnaire. J Hum Nutr Diet 1:115–127.
- Ness AR, Leary SD, Mattocks C, Blair SN, Reilly JJ, Wells J, Ingle S, Tilling K, Smith GD, Riddoch C. 2007. Objectively measured physical activity and fat mass in a large cohort of children. PLoS Med 4:476–484.

Nesse RM. 2001. How is Darwinian medicine useful? West J Med 174:358-60.

- Newman SL, Gowland RL. 2017. Dedicated followers of fashion? Bioarchaeological perspectives on socio-economic status, inequality, and sealth in urban children from the industrial revolution (18th–19th C), England. Int J Osteoarchaeol 27:217–229.
- Nicolson A, Hanley C. 1953. Indices of physiological maturity: derivation and interrelationships. Child Dev 24:3–38.
- Norgan N. 1997. The beneficial effects of body fat and adipose tissue in humans. Int J Obes 21:738–746.
- Novotny R, Davis J, Wasnich R, Biernacke I, Onaka A. 2000. Maternal pelvic size, measured by dual energy X-ray absorptiometry, predicts infant birthweight. Am J Hum Biol 12:552–557.
- Ogden CL, Kuczmarski RJ, Flegal KM, Mei Z, Guo S, Wei R, Grummer-Strawn LM, Curtin LR, Roche AF, Johnson CL. 2002. Centers for Disease Control and Prevention 2000 Growth Charts for the United States: Improvements to the 1977 National Center for Health Statistics Version. Pediatrics 109:45–60.
- Ogle GD, Allen JR, Humphries IRJ, Lu PW, Briody JN, Morley K, Howman-Giles R, Cowell CT. 1995. Body-composition assessment by dual-energy x-ray absorptiometry in subjects aged 4-26 y. Am J Clin Nutr 61:746–753.
- Ong KK, Northstone K, Wells JCK, Rubin C, Ness AR, Golding J, Dunger DB. 2007. Earlier mother's age at menarche predicts rapid infancy growth and childhood obesity. PLoS Med 4:737–742.
- Owsley DW, Bradtmiller B. 1983. Mortality of pregnant females in Arikara villages: Osteological evidence. Am J Phys Anthropol 61:331–336.
- Paley MR, Ros PR. 1997. MRI of the gastrointestinal tract. Eur Radiol 7:1387–1397.
- Pan H, Cole TJ. 2010. LMS Chartmaker Light, a program to construct growth references using the LMS method.
- Parente RCM, Bergqvist LP, Soares MB, Filho OBM. 2011. The history of vaginal birth. Arch Gynecol Obstet 284:1–11.
- Parsons TJ, Power C, Manor O. 2005. Physical activity, television viewing and body mass index: A cross-sectional analysis from childhood to adulthood in the 1958 British cohort. Int J Obes 29:1212–1221.
- Pearson K. 1896. Mathematical Contributions to the Theory of Evolution. III. Regression, Heredity, and Panmixia. Philos Trans R Soc A Math Phys Eng Sci 187:253–318.

- Pedersen CA, Boccia ML. 2002. Oxytocin links mothering received, mothering bestowed and adult stress responses. Stress 5:259–267.
- Perez-Lopez F, Chedraui P, Cuadros-Lopez J. 2010. Bone Mass Gain During Puberty and Adolescence: Deconstructing Gender Characteristics. Curr Med Chem 17:453–466.
- Pérez JR, Martín AG. 2007. A multidisciplinary approach reveals an extraordinary double inhumation in the osteoarchaeological record. J Taphon 1:91–101.
- Perini TA, de Oliveira GL, Ornelia JS, de Oliveira FP. 2005. Technical error of measurement in anthropometry. Rev Bras Med do Esporte 11:81–85.
- Perola M, Sammalisto S, Hiekkalinna T, Martin NG, Visscher PM, Montgomery GW, Benyamin B, Harris JR, Boomsma D, Willemsen G, Hottenga JJ, Christensen K, Kyvik KO, Sørensen TIA, Pedersen NL, Magnusson PKE, Spector TD, Widen E, Silventoinen K, Kaprio J, Palotie A, Peltonen L. 2007. Combined genome scans for body stature in 6,602 European twins: Evidence for common caucasian loci. PLoS Genet 3:1019–1028.
- Pfeiffer S. 2011. Pelvic stress injuries in a small-bodied forager. Int J Osteoarchaeol 21:694–703.
- Pfeiffer S, Doyle LE, Kurki HK, Harrington L, Ginter JK, Merritt CE. 2014. Discernment of mortality risk associated with childbirth in archaeologically derived forager skeletons. Int J Paleopathol 7:15–24. A
- Pierce MB, Leon DA. 2005. Age at menarche and adult BMI in the Aberdeen Children of the 1950s Cohort Study. Am J Clin Nutr 82:733–739.
- Piga G, Guirguis M, Thompson TJU, Isidro A, Enzo S, Malgosa A. 2016. A case of semi-combusted pregnant female in the Phoenician-Punic necropolis of Monte Sirai (Carbonia, Sardinia, Italy). HOMO- J Comp Hum Biol 67:50–64.
- Pomeroy E, Macintosh A, Wells JCK, Cole TJ, Stock JT. 2018. Relationship between body mass, lean mass, fat mass, and limb bone cross-sectional geometry: Implications for estimating body mass and physique from the skeleton. Am J Phys Anthropol 166:59–69.
- Pomeroy E, Stock JT. 2012. Estimation of stature and body mass from the skeleton among coastal and mid-altitude Andean populations. Am J Phys Anthropol 147:264–79.
- Pomeroy E, Stock JT, Stanojevic S, Miranda JJ, Cole TJ, Wells JCK. 2012. Tradeoffs in relative limb length among Peruvian children: Extending the thrifty phenotype hypothesis to limb proportions. PLoS One 7:e51795.

