

2020

Expanding the evolutionary explanations for sex differences in the human skeleton

Holly M. Dunsworth

University of Rhode Island, holly_dunsworth@uri.edu

Follow this and additional works at: https://digitalcommons.uri.edu/soc_facpubs

The University of Rhode Island Faculty have made this article openly available.
Please let us know how Open Access to this research benefits you.

This is a pre-publication author manuscript of the final, published article.

Terms of Use

This article is made available under the terms and conditions applicable towards Open Access Policy Articles, as set forth in our [Terms of Use](#).

Citation/Publisher Attribution

Dunsworth, HM. Expanding the evolutionary explanations for sex differences in the human skeleton.

Evolutionary Anthropology. 2020; 29: 108-116. <https://doi.org/10.1002/evan.21834>

Available at: <https://doi.org/10.1002/evan.21834>

This Article is brought to you for free and open access by the Sociology & Anthropology at DigitalCommons@URI. It has been accepted for inclusion in Sociology & Anthropology Faculty Publications by an authorized administrator of DigitalCommons@URI. For more information, please contact digitalcommons@etal.uri.edu.

1
2 **Expanding the evolutionary explanations for sex differences in the human skeleton**

3
4 Holly M. Dunsworth

5 Department of Sociology and Anthropology, University of Rhode Island

6
7 **Running title:** Evolved sex differences in the human skeleton

8
9 October 1, 2019

10
11
12
13 **Abstract**

14 While the anatomy and physiology of human reproduction differ between the sexes, the effects
15 of hormones on skeletal growth do not. Human bone growth depends on estrogen. Greater
16 estrogen produced by ovaries causes bones in female bodies to fuse before males' resulting in
17 sex differences in adult height and mass. Female pelvises expand more than males' due to estrogen
18 and relaxin produced and employed by the tissues of the pelvic region and potentially also due to
19 greater internal space occupied by female gonads and genitals. Evolutionary explanations for
20 skeletal sex differences (aka sexual dimorphism) that focus too narrowly on big competitive men
21 and broad birthing women must account for the adaptive biology of skeletal growth and its
22 dependence on the developmental physiology of reproduction. In this case, dichotomizing
23 evolution into proximate-ultimate categories may be impeding the progress of human
24 evolutionary science, as well as enabling the popular misunderstanding and abuse of it.

25
26 **Key words:** sexual dimorphism, height, growth, pelvis, estrogen, childbirth, male competition

27
28 **1 Introduction**

29 Scholarship on sex differences in the human skeleton, from the focused to the tangential,
30 explains how males are taller due to sexual selection for contest winners, and how females are
31 broader due to natural selection for childbirth. (For just two recent examples see refs 1 and 2,
32 respectively; for textbook examples, see refs 3-5) While discourse among anthropologists and
33 fellow travelers on skeletal sex differences is often nuanced about causal complexity and
34 unknowns, it does not seem to have affected the discussion beyond these circles, where public
35 perception of evolutionary causality is far simpler. The narrow emphasis on competitive men and
36 birthing women harkens back to the origins of human evolutionary biology^{6,7}, and these
37 explanations for biological sex differences dominate the popular understanding of human
38 evolution today.

39
40 Here, 'female' refers to humans of all genders with anatomy that is commonly assigned to be
41 female, and the same gender inclusivity applies to 'male'—with the understanding that neither
42 sex nor gender divide into uniform, discrete, or binary categories, which is why "sex differences"
43 rather than "sexual dimorphism" is employed throughout this essay.⁸⁻¹⁰

44

45 This essay briefly reviews the complex biology of sex differences in human stature and pelvic
46 dimensions, focusing mainly on the role of estrogen. Investigating how these differences develop
47 expands their evolutionary explanations. Peering from this angle stirs skepticism of the
48 traditional, narrow emphasis on the dominant ideas, and creates opportunities for testing them.
49 To be clear, the reigning explanations (male competition for skeletal size differences, and
50 childbirth for pelvic differences) are neither extensively reviewed nor rebutted in this essay.
51 Instead, the goal of this essay is to highlight some additional context for the evolution of sex
52 differences in the skeleton. In order to present a fresh approach to a familiar topic, this essay
53 primarily asks why skeletal sex differences exist at all, rather than starting with comparisons of
54 degrees of sex differences between humans and other primates.

55
56 Throughout this essay, and perhaps already by now, many readers will be partitioning evidence
57 into the dichotomous realms of proximate and ultimate evolutionary explanations¹¹ —the
58 ultimate ones being childbirth and male competition and the proximate ones being mechanisms
59 of growth and development of the skeleton. However, this convention is not a requirement of
60 evolutionary thinking¹² and it is neither espoused nor endorsed here.

61
62 Lastly, in anthropology the sociocultural consequences of the scientific truth are as equally
63 important as the truth itself. The current pop culture narrative where men are specially built *for*
64 competition and women are specially built *for* reproduction helps root socioculturally prescribed
65 and proscribed sex roles, and rigid gender rules and stereotypes, in “human nature.” Expanding
66 the dominant origins story for sex differences in height and pelvic dimensions will not just
67 improve science but will also help rip human evolution out of the patriarchal playbook.

68 69 **2 Why are there sex differences in human stature?**

70 While human height varies globally, all human populations exhibit the same pattern where mean
71 adult male height is greater than mean adult female height. In the U.S. (Figure 1),¹³ after nearly
72 the same growth trajectory from 2 years of age, both males and females are roughly 62 inches
73 (157 cm) tall at 13 years. After that, the female growth curve flattens to reach the average final
74 height of about 64 inches (163 cm). Conversely, in males, the growth curve continues on roughly
75 the same trajectory for at least 1.5 more years until it then flattens to reach the average final
76 height of about 70 inches (178 cm). This is an additional 9% of growth in stature compared to
77 females. Average age of menarche in the U.S. occurs at about 13 years¹⁶ (which matches that
78 reported in at least one small-scale subsistence society¹⁷). Thus, while males continue to grow in
79 stature, females slow to a stop, and simultaneously begin monthly cycling. Females who reach
80 menarche relatively later continue to grow-at the faster prepubertal rate until onset of menses and
81 end up being relatively taller adults.¹⁸ The synchrony of menarche with growth deceleration and
82 subsequent arrest is not mere coincidence. Both the menstrual cycle and skeletal growth depend
83 on estrogen.

84
85 For males and females, long bone growth and epiphyseal closure are highly dependent on
86 estradiol, the most important of the naturally occurring estrogens, and hereafter also referred to
87 as "estrogen" (the literature cited also employs the terms interchangeably).¹⁹⁻²¹ Estrogen
88 accelerates the loss of progenitor cells in the resting zone of the long bone growth plate, which
89 causes senescence in the growth plate and results in cessation of growth.²²⁻²⁴ Estrogen is
90 produced in both the ovaries and testes, where androgens are converted into estrogen by the

91 enzyme aromatase. Aromatase is expressed in the growth plates, too, and so some of the
92 hormone conversion related to bone growth is local.²⁵ Estrogen's effects are biphasic with one
93 level stimulating bone growth and an even higher level stimulating epiphyseal closure.¹⁹
94 Prepubertal females have eight times the estradiol levels of males at the same age,¹⁹ which helps
95 explain both their earlier growth spurt (i.e. peak growth rate, which is not obvious on Figure 1
96 but see Bogin¹⁴) and earlier growth arrest compared to males. An excess of estrogen causes
97 medically diagnosed short stature in both sexes.²⁵ Estrogen in low doses enhances growth
98 hormone (GH) and IGF-1 production (the GH-IGF-1 axis) which are key to linear bone growth,²⁶
99 but at high doses estrogen inhibits IGF-1. Androgens do stimulate GH, but in their absence,
100 normal growth can occur as long as there is sufficient estrogen.²⁰ As androgen production
101 increases, males also reach critical levels of estradiol to stimulate the process of growth plate
102 fusion starting around 16 years of age.²⁷ These levels are likely to be lower than they are in
103 females because at this older age the growth plates are more senescent and require a more brief
104 exposure to estradiol.²² In addition, levels of estrogen at this time are critical to bone mass
105 maintenance,²¹ an important function of estrogen in all humans.