- Pomeroy E, Wells JCK, Cole TJ, O'Callaghan M, Stock JT. 2015a. Relationships of maternal and paternal anthropometry with neonatal body size, proportions and adiposity in an Australian cohort. Am J Phys Anthropol 156:625–636.
- Pomeroy E, Wells JCK, Stanojevic S, Miranda JJ, Moore LG, Cole TJ, Stock JT. 2015b. Surname-Inferred andean ancestry is associated with child stature and limb lengths at high altitude in Peru, but not at sea level. Am J Hum Biol 27:798– 806.
- Ponce de León MS, Huseynov A, Zollikofer CPE. 2016. Reply to Mitteroecker and Fischer: Developmental solutions to the obstetrical dilemma are not Gouldian spandrels. Proc Natl Acad Sci 113:E3597–E3598.
- Prader A. 1984. Biomedical and endocrinological aspects of normal growth and development. In: Borms J, Hauspie R, Sand A, Hebbelinck M, editors. Human growth and development. New York: Plenum. p 1–22.
- Prasad M, Al-Taher H. 2002. Maternal height and labour outcome. J Obstet Gynaecol (Lahore) 22:513–515.
- Prather H, Hunt D, McKeon K, Simpson S, Meyer EB, Yemm T, Brophy R. 2016. Are elite female soccer athletes at risk for disordered eating attitudes, menstrual dysfunction, and stress fractures? PM R 8:208–213.
- Preece M. 1986. Prepubertal and pubertal endrocrinology. In: Faulkner F, Tanner J, editors. Human Growth Volume II. Second edi. New York: Plenum. p 211–224.
- Preece M, Cameron N, Donnall M, Dunger DB, Holder A, Taylor A. 1984. The endocrinology of male puberty. In: Borms J, Hauspie R, Sand A, Hebbelinck M, editors. Human growth and development. New York: Plenum. p 23–37.
- Prentice A. 2003. Micronutrients and the bone mineral content of the mother, fetus and newborn. J Nutr 133:1693S–1699S.
- Prentice A, Prentice A. 1988. Energy costs of lactation. Annu Rev Nutr 8:63-79.
- Prynne CJ, Mishra GD, O'Connell M a, Muniz G, Laskey MA, Yan L, Prentice A, Ginty F. 2006. Fruit and vegetable intakes and bone mineral status: a cross sectional study in 5 age and sex cohorts. Am J Clin Nutr 83:1420–1428.
- Rauch F, Bailey DA, Baxter-Jones A, Mirwald R, Faulkner R. 2004. The "musclebone unit" during the pubertal growth spurt. Bone 34:771–775.
- Raxter MH, Auerbach BM, Ruff CB. 2006. Revision of the Fully technique for estimating statures. Am J Phys Anthropol 130:374–384.

- Reubinoff BE, Grubstein A, Meirow D, Berry E, Schenker JG, Brzezinski A. 1995. Effects of low-dose estrogen oral contraceptives on weight, body composition, and fat distribution in young women. Fertility Steril 63:516–521.
- Reynolds E. 1947. The bony pelvis in prepuberal childhood. Am J Phys Anthropol 5:165–200.
- Reynolds E, Wines J. 1948. Individual differences in physical changes associated with adolescence in girls. Am J Dis Child 75:329–350.
- Riches D. 1974. The Netsilik Eskimo: A Special Case of Selective Female Infanticide. Ethnology 13:351–361.
- Riddoch CJ, Leary SD, Ness AR, Blair SN, Deere K, Mattocks C, Griffiths A, Davey Smith G, Tilling K. 2009. Prospective associations between objective measures of physical activity and fat mass in 12-14 year old children: the Avon Longitudinal Study of Parents and Children (ALSPAC). Bmj 339:b4544–b4544.
- Risser WL, Gefter LR, Barratt MS, Risser JMH. 1999. Weight change in adolescents who used hormonal contraception. J Adolesc Heal 24:433–436.
- Rizk DEE, Czechowski J, Ekelund L. 2004. Dynamic assessment of pelvic floor and bony pelvis morphologic condition with the use of magnetic resonance imaging in a multiethnic, nulliparous, and healthy female population. Am J Obstet Gynecol 191:83–89.
- Robinson PD, Högler W, Craig ME, Verge CF, Walker JL, Piper a C, Woodhead HJ, Cowell CT, Ambler GR. 2006. The re-emerging burden of rickets: A decade of experience from Sydney. Arch Dis Child 91:564–568.
- Roff D. 1992. The evolution of life histories: Theory and analysis. New York: Chapman & Hall.
- Rogol AD, Roemmich JN, Clark PA. 2002. Growth at puberty. J Adolesc Heal 31:192–200.
- Roseman CC, Auerbach BM. 2015. Ecogeography, genetics, and the evolution of human body form. J Hum Evol 78:80–90.
- Rosenberg K. 1992. The evolution of modern human childbirth. Yearb Phys Anthropol 35:89–124.
- Rosenberg K.R. 2007. Neandertal Pelvic Remains from Krapina: Peculiar or Primitive? Period Biol 109:387–392.
- Rosenberg K, Trevathan W. 1995. Bipedalism and human birth: The obstetrical dilemma revisited. Evol Anthropol 4:161–168.