106
107 The sex difference in estradiol levels is due to its greater involvement in ovulation and
108 menstruation than in spermatogenesis and related processes, where it is also vital. Estradiol
109 regulates spermatogenesis by testicular Sertoli cells by both inhibiting and stimulating, in a dose-
110 dependent and temporally sensitive process.²⁸⁵ Aromatase activity is higher in motile as opposed
111 to immotile sperm and was found to be significantly decreased in a population of infertile men.²⁹
112 In puberty and in adulthood, excess estrogen can inhibit penile erection.²⁸ In all humans, a
113 delicate balance of estrogen/aromatase is as fundamental to reproduction as it is to skeletal
114 growth. We are safe to assume that *Homo sapiens*' prolific biology of reproduction is adaptive.

115
116 In addition to the effects of estrogen on stature, there may be a pubertal onset of energetic,
117 metabolic, and nutritional costs that force a tradeoff with skeletal growth. When energy intake
118 and physical activity are held constant, basal metabolic rate (BMR) varies significantly across
119 the menstrual cycle, with the lowest BMR occurring approximately one week before ovulation,
120 subsequently rising until the beginning of the next menstrual period, then decreasing at
121 menstruation. Several studies have quantified changing energy intake across the menstrual cycle.
122 Peak and minimum intake across the cycle differ by 359 kcal/day (n=6).³⁰ Ten days before
123 menstruation, mean energy intake is approximately 500 kcal/day higher than 10 days after (n=
124 8).³¹ Compared to the follicular/ovulatory phase, the luteal phase corresponds to an increased
125 energy intake of 685 kJ/day (or 164 kcal/day).³² These data point to the metabolic effects of the
126 changing estrogen/progesterone ratios across the cycle, including the costly thickening of the
127 endometrium.³³⁻³⁵ These costs may differ not just individually but across the reproductive
128 lifespan. Reiches et al.³⁶ described a relatively higher cost of menstruation in younger adolescents
129 compared to older adolescents.

130
131 As this brief review of the relationship of estrogen to skeletal growth has shown, the
132 reproductive systems of males and females differently affect a skeletal system that is shared by
133 males and females. So, the evolutionary explanation for the existence of sex differences in
134 human height is rooted in the origins of estrogen and its subsequent importance in all vertebrate
135 bodies some 500 million years ago.³⁷ Also of crucial importance are the origins of internal
136 fertilization and viviparity.

137
138 Great apes develop sex differences in body mass like humans do, where both sexes follow
139 similar growth trajectories until the pubertal transition when the females stop growing and the
140 males continue to grow for a longer period of time.³⁸ Though levels of sex differences in body
141 size differ between species, among the living hominids (great apes and humans) there is likely to
142 be significant shared fundamental biology of reproduction and skeletal growth. Thus, the
143 existence of human sex differences in stature is rooted in ancestry. It remains to be known
144 whether there are important sex- and species-level differences in the biology of skeletal growth
145 among hominids, and whether these could explain the differing degrees of sex differences in
146 body size across primates, even after accounting for allometry and for estrogen production—
147 which is potentially constrained by testes size and may be an important factor in the extended
148 growth of male gorillas and orangutans. (For a discussion of how these “somatic strategies”
149 might occur at primate puberty see ref 39.)

150 However, the traditional and enduring textbook explanation for sex differences in hominid body
151 size is sexual selection³⁸—with large ancestral males winning competitions, which boosted their
152 reproductive success compared to smaller males.⁴⁰ Because gorillas have both intense male
153 competition and large male bodies, the mere existence of sex differences in human body size
154 serves as evidence of sexual selection being the driver of these differences.¹

155
156 But as Plavcan^{41,42} has cautioned, there is not a straight-forward relationship between sexual
157 selection and primate male body size, largely because the sorts of data that are required to
158 investigate this relationship are difficult to obtain. It is also difficult to tease selection on male
159 body size apart from selection on female body size through the generations, which is sometimes
160 understood within the framework of females as the “ecological sex”.⁴³⁻⁴⁵ Given the nutritional,
161 energetic, metabolic, and locomotor costs of pregnancy, lactation, and mothering,^{46,47} there are
162 (context-specific) limits to female body size,⁴⁸ perhaps leading to biology that favors
163 reproduction over growth.^{49,50}

164
165 Yet even within this more complete “ultimate” narrative, with selection optimizing the two
166 sexes’ skeletal growth separately, the sexual selection perspective on male height seems
167 unnecessary. That provocative last sentence is not a claim that the sexual selection explanation is
168 wrong or that it is implausible. But in light of what is known and still unknown about skeletal
169 development and its relationship to the endocrinology of reproduction, suddenly there is room
170 for skepticism about the relevance of male competition and female choice as an explanation for
171 the existence of sex differences in stature, let alone its singular dominance of the narrative. More
172 work is needed if sexual selection is to be held up as *the* explanation for why male hominids
173 have longer bones than female hominids do.

174
175 Given the complex, shared biological systems briefly outlined above, which are intricately tied to
176 successful reproduction and that contribute to terminal height, stature differences within adult
177 males are probably weaker targets of selection than is assumed by sexual selection scenarios. In
178 their recent overview of the evolution of human height variation, Stulp and Barrett⁵¹ made a
179 similar point when they wrote that, “height itself is less important as a trait than the underlying
180 components of growth rates and the timing of reproductive maturity that give rise to it. This

181 raises the question of whether height does, in fact, carry any selective advantage independent of
182 its links to life history.”(p. 220)

183
184 Singularly upholding the male competition hypothesis for sex differences in human stature
185 requires, for example, the demonstration that men’s estradiol/aromatase production, levels,
186 receptors, and timing are primarily due to the fitness rewards of being taller than females, or
187 primarily due to the fitness rewards of being taller than other males. It also requires, for example,
188 the demonstration that men’s estradiol/aromatase production, levels, receptors, and timing are
189 not primarily due to something fundamental to male gonad, genital, and gamete maturation or
190 function, and not primarily due to shared biology with females.

191
192 Data from tracking the reproductive success of human males fails to comprehensively answer the
193 question of why there are sex differences in human height,⁵² and further work of this kind, even
194 across primates, will continue to be insufficient for elevating the sexual selection explanation for
195 sex differences in height if it is not integrated with some insightful combination of physiological,
196 endocrinological, developmental, and/or genetic approaches. Perspectives that assume the
197 extended skeletal development of males is a delay in body size maturation and/or is a cost that
198 *requires* a male-specific selection-based explanation must reckon with the risks that changes to
199 the biology of male skeletal growth would also pose to the biology of male fertility. Further,
200 investigations of these issues need not assume that a lack of sex differences in the skeleton (i.e.
201 “monomorphy”) is the biological baseline or default in all primates and, thus, that sex differences
202 in the duration of skeletal growth or in long bone length have been *directly* driven apart by sex-
203 specific sexual or natural selection on skeletal growth. Free from these assumptions, there is
204 potential for exciting advances including, perhaps, the discovery that sexual selection does
205 indeed play the lead role in this story.

206
207 For humans and likely other hominids, male skeletons continue to grow after females’ stop
208 because their bodies take longer to produce enough estradiol to surpass the amount that
209 stimulates continued growth and to achieve a level that closes long bone epiphyses. As of now,
210 no advantage to being taller or more massive is required to make sense of this phenomenon
211 which may be largely a by-product of the adaptive reproductive biology that differs between the
212 sexes. Dominance⁵³ and competition may be consequences of greater height and mass, but the
213 claim that they cause sex differences in the skeleton requires far more investigation.

214 215 **3 Why are there sex differences in human pelvic dimensions?**

216 On average, human female pelves have longer pubes, more laterally flaring ischial spines and
217 tuberosities, and relatively shorter and wider sacra. Thus, they often have inlets (often measured
218 from sacral promontory to the superior pubic symphysis), midplanes (often measured as the
219 distance between ischial spines), and outlets (often measured from coccyx to inferior pubic
220 symphysis or measured as the distance between ischial tuberosities) that are relatively larger in
221 diameter than those of males⁵⁴⁻⁵⁶. These dimensions together comprise the “true pelvis” or “birth
222 canal” which is relatively larger in females than in males.⁵⁴ So, while there is geographic
223 variation in human pelvic morphology⁵⁷ and while typical female pelvic inlet shape may be
224 “android” like males (contra traditional expectations that they be distinctly “gynecoid”⁵⁸), there
225 are consistent and patterned sex differences in human pelvic morphology pertaining to the size of
226 the space inside the pelvic cavity.