- Rosenberg KR, Trevathan WR. 2018. Evolutionary perspectives on cesarean section. Ev Med Public Health 1:67–81.
- Rosset A, Spadola L, Ratib O. 2004. OsiriX: An open-source software for navigating in multidimensional DICOM images. J Digit Imaging 17:205–216.
- Roy DK, Berry JL, Pye SR, Adams JE, Swarbrick CM, King Y, Silman AJ, O'Neill TW. 2007. Vitamin D status and bone mass in UK South Asian women. Bone 40:200–204.
- Ruff C. 2002. Variation in Human Body Size and Shape. Annu Rev Anthropol 31:211–232.
- Ruff C. 2010. Body size and body shape in early hominins implications of the Gona Pelvis. J Hum Evol 58:166–178.
- Ruff C. 2017. Mechanical Constraints on the hominin pelvis and the obstetrical dilemma? Anat Rec 300:946–955.
- Ruff C, Niskanen M, Junno J-A, Jamison P. 2005. Body mass prediction from stature and bi-iliac breadth in two high latitude populations, with application to earlier higher latitude humans. J Hum Evol 48:381–392.
- Ruff CB. 1991. Climate and body shape in hominid evolution. J Hum Evol 21:81–105.
- Ruff CB. 1994. Morphological adaptation to climate in modern and fossil hominids. Am J Phys Anthropol 37:65–107.
- Ruff CB. 1995. Biomechanics of the hip and birth in early Homo. Am J Phys Anthropol 98:527–574.
- Ruff CB. 2000. Body mass prediction from skeletal frame size in elite athletes. Am J Phys Anthropol 113:507–517.
- Ruff CB, Holt BM, Niskanen M, Sladék V, Berner M, Garofalo E, Garvin HM, Hora M, Maijanen H, Niinimäki S, Salo K, Schuplerová E, Tompkins D. 2012. Stature and body mass estimation from skeletal remains in the European Holocene. Am J Phys Anthropol 148:601–617.
- Ruff CB, Trinkaus E, Holliday TW. 1997. Body mass and encephalization in Pleistocene Homo. Nature 387:173–176.
- Rühli FJ. 2015. Short Review: Magnetic Resonance Imaging of Ancient Mummies. Anat Rec 298:1111–1115.
- Rühli FJ, von Waldburg H, Nielles-Vallespin S, Böni T, Speier P. 2007. Clinical Magnetic Resonance Imaging of Ancient Dry Human Mummies Without Rehydration. JAMA 298:2617.
- Runyan SM, Stadler DD, Bainbridge CN, Miller SC, Moyer-Mileur LJ. 2003. Familial resemblance of bone mineralization, calcium intake, and physical activity in early-adolescent daughters, their mothers, and maternal grandmothers. J Am Diet Assoc 103:1320–1325.
- Ruszkowski JT, Damadian R, Giambalvo A, Comes A, Hertz D, Lufkin R, Smith SD, Wortham D. 1986. MRI angiography of the carotid artery. Magn Reson Imaging 4:497–502.
- Satake T, Satake T, Malina R, Malina R, Tanaka S, Tanaka S. 1994. Individual Variation in the Sequence of Ages At Peak Velocity in 7 Body Dimensions. Am J Hum Biol 6:359–367.
- Sayer D, Dickinson SD. 2013. Reconsidering obstetric death and female fertility in Anglo-Saxon England. World Archaeol 45:285–297.
- Schattmann A, Bertrand B, Vatteoni S, Brickley M. 2016. Approaches to cooccurrence: Scurvy and rickets in infants and young children of 16-18th century Douai, France. Int J Paleopathol 12:63–75.
- Schneider JE, Wade GN. 1989. Availability of metabolic fuels controls estrous cyclicity of Syrian hamsters. Science 244:1326–1328.
- Schneider JE, Wade GN. 1990. Decreased availability of metabolic fuels induces anestrus in golden hamsters. Am J Physiol 258:R750-755.
- Schneider JE, Zhou D, Blum RM. 2000. Leptin and metabolic control of reproduction. Horm Behav 37:306–326.
- Scholl TO, Chen X, Khoo CS, Lenders C. 2004. The Dietary Glycemic Index during Pregnancy: Influence on Infant Birth Weight, Fetal Growth, and Biomarkers of Carbohydrate Metabolism. Am J Epidemiol 159:467–474.
- Schultz AH. 1949. Sex differences in the pelves of primates. Am J Phys Anthropol 7:401–423.
- Scott RM, Buckley HR. 2010. Biocultural interpretations of trauma in two prehistoric Pacific Island populations from Papua New Guinea and the Solomon Islands. Am J Phys Anthropol 142:509–518.
- Sear R, Mace R. 2008. Who keeps children alive? A review of the effects of kin on child survival. Evol Hum Behav 29:1–18.
- Seguí EL, Giménez PT, Muñoz JQ, Báñez M, Poveda C. 2005. La necrópolis islámica de L'Alfossar (Novelda, Alicante). Recer del Mus d'alcoi 14:143–156.

- Selander J, Cantor A, Young SM, Benyshek DC. 2013. Human maternal placentophagy: A survey of self-reported motivations and experiences associated with placenta consumption. Ecol Food Nutr 52:93–115.
- Shapiro SS, Wilk MB. 1965. An analysis of variance test for normality (complete samples). Biometrika 52:591–611.
- Sharafeldin MA, Ragab MT, Kandeel AA. 1971. Behaviour of ewes during parturition. J Agric Sci 76:419–422.
- Sharma K. 2002. Genetic basis of human female pelvic morphology: A twin study. Am J Phys Anthropol 117:327–333.
- Sharma K, Gupta P, Shandilya S. 2016. Age related changes in pelvis size among adolescent and adult females with reference to parturition from Naraingarh, Haryana (India). HOMO J Comp Hum Biol 67:273–293.
- Sharma K, Gupta S, Shandilya S, Chopra S. 2014. Age related change in pelvis size among adolescent and adult females from Naraingarh, Haryana (India).
- Shaw CN, Stock JT. 2013. Extreme mobility in the Late Pleistocene? Comparing limb biomechanics among fossil Homo, varsity athletes and Holocene foragers. J Hum Evol 64:242–249.
- Sheehy A, Gasser T, Molinari L, Largo RH. 1999. An analysis of variance of the pubertal and midgrowth spurts for length and width. Ann Hum Biol 26:309–331.
- Sheiner E, Levy A, Katz M, Mazor M. 2005. Short stature--an independent risk factor for Cesarean delivery. Eur J Obstet Gynecol Reprod Biol 120:175–178.
- Sherer DM, Sokolovski M, Dalloul M, Khoury-Collado F, Osho JA, Lamarque MD, Abulafia O. 2006. Fetal clavicle length throughout gestation: A nomogram. Ultrasound Obstet Gynecol 27:306–310.
- Shieh G. 2011. Clarifying the role of mean centring in multicollinearity of interaction effects. Br J Math Stat Psychol 64:462–477.
- Shirley MK, Arthurs OJ, Seunarine KK, Cole TJ, Eaton S, Williams JE, Clark CA, Wells JCK. 2018. Metabolic rate of major organs and tissues in young adult South Asian women. Eur J Clin Nutr
- Shrewsbury V, Wardle J. 2008. Socioeconomic status and adiposity in childhood: A systematic review of cross-sectional studies 1990-2005. Obesity 16:275–284.
- Shuler K a., Hodge SC, Danforth ME, Lynn Funkhouser J, Stantis C, Cook DN, Zeng P. 2012. In the shadow of Moundville: A bioarchaeological view of the transition to agriculture in the central Tombigbee valley of Alabama and Mississippi. J Anthropol Archaeol 31:586–603.