227
228 Fetal pelvis, between seven months and birth, display sex differences that already hint at those in
229 adults.⁵⁹ Around the transition to adulthood, female pelvis tend to fuse earlier than those of
230 males at all sites. This pattern parallels the sex differences in long bone fusion. The site with the
231 greatest sex difference in closure is the anterior epiphysis of the acetabulum which articulates
232 with the pubis and is actively fusing between 11-16 years in females but not until ages 14-17 in
233 males.⁵⁹ Sex differences in pelvic morphology become pronounced during this stage in life.
234 LaVelle⁶⁰ found that between ages 8 and 18, female pelvis expand slightly more than males' in
235 the dimensions of the true pelvis. Some of the most conspicuous change occurs in pubis length.
236 Likewise, Greulich and Thoms found greater transverse dimensions in developing and adult
237 female pelvis.⁶¹ Huseynov and colleagues observed, in a cross-sectional sample, that true pelvic
238 dimensions expanded from puberty until the ages of 25-30 years, then after 40 years these
239 dimensions diminished in magnitude.⁶² Whether and how the development of sex differences in
240 the pelvis can be causally linked to intra- and inter- sex differences in the timing of the fusion of
241 the pelvic bones remains to be determined.

242
243 Because estrogen is produced in greater amounts in female bodies and those amounts change
244 across the life course, estrogen is the established explanation for ontogenetic changes to female
245 pelvic anatomy as compared to that of males.^{61, 63} This holds even for the neonatal sex
246 differences because the last few weeks of fetal development occur while estrogen levels are
247 highest in gestation.⁶⁴ But if estrogen is a primary driver of long bone growth and fusion (as
248 discussed above), then how does it act locally just on the bones of the pelvis and only in
249 females?

250
251 Rodent experiments from 1929-1935 suggest that dosing a male body with estrogen "feminizes"
252 the pelvis,⁶¹ suggesting the system works on any pelvis. However, a review of skeletal biology in
253 2005²⁶ reported that the stimulatory effects of estrogen on skeletal growth and maturation in
254 humans are poorly reproduced in rodent studies. Regardless of these important issues that may
255 complicate a comparative approach, estrogen's hypothesized localized effects on female pelvis
256 warrants deeper consideration. This is especially necessary in light of a recent study that failed to
257 correlate within-individual levels of sex differences in the skull with that in the pelvis,
258 suggesting that a "single systemic influence, such as hormone levels, is not solely responsible for
259 sex differences in the size and shape of these skeletal elements."⁶⁵

260
261 The muscles of the pelvic floor, like the levator ani, the round ligament of the uterus (which is
262 actually a smooth muscle, not a ligament), and other uterine ligaments (the pubocervical,
263 uterosacral, and cardinal (transverse cervical) ligaments) contain estrogen receptors (ER), which
264 suggests they are targets for estrogen.⁶⁶⁻⁷⁰

265
266 ER are absent in typical skeletal muscles like the rectus abdominis and erector spinae,^{66, 67}
267 supporting the hypothesis that the pelvic muscles are under special hormonal control. Pelvic
268 floor muscles also contain ER in their connective tissue cells, which are the "glue" that fixes the
269 muscles together and to the pelvic bones.⁶⁷ The round ligament grows during pregnancy and
270 shrinks (not slacks) after parturition, which could influence skeletal remodeling.⁷¹ The markedly
271 earlier fusion in females, described above, of the anterior epiphysis of the acetabulum (which
272 forms the iliopubic eminence) could be influenced by its close proximity to the deep inguinal

273 ring, which transmits the round ligament. All of this suggests that the muscles and ligaments of
274 the female pelvis influence the bones to which they are adjacent or anchored in ways that differ
275 from other muscle- and ligament-bone interfaces, given the known effects that estrogen has on
276 bone growth and remodeling. In addition, relaxin, which is produced by the ovary and placenta,
277 induces the production of osteoclasts⁷² which are key to bone resorption and remodeling—a well-
278 documented phenomenon when it comes to resorption of the human, nonhuman primate, and
279 nonhuman mammal pubis.⁷³ Greater parity increases estrogen and relaxin exposure, which leads
280 to the expectation that greater parity would be correlated to expanding pelvic dimensions, but at
281 least one recent study failed to distinguish non-parous from parous female pelvises.⁶²

282
283 What is more, the volume occupied by internal female organs—in addition to the bladder and
284 rectum housed within all pelvises—may be causing the expansion of the true pelvis. The last few
285 weeks of fetal growth show marked changes in uterus size, position, and angle of flexion.⁷⁴
286 Neonatal uteruses are 3.5 cm long and 1.4 cm thick.⁷⁵ Between the ages of 1-13, uterine volume
287 increases from 0.91 cm³ to 16.15 cm³,⁷⁶ between ages 16-17.5 it is 60 cm³, and between ages 24-
288 29 it is 79 cm³.⁷⁷ The uterus begins a more rapid growth rate around 10 years of age, with the
289 onset of puberty and during concomitant increases in luteinizing hormone (LH), follicle
290 stimulating hormone (FSH), and estradiol.⁷⁸ At this time, roughly 2-3 years before menarche, the
291 vaginal and vulvar epithelia thicken and, along with the cervix and clitoris, they increase in
292 size.⁷⁸ Ovarian volume is 1 cm³ in the first year of life (which is larger than the second year)⁷⁵,
293 from ages 7-12.5, it increases from 1.4 to 4.9 cm³, and then from ages 16-17.5 it measures 8.9
294 cm³.⁷⁷ (Unlike the expanding uterus, ovarian volume from ages 24-29 is reduced, measuring 7.2
295 cm³.) The size of the uterus and cervix increase over a lifetime with parity.⁷⁹ During the luteal
296 phase of the menstrual cycle, when the endometrium thickens, the uterus expands to at least 1.6
297 its volume, likely more.⁸⁰ Vaginal epithelium reaches its peak thickness mid cycle and vaginal
298 muscle fibers thicken late in pregnancy.⁷⁸ While these data were collected from small samples,
299 datasets from different sources concur. Volumetric data on the clitoris proved elusive, but linear
300 dimensions—like those of the bulbs measuring 3-4 cm long when flaccid and 7 cm when erect,
301 and the crura measuring 5-9 cm long—are listed in ref. 81. In contrast, the only internal organ of
302 comparable size that is specific to male pelvises is the prostate which develops from 1.4 cm³ (ages
303 0-9) to 6.9 cm³ (ages 10-19), and to 15.3 cm³ (ages 20-29), remaining much smaller than the
304 uterus across those age groups.⁸² In addition, the prostate is nestled under the bladder while the
305 uterus and ovaries are situated higher up, within the pelvic inlet or brim, in direct line between
306 the pubic symphysis and the sacral promontory. That is, the vagina, uterus, and ovaries are not
307 just taking up more volume but are also, arguably, situated within a more skeletally constrained
308 region of the pelvis compared to the prostate. Publically posted pelvic MRIs allow for visual
309 inspection of internal pelvic anatomy and the comparison of one male and one female.⁸³ What, if
310 any, effects that age-related prostate enlargement may have on the male pelvis are apparently
311 unknown, but are not predicted to mimic what is hypothesized here for developing females
312 because of the difference in context, both in terms of age and estrogen.

313
314 The increase in size over the lifetime and the periodic expansion (during intercourse, the
315 menstrual cycle, and pregnancy) of internal gonads and genitals may be spurring changes to the
316 bones that form the cavity they occupy similar to the ways that organs and bones expand together
317 elsewhere in the body. As brains and skulls develop together, signals for growth are recognized
318 by both neural and skeletal tissues in an integrated manner.⁸⁴ Growth signals may be mediated by

319 tensile strain, caused by mechanical stress on the bones by the growing soft tissue. In a similar
320 fashion, growth of the bony orbit likely responds to the development of the eye.⁸⁵ Such processes
321 are potentially occurring in the thorax, coupling the developing heart and lungs with an
322 expanding ribcage. Habitual human swimmers provide a natural experiment for investigating this
323 phenomenon. Documented increases in swimmers' lung volumes, especially in athletes who train
324 intensely from childhood, are correlated with the development of physically wider chests^{86,87}
325 perhaps due to the increased pressure while actively inhaling and exhaling while immersed in
326 water. The pelvis may be no exception when it comes to the skeleton's plastic accommodation
327 for developing soft tissue. Further, differences in the shapes of the internal skeletal spaces may
328 vary according to the shapes of the organs within. Variation in uterine shape, existing as early as
329 fetal development, could influence variation in pelvic proportions, and vice versa. Fetal uteruses
330 are cylindrical, pear-shaped, heart-shaped, or hourglass-shaped.⁷⁴ Amount and direction of
331 uterine flexion, and the pace of its development, may also factor into how the pelvis develops,
332 and vice versa. The decrease in uterine volume after peak fertility may help explain why
333 Huseynov and colleagues⁶² found that older adult female pelves are less expansive.