- Siek T. 2015. An exploration of tactile interaction in osteology and material culture. PlatForum 14:147–164.
- Silventoinen K, Sammalisto S, Perola M, Boomsma DI, Cornes BK, Davis C, Dunkel L, de Lange M, Harris JR, Hjelmborg JVB, Luciano M, Martin NG, Mortensen J, Nisticò L, Pedersen NL, Skytthe A, Spector TD, Stazi MA, Willemsen G, Kaprio J. 2003. Heritability of adult body height: A comparative study of twin cohorts in eight countries. Twin Res 6:399–408.
- Simmons K, Greulich WW. 1943. Menarcheal age and the height, weight, and skeletal age of girls age 7 to 17 years. J Pediatr 22:518–548.
- Simpson S, Quade J, Levin N, Butler R, Dupont-Nivet G, Everett M, Semaw S. 2008. A Female Homo erectus Pelvis from Gona, Ethiopia. Science (80-) 322:1089– 1092.
- Singh D, Young RK. 1995. Body weight, waist-to-hip ratio, breasts and hips: Role in judgements of female attractiveness and desirability for relationships. Ethol Sociobiol 16:483–507.
- Singhal A, Wells J, Cole TJ, Fewtrell M, Lucas A. 2003. Programming of lean body mass: a link between birth weight, obesity, and cardiovascular disease? Am J Clin Nutr 77:726–730.
- Slemenda C, Miller J, Hui S, Reister T, Johnston JR C. 1991. Role of physical activity in the development of skeletal mass in children. J Bone Miner Res 6:1227–1233.
- Smeltzer J. 1986. Prevention and management of shoulder dystocia. Clin Obstet Gynecol 29:299–308.
- Snijder MB, Visser M, Dekker JM, Seidell JC, Fuerst T, Tylavsky F, Cauley J, Lang T, Nevitt M, Harris TB. 2002. The prediction of visceral fat by dual-energy X-ray absorptiometry in the elderly: A comparison with computed tomography and anthropometry. Int J Obes 26:984–993.
- Souza JP, Gülmezoglu AM, Lumbiganon P, Laopaiboon M, Carroli G, Fawole B, Ruyan P. 2010. Caesarean section without medical indications is associated with an increased risk of adverse short- term maternal outcomes: the 2004-2008 WHO Global Survey on Maternal and Perinatal Health. BMC Med 8:71.
- Spörri S, Hänggi W, Braghetti A, Vock P, Schneider H. 1997. Pelvimetry by magnetic resonance imaging as a diagnosic tool to evaluate dystocia. Obstet Gynecol 89:902–908.
- Stark DD, McCarthy SM, Filly RA, Parer JT, Hricak H, Callen PW. 1985. Pelvimetry by magnetic resonance imaging. Am J Roentgenol 144:947–950.

Statistics O for N. 2010. 'Average' Briton highlighted on UN World Statistics Day.

Stearns S. 1992. The evolution of life histories. Oxford: Oxford University Press.

- Steckel RH, Rose JC, Larsen CS, Walker PL. 2002. Skeletal Health in the Western Hemisphere from 4000 B.C. to the Present. Evol Anthropol 11:142–155.
- Stevenson P. 1924. Age order of epiphyseal union in man. Am J Phys Anthropol 7:53–93.
- Stock JT. 2006. Hunter-gatherer postcranial robusticity relative to patterns of mobility, climatic adaptation, and selection for tissue economy. Am J Phys Anthropol 131:194–204.
- Stock JT, O'Neill MC, Ruff CB, Zabecki M, Shackelford L, Rose JC. 2011. Body size, skeletal biomechanics, mobility and habitual activity from the Late Palaeolithic to the Mid-Dynastic Nile Valley. In: Pinhasi R, Stock JT, editors. Human bioarchaeology of transition to agriculture. First. Chichester: John Wiley & Sons Inc. p 347–371.
- Strayer LM. 1943. The Embryology of the Human Hip Joint. Yale J Biol Med 16:13– 26.6.
- Strayer LM. 1971. The Embryology of the Human Hip Joint. Clin Orthop Relat Res 74:221–240.
- Student. 1908. The probable error of a mean. Biometrika 6:1–25.
- Stulp G, Verhulst S, Pollet T V., Nettle D, Buunk AP. 2011. Parental height differences predict the need for an emergency Caesarean section. PLoS One 6:1–8.
- Sze EH, Kohli N, Miklos JR, Roat T, Karram MM. 1999. Computed tomography comparison of bony pelvis dimensions between women with and without genital prolapse. Obstet Gynecol 93:229–232.
- Tague RG. 1992. Sexual dimorphism in the human bony pelvis, with a consideration of the Neandertal pelvis from Kebara Cave, Israel. Am J Phys Anthropol 88.
- Tague RG. 1994. Maternal mortality or prolonged growth: Age at death and pelvic size in three prehistoric Amerindian populations. Am J Phys Anthropol 95:27–40.
- Tague RG. 2000. Do big females have big pelves? Am J Phys Anthropol 112:377– 93.
- Tague RG. 2005. Big-bodied males help us recognize that females have big pelves. Am J Phys Anthropol 127:392–405.

- Tague RG, Lovejoy CO. 1986. The obstetric pelvis of A.L. 288-1 (Lucy). J Hum Evol 15:237–255.
- Tanner J. 1962. Growth and adolescence. 2nd ed. Oxford: Blackwell Scientific Publications.
- Tanner J. 1981. A history of the study of human growth. Cambridge: Cambridge University Press.
- Tanner J, Whitehouse R. 1976. Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. Arch Dis Child 51:170–179.
- Tanner J, Whitehouse R, Takaishi M. 1966. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. II. Arch Dis Child 41:454–471.
- Tanner JM. 1981b. Growth and maturation during adolescence. Nutr Rev 39:43-55.
- Tanner JM, Whitehouse RH. 1976b. Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. Arch Dis Child 51:170–179.
- Taouli B, Tolia AJ, Losada M, Babb JS, Chan ES, Bannan MA, Tobias H. 2007. Diffusion-weighted MRI for quantification of liver fibrosis: Preliminary experience. Am J Roentgenol 189:799–806.
- Taranger J, Engström I, Lichtenstein H, Svennberg- Redegren I. 1976. VI. Somatic pubertal development. Acta Paediatr Scand Suppl:121–135.
- The British Dietetic Association. 2017. Calcium. Food Fact Sheets [Internet]:2. Available from: www.bda.uk.com%3Epdf%3Eprovider
- Thoms H, Godfried MS. 1939. The interrelationships between fetal weight, size of pelvic inlet, and maternal height. Yale J Biol Med 11:355–362.
- Thomsen CR, Uldbjerg N, Hvidman L, Atladóttir HÓ, Henriksen TB, Milidou I. 2014. Seasonal variation of dystocia in a large Danish cohort. PLoS One 9:e94432-.
- To WWK, Wong MWN, Lam IYL. 2005. Bone mineral density differences between adolescent dancers and non-exercising adolescent females. J Pediatr Adolesc Gynecol 18:337–42.
- Tobolsky VA, Yegian A, Holowka N, Jemuutai J, Segei T, Ojiambo R, Lieberman DE. 2018. The role of energy status in determining childhood growth pattrns in western Kenya. In: American Association of Physical Anthropology. . p 276.

- Toh-Adam R, Srisupundit K, Tongsong T. 2012. Short stature as an independent risk factor for cephalopelvic disproportion in a country of relatively small-sized mothers. Arch Gynecol Obstet 285:1513–1516.
- Tollånes MC, Thompson JMD, Daltveit AK, Irgens LM. 2007. Cesarean section and maternal education; Secular trends in Norway, 1967-2004. Acta Obstet Gynecol Scand 86:840–848.
- Tóth GA, Molnár P, Suskovics C. 2012. Gender differences and secular trends in height, pattern of growth and maturation during puberty. Hum Biol Rev 1:16–21.
- Tovée MJ, Cornelissen PL. 2001. Female and male perceptions of female physical attractiveness in front-view and profile. Br J Psychol 92 Part 2:391–402.
- Towne B, Czerwinski SA, Demerath EW, Blangero J, Roche AF, Siervogel RM. 2005. Heritability of age at menarche in girls from the Fels Longitudinal Study. Am J Phys Anthropol 128:210–219.
- Trevathan W. 2015. Primate pelvic anatomy and implications for birth. Philos Trans R Soc B Biol Sci 370:20140065.
- Trevathan W, Rosenberg K. 2000. The shoulders follow the head: postcranial constraints on human childbirth. J Hum Evol 39:583–586.
- Trivers R. 1974. Parent-Offspring Conflict. Am Zool 14:249-264.
- Tukey J. 1949. Comparing individual means in the analysis of variance. Biometrics 5:99–114.
- Ulijaszek SJ, Johnston FE, Preece MA. 1998. The Cambridge Encyclopedia of Human Growth and Development.
- Ulijaszek SJ, Kerr DA. 1999. Review article Anthropometric measurement error and the assessment of nutritional status. Br J Nutr 82:165–177.
- Van Hooff MHA, Voorhorst FJ, Kaptein MBM, Hirasing RA, Koppenaal C, Schoemaker J. 1998. Relationship of the menstrual cycle pattern in 14-17 year old adolescents with gynaecological age, body mass index and historical parameters. Hum Reprod 13:2252–2260.
- van 't Hof MA, Wit JM, Roede MJ. 1985. A method to construct age references for skewed skinfold data, using Box-Cox transformations to normality. Hum Biol 57:131–9.
- van Marken Lichtenbelt WD, Westerterp KR, Wouters L. 1994. Deuterium dilution as a method for determining total body water: effect of test protocol and sampling time. Br J Nutr 72:491–497.

- Verbruggen SW, Nowlan NC. 2017. Ontogeny of the Human Pelvis. Anat Rec 300:643–652.
- Vlachopoulos D, Gracia-marco L, Barker AR, Huybrechts I, Moreno LA, Mouratidou T. 2016. Bone Health : The Independent and Combined Effects of calcium, vitamin D and exercise in children and adolescents. In: Food and nutritional components in focus. . p 530–546.
- Völgyi E, Tylavsky FA, Xu L, Lu J, Wang Q, Alén M, Cheng S. 2010. Bone and body segment lengthening and widening: a 7-year follow-up study in pubertal girls. Bone 47:773–782.
- Wadsworth MEJ, Hardy RJ, Paul AA, Marshall SF, Cole TJ. 2002. Leg and trunk length at 43 years in relation to childhood health, diet and family circumstances; Evidence from the 1946 national birth cohort. Int J Epidemiol 31:383–390.
- Waldron T. 2009. Palaeopathology. Cambridge: Cambridge University Press.
- Wall-Scheffler CM. 2012. Energetics, locomotion, and female reproduction: Implications for human evolution. Annu Rev Anthropol 41:71–85.
- Wall-Scheffler CM, Myers MJ. 2013. Reproductive costs for everyone: How female loads impact human mobility strategies. J Hum Evol 64:448–456.
- Walrath D. 2003. Rethinking pelvic typologies and the human birth mechanism. Curr Anthropol 44:5–31.
- Walrath DE, Glanz MM. 1996. Sexual dimorphism in the pelvic midplane and its relationship to Neandertal reproductive patterns. Am J Phys Anthropol 100:89–100.
- Wang J, Zhou J, Cheng CM, Kopchick JJ, Bondy C a. 2004. Evidence supporting dual, IGF-I-independent and IGF-I-dependent, roles for GH in promoting longitudinal bone growth. J Endocrinol 180:247–255.
- Warrener A. 2012. Lower limb joint mechanics in men and women. Am J Phys Anthropol S52:297.
- Warrener AG. 2017. Hominin Hip Biomechanics: Changing Perspectives. Anat Rec 300:932–945.
- Warrener AG, Lewton KL, Pontzer H, Lieberman DE. 2015. A wider pelvis does not increase locomotor cost in humans, with implications for the evolution of childbirth. PLoS One 10:e0118903.