334
335 In sum, sex differences in the dimensions of the true pelvis are influenced by localized effects of
336 estrogen and relaxin within a system of gonads, genitals, ligaments, muscles, and bones in ways
337 that are not fully understood. There is potential for sex differences to arise due to the plasticity of
338 the pelvic bones to accommodate the greater volume of developmentally and functionally
339 dynamic gonads and genitals housed within the female pelvis. As with height differences, the
340 explanation is fundamentally rooted in the ancient origins of estrogen, internal fertilization, and
341 pregnancy, the soft tissue differences between the sexes that evolved as a consequence, and how
342 they affect the local skeleton differently in males and females with different hormone levels.
343 Because sex differences in pelvic dimensions are common across primates,⁵⁴ this is an ancestral
344 condition in humans.

345
346 But, the widespread explanation for sex differences in the human pelvis is merely and simply
347 childbirth. For example, "Females have big pelves because they give birth to big babies."⁸⁸
348 While intuitive, this explanation for human pelves or for other primates' is no longer a strong
349 one—at least not in isolation as it is frequently provided.

350
351 Moffett investigated whether primates with greater cephalopelvic proportions (size of neonatal
352 head compared to pelvic inlet) had greater pelvic sex differences and they did, but humans have
353 even more than is explained by cephalopelvic proportions.⁵⁴ That is, primates like *Hylobates*
354 have similar cephalopelvic proportions to humans but exhibit smaller sex differences in the
355 pelvis. Even chimpanzees, which have small enough neonates to fit through the male pelvis
356 (inferred from measures published in ref. 54 and 89), still have sex differences, with female
357 pelves being more capacious than males'.

358
359 It is possible that differences in type and magnitude of sex differences in primate pelves reflect
360 differences in soft tissue anatomy, reproductive physiology, and effects/amounts of estrogen and
361 relaxin and their receptors. Uterus location, size, flexion, and function could vary in important
362 ways that impact the skeleton. Whether primate males have descended testes or not is also likely
363 a factor. Differences across primates in clitoral anatomy as well as anatomy involved in estrus
364 swelling could contribute to differences between species in sex differences, too. The round

365 ligament grows during pregnancy and shrinks after parturition in other primates as it does in
366 humans,⁹⁰ which may contribute to their pelvic remodeling. Interestingly, rodents and
367 lagomorphs have a different system for suspending the uterus and related organs⁹¹ and so if this
368 is affecting their pelvic architecture, it is another reason (added to the estrogen issue mentioned
369 above) that rat and mouse models may be inappropriate for explaining primate/human sex
370 differences in the skeleton. When Kurki and also Fischer and Mitteroecker observed that shorter
371 women have relatively large “obstetric” dimensions^{92,93} perhaps it is due to the allometry and/or
372 conservation of size and function of soft tissues, no matter the stature. Finally, human sex
373 differences in the pelvis could be more pronounced than expected compared to other primates’
374 because of the more tubular or constricted construction of the hominin pelvis, and how pelvises
375 with and without internal female organs and greater estrogen exposure develop in the context of
376 bipedalism.

377
378 A genital, gonadal, and hormonal view of the evolution of pelvic sex differences (and of the
379 conservation of “obstetric” dimensions even in small bodied females) contrasts the traditional
380 “ultimate” evolutionary approach that downplays developmental dynamics. A developmental
381 perspective has less room for ideations of genetically programmed population- and species-
382 specific tweaks of the space between pelvic bones in females versus males. Investigations of
383 these issues need not assume that a lack of pelvic sex differences is the biological baseline or
384 default and, thus, that skeletal differences have been driven apart by sex-specific selection on
385 adult skeletal morphology. We need not assume the logic of the obstetrical dilemma hypothesis,
386 where female pelvises would be like males’ if only selection for childbirth had not forced a
387 compromise.

388
389 There is a crucial, constant function of the human female pelvis no matter the sex, age, or parity
390 and that is to house developing, functioning organs. Female bodies and pelvises contain tissues
391 during the entire life course that stimulate pelvic bone growth and remodeling. Thus, the internal
392 dimensions of the female pelvis are far more ovarian, uterine, clitoral, and vaginal than they are
393 ‘obstetric.’ In the end, it may be that females give birth to big babies because they have big
394 pelvises.

395
396 **4 Concluding Remarks**

397 Investigations of sex differences in the human skeleton have faced many of the challenges in
398 evolutionary biology that Smith described: “Some narrative explanations rely on theory-driven
399 assumptions that may not be shared by readers... Some will not use good judgement when taking
400 into account how underdetermination inevitably limits what can be inferred from historical
401 data... Some will make unreasonable assumptions about what the current utility of a feature can
402 tell us about its historical role... Some will make unreasonable assumptions that simplify the
403 contingency of the historical situation... Some will allow coherence and simplicity in narratives
404 to substitute for evidence.”⁹⁴

405
406 Sex differences in human height and pelvic dimensions require a bigger, more complicated, and
407 more interesting story than simply “male competition” and “childbirth.” Greater estrogen results
408 in the bones in female bodies fusing before males’ leading to sex differences in adult height and
409 mass. Female pelvises expand more than males’ due, potentially, to the space taken by vaginas,
410 clitorides, uteruses, and ovaries and because of the estrogen and relaxin produced and employed

411 by the tissues of the pelvic region. Generally speaking, these evolved processes are not unique to
412 *Homo sapiens*. Understanding the details of the developmental biology of the skeletal and
413 reproductive systems of human males and females, and understanding that development in
414 phylogenetic context, is crucial to formulating and testing evolutionary hypotheses concerning
415 sex differences in the skeleton. With its focus on just some of the drivers of skeletal
416 development, this essay is only one step towards expanding our evolutionary explanation for sex
417 differences in skeletal growth.

418
419 Answers to questions about sex differences in the human skeleton should include what is
420 increasingly known about the evolution of gonads and genitals, their growth at puberty, and their
421 functions during skeletal maturation, sexual intercourse, the menstrual cycle, pregnancy, and
422 menopause, as well as the sensitivities of different tissues to estrogens and androgens. The basis
423 for the existence of sex differences deserves more attention before it will be possible to explain
424 why humans have a certain degree of sex difference compared to other species. This may require
425 some evolutionary research that breaks free from Mayr's proximate-ultimate convention. Laland
426 et al.¹² write that, "progress within biology demands dismantling of Mayr's identification of
427 proximate with ontogenetic processes and ultimate with evolutionary processes." (p. 1516) The
428 so-called "proximate" causes of sex differences in the skeleton are not only as much evolutionary
429 ones as the "ultimate," but they hold great potential to advance investigations into how male
430 competition and childbirth feature in the evolution of skeletal sex differences in humans.
431 If we do not hold evolutionary hypotheses to higher standards, while also including all biology
432 into the category of "evolution," then so many "ultimate" answers to important questions in
433 human evolution will loom larger and longer than they deserve.

434
435 Finally, a human evolutionary narrative that expands to include the present state of knowledge
436 about skeletal and reproductive biology and their harmonious development is not just better
437 science. It is also less likely than the traditional scientific view to unintentionally evoke or
438 reinforce unscientific beliefs about genetic determinism and genetic essentialism in the zeitgeist.
439 An updated answer to why there are sex differences in the human skeleton is less likely to be
440 interpreted to justify cultural conceptions of masculinity, femininity, and rigid binaries of sex
441 and gender with "human nature." If we improve the scientific explanations of visible sex
442 differences, then they are less likely to inspire unscientific beliefs about invisible ones. Fewer
443 minds would leap illogically from 'men are taller' to '*men* evolved for competition and
444 dominance'. Likewise, fewer would observe that women are broader and conclude that '*women*
445 evolved for reproduction.' As we advance science and its dissemination, fewer will mistake the
446 human body for a blueprint