Washburn SL. 1960. Tools and human evolution. Sci Am 203:63–75.

- Waterlow J, Buzina R, Keller W, Lane J, Nichaman M, Tanner J. 1977. The presentation and use of height and weight data for comparing nutritional-status of group of children under age of 10 years. Bull World Health Organ 55:489–498.
- Weaver TD, Hublin J-J. 2009. Neandertal birth canal shape and the evolution of human childbirth. Proc Natl Acad Sci USA 106:8151–6. A
- Welch JM, Weaver CM. 2005. Calcium and exercise affect the growing skeleton. Nutr Rev 63:361–373.
- Wells J, Haroun D, Williams J, Wilson C, Darch T, Viner R, Eaton S, Fewtrell M. 2010. Evaluation of DXA against the four-component model of body composition in obese children and adolescents aged 5–21 years. Int J Obes 34:649–655.
- Wells J, Stock JT. 2007. The biology of the colonizing ape. Am J Phys Anthropol Suppl 45:192–222.
- Wells JC. 2010a. The evolutionary biology of human body fatness: Thrift and control. Cambridge: Cambridge University Press.
- Wells JC. 2015. Between Scylla and Charybdis: Renegotiating resolution of the 'obstetric dilemma' in response to ecological change. Philos Trans R Soc B Biol Sci 370:1–12.
- Wells JC. 2018. Life history strategy and the partitioning of maternal investment: Implications for health of mothers and offspring. Evol Med Public Heal Submitted:153–166.
- Wells JCK. 2006. The evolution of human fatness and susceptibility to obesity: an ethological approach. Biol Rev 81:183.
- Wells JCK. 2010b. The evolutionary biology of human body fatness : thrift and control. Cambridge: Cambridge University Press.
- Wells JCK. 2016. The Metabolic Ghetto. Cambridge: Cambridge University Press.
- Wells JCK. 2017. The New "Obstetrical Dilemma ": Stunting, Obesity and the Risk of Obstructed Labour. Anat Rec 300:716–731.
- Wells JCK, Chomtho S, Fewtrell MS. 2007. Programming of body composition by early growth and nutrition. Proc Nutr Soc 66:423–434.
- Wells JCK, DeSilva JM, Stock JT. 2012a. The obstetric dilemma: an ancient game of Russian roulette, or a variable dilemma sensitive to ecology? Am J Phys Anthropol 149 Suppl:40–71.

- Wells JCK, Figueiroa JN, Alves JG. 2017. Maternal pelvic dimensions and neonatal size: Implications for growth plasticity in early life as adaptation. Evol Med Public Health 2017:191–200.
- Wells JCK, Haroun D, Williams JE, Nicholls D, Darch T, Eaton S, Fewtrell MS. 2015. Body composition in young female eating-disorder patients with severe weight loss and controls: Evidence from the four-component model and evaluation of DXA. Eur J Clin Nutr 69:1330–1335.
- Wells JCK, Stanley M, Laidlaw AS, Day JME, Stafford M, Davies PSW. 1997. Investigation of the relationship between infant temperament and later body composition. Int J Obes 21:400–406.
- Wells JCK, Williams JE, Chomtho S, Darch T, Grijalva-Eternod C, Kennedy K, Haroun D, Wilson C, Cole TJ, Fewtrell MS. 2012b. Body-composition reference data for simple and reference techniques and a 4-component model: A new UK reference child. Am J Clin Nutr 96:1316–1326.
- Whitcome KK, Miller EE, Burns JL. 2017. Pelvic rotation effect on human stride length: Releasing the constraint of obstetric selection. Anat Rec 300:752–763.
- White TD, Asfaw B, Beyene Y, Haile-selassie Y, Lovejoy CO, Suwa G, Woldegabriel G. 2009. Ardipithecus ramidus and the paleobiology of early hominids. Science (80-) 326:75–86.
- White TD, Folkens PA. 2005. The human bone manual. (Elsevier Academic Press, editor.). Burlington.
- WHO. 2006. Guidelines on food fortification with micronutrients. Available from: http://www.unscn.org/layout/modules/resources/files/fortification\_eng.pdf
- WHO. 2014. WHO Infographic Saving mothers' lives 1990-2013. Available from: http://www.who.int/reproductivehealth/publications/monitoring/maternal-mortalityinfographic.pdf?ua=1
- Wiley AS. 2005. Does milk make children grow? Relationships between milk consumption and height in NHANES 1999-2002. Am J Hum Biol 17:425–441.
- Wiley AS. 2011. Milk intake and total dairy consumption: Associations with early menarche in NHANES 1999-2004. PLoS One 6.
- Wiley AS. 2012. Cow milk consumption, insulin-like growth factor-I, and human biology: A life history approach. Am J Hum Biol 24:130–138.
- Wiley AS, Cullin JM. 2016. What do anthropologists mean when they use the term biocultural? Am Anthropol 118:554–569.

- Will M, Pablos A, Stock JT. 2017. Long-term patterns of body mass and stature evolution within the hominin lineage. R Soc Open Sci 4:171339.
- Williams GC, Nesse RM. 1991. The Dawn of Darwinian Medicine. Q Rev Biol 66:1– 22.
- Williams J, Wells J, Wilson C, Haroun D, Lucas A, Fewtrell M. 2005. Evaluation of Lunar Prodigy dual-energy X-ray absorptiometry for assessing body composition in healthy individuals and patients by comparison with the four-component model. Am J Clin Nutr 68:1047–1054.
- Willis A, Oxenham MF. 2013. A Case of Maternal and Perinatal Death in Neolithic Southern Vietnam, c. 2100-1050 BCE. Int J Osteoarchaeol 23:676–684.
- Wittman AB, Wall LL. 2007. The evolutionary origins of obstructed labor: Bipedalism, encephalization and the human obstetric dilemma. Obstet Gynecol Surv 62:739–748.
- WL J. 1982. Lucy's limbs: skeletal allometry and locomotion in Australopithecus afarensis. Nature 297:676.
- Wong JY, Oh AK, Ohta E, Hunt AT, Rogers GF, Mulliken JB, Deutsch CK. 2008. Validity and reliability of craniofacial anthropometric measurement of 3D digital photogrammetric images. Cleft Palate-Craniofacial J 45:232–239.
- Wood JW, Milner GR, Harpending HC, Weiss KM, Cohen N, Eisenberg LE, Hutchinson DL, Jankauskas R, Česnys G, Katzenberg A, Lukacs JR, Mcgrath JW, Roth EA, Ubelaker DH, Wilkinson RG. 1992. The osteological paradox. Curr Anthropol 33:343–370.
- Woodman PJ, Swift SE, O'Boyle AL, Valley MT, Bland DR, Kahn MA, Schaffer JI. 2006. Prevalence of severe pelvic organ prolapse in relation to job description and socioeconomic status: A multicenter cross-sectional study. Int Urogynecol J 17:340–345.
- World Health Organization (WHO). 2015. WHO statement on caesarean section rates (RHR/15.02).
- Worthman C. 1993. Biocultural interactions in human development. In: Perieira M, Fairbanks L, editors. Juvenile primates: Life history, development and behaviour. New York: Oxford University Press. p 339–357.
- Wren T, Kalkwarf H, Zemel B, Lappe J, Oberfield S, Shepherd J, Winer K, Gilsanz V. 2014. Longitudinal tracking of DXA bone measures over 6 years in children and adolescenets: Persistence of low bone mass to maturity. J Pediatr 164:1280– 1285.

- Ye J, Zhang J, Mikolajczyk R, Torloni MR, Gülmezoglu AM, Betran AP. 2016. Association between rates of caesarean section and maternal and neonatal mortality in the 21st century: A worldwide population-based ecological study with longitudinal data. BJOG An Int J Obstet Gynaecol 123:745–753.
- Young SM, Benyshek DC, Lienard P. 2012. The conspicuous absence of placenta consumption in human postpartum females: The fire hypothesis. Ecol Food Nutr 51:198–217.
- Zaffarini E, Mitteroecker P. 2019. Secular changes in body height predict global rates of caesarean section. Proc R Soc B Biol Sci 286:20182425.
- Zhu K, Prince RL. 2012. Calcium and bone. Clin Biochem 45:936–942.
- Zuckerman M, Armelagos GJ. 2011. The origins of biocultural dimensions in bioarchaeology. In: Agarwal SC, Glencross BA, editors. Social Bioarchaeology. Blackwell Publishing Ltd. p 13–43.

#### **Appendix One**

## Institute of Child Health

and Great Ormond Street Hospital for Children NHS Trust UNIVERSITY COLLEGE LONDON

12 December 2000

Dr MS Fewtrell Childhood Nutrition Research Centre ICH

The child first and always

30 Guilford Street London WC1N 1EH

Telephone: 020 7242 9789 Fax: 020 7813 2134

Dear Dr Fewtrell,

**99NT15** Collection of bone mineralisation reference data in normal healthy children.

#### Notification of ethical approval

The above research has been given ethical approval after review by the Great Ormond Street Hospital for Children NHS Trust / Institute of Child Health Research Ethics Committee subject to the following conditions.

- Your research must commence within twelve months of the date of this letter and ethical approval is given for a period of 12 months from the commencement of the project. If you wish to start the research more than twelve months from the date of this letter or extend the duration of your approval you should seek Chairman's approval.
- 2. You must seek Chairman's approval for proposed amendments to the research for which this approval has been given. Ethical approval is specific to this project and must not be treated as applicable to research of a similar nature, eg. using the same procedure(s) or medicinal product(s). Each research project is reviewed separately and if there are significant changes to the research protocol, for example in response to a grant giving body's requirements you should seek confirmation of continued ethical approval.
- Researchers are reminded that REC approval does not imply approval by the GOS Trust. Researchers should confirm with the R&D office that all necessary permissions have been obtained before proceeding.



- It is your responsibility to notify the Committee immediately of any information which would raise questions about the safety and continued conduct of the research.
- 5. On completion of the research, you must submit a report of your findings to the Research Ethics Committee.
- 6. Specific conditions pertaining to the approval of this project are:
  - The use of the enclosed standard consent forms for the research. A copy of the signed consent form must be placed in the patient's clinical records and a copy must be kept by you with the research records.

Yours sincerely

. Shoils

Orlagh Sheils Administrator to the Research Ethics Committee





**Great Ormond Street Hospital** for Children NHS Trust / The Institute of Child Health Local Research Ethics Committee Institute of Child Health 30 Guilford Street London WC1N 1EH

> Tel: 020 7905 2620 Fax: 020 7905 2201 Email: I.howe@ich.ucl.ac.uk

22<sup>nd</sup> December 2003

Dr M Fewtrell Nutrition ICH

Dear Dr Fewtrell,

Title:

#### Collection of bone minieralization reference data in normal healthy children R&D registration number: 99NT15 Protocol number/version: N/A

Thank you for your letter dated 15<sup>th</sup> December 2003. The Chairman of the Research Ethics Committee, Dr Victor Larcher, has on behalf of the Committee approved your request for an extension for a period of thirty-six months. Ethics approval will expire on 21<sup>st</sup> December 2006, so please inform us if you wish to continue beyond this date.

The decision will be ratified at the full Committee meeting that will take place on Wednesday 4<sup>th</sup> February 2004 (Please note that you will not receive a letter confirming the above ratification).

Yours sincerely

1 tong

Laura Howe **Research Ethics Coordinator** 

An advisory committee to North Central London Strategic Health Authority

Mrs Jill Noble Ethics Committee Secretary



Dr Jay Stock Department of Archaeology & Anthropology Downing Street Cambridge CB2 3DZ

29 June 2016

Dear Dr Stock

## Ethical approval: An examination of the biological and biosocial influences on the developmental origins of female pelvic growth

The Chair of the Ethics Committee for the School of the Humanities and Social Sciences, acting on the Committee's behalf, has considered the documentation you provided, which followed the procedures concerning ethical approval of research.

I am able to inform you that approval, with respect to ethical considerations, has now been given to your project. Please note that this clearance is based on the documentation you have submitted. You must resubmit your application to the Ethics Committee should you subsequently make any substantive changes relating to matters reviewed by the Committee.

We are content for this letter to be forwarded to your grant sponsors, Parkes Foundation.

Yours sincerely

Jill Noble Ethics Committee Secretary

cc Sarah-Louise Decrausaz, student Katie Teague, Administrator

> 17 Mill Lane Cambridge CB2 1RX

Tel: +44 (0) 1223 766238 Fax: +44 (0) 1223 760433 Email: cshssethics@admin.cam.ac.uk www.cshss.cam.ac.uk



## Appendix Two



## Appendix Three

BODY COMPOSITION: REFEREN	
Social Data Uni	ique file identifier
Today's date	Use own initials, number of file and first 2 letters + 3 digit study number e.g. JW1DX029
Number of people in home (incl subject)	
Number of rooms in home (incl dining, living + be	edrooms, excl kitchen + bathroom)
Is nome own or rented? O Owned O Rented Is mother married? O Yes O No O Living with partner Mother's date of birth ////////////////////////////////////	If home is rented, is it from: O Private landlord O Council O Housing associatio O Not rented O irrelevant SE's below C grade rade A-C Highers, NVQ level 3, ng (incl SRN/RGN/RM/RHV), NVQ levels
4/5, BEC/TEC Higher	
Mother's educational attainments (highest	completed)
Mother's highest qualification	
Mother's occupation	
How many months employed in the last yea	r?
	Page 1.1

	ta contd
	Father's date of birth / 19
1 No ed 3 >3 CSI 4 A level 5 Degree 4/5, BEC	quals 2 <3 CSE's or GCSE's below C grade E's or any O levels or GCSE grade A-C s, ONC/OND/BEC/TEC, SCE Highers, NVQ level 3, //HND/HNC professional training (incl SRN/RGN/RM/RHV), NVQ levels /TEC Higher
ather's educ	ational attainments (highest completed)
ther's highes	st qualification
ther's occup	ation
v many mon	ths employed in the last year?
o is the prima	ary earner for the family? O Father
	O Mother
	O Grandparent
	O missing
	- meening
ocial class (	use primary earner's occupation)
ocial class (	use primary earner's occupation) Social code as below
ocial class (	use primary earner's occupation) Social code as below



D		1		
		1		

#### Social Data contd

How many children live in the family home?

How many are mother's own children?

### Ethnic origin

Please tick the box that you think best describes your ethnic origin

White Ο Black-Caribbean O Black-African Ο Black-other Ο Indian Ο Pakistani Ο Bangladeshi 0 Chinese Ο Asian-other Ο Other Ο Refused Ο

Page 1.3

" <b>h</b>
3452

## **BODY COMPOSITION STUDY: REFERENCE DATA**



## Parent's opinion of the child's physical activity levels

Total hours child spents in vigorous activity per week
Level of child's activity compared to peers 1= much less than peers 2= less 3= same 4= more 5= much more
Pubertal Development (if 9 or above) data to transfer from confidential data sheet
Breast/genital development stage (1-5)
Pubic hair development (1-5)
Age periods commenced (months)
Have periods started yes
no o
not annlicable O
not applicable ○
not applicable $\bigcirc$ Oral contraceptive or implant?Yes = 1 No = 2
Not applicable $\bigcirc$ Oral contraceptive or implant?       Yes = 1 No = 2         Date of first day of last menstrual period       / /
Number of days in whole menstrual cycle
Number of days in whole menstrual cycle   For example; 5 days menstruation then 25 days to next menstruation = 30 day cycle. Yes = 1 Yes = 1 No = 2 No = 2 Solution of the second se

BODY COMPOSITION STUDY: ID D X.
Children's Exercise Questionnaire
How many PE lessons do you have per week
How long is each PE lesson, in minutes

How many hours per week do you spend on each of these activities outside PE classes:

Riding bike	Tennis .
Swimming .	Netball
Running .	Hockey
Football	Basketball
Aerobics/dancing	Rugby
Gymnastics .	Skating .
Walking .	Other sport
Other, please specify	

Reference Study: Exercise/puberty (1 of 2)

#### **Appendix Four**

LUCL UCL INSTITUTE OF CHILD HEALTH Great Ormond Street NHS Hospital for Children

# Would you like to earn £30 and learn about your body composition?

We would like to invite women of Indian, Pakistani, Bangladeshi and Sri Lankan ancestry between the ages of 20-28 to take part in a research study looking at relationships between muscle and fat tissues in the body If you're interested, please contact us for further information (contact details found below)

The aim of the study: to better understand relationships between muscle and fat tissues in the body, and how these relationships may link with early life development and later life metabolic health

If you agree to take part, we will do a number of measurements (including MRI and anthropometry, pictured below with the MRI scanner on the left) as you visit the UCL Institute of Child Health/Great Ormond Street Hospital for one half day (~4 hours including a break for food and drink) or on two separate, shorter visits at your convenience. Weekends and bank holidays are a possibility if preferable to a weekday. We will provide food and drink on the day, and any travel expenses will be paid back to you.





Researcher contact details:

Ms Meghan Shirley UCL Institute of Child Health Tel: 07923691733 Email: <u>meghan.shirley.13@ucl.ac.uk</u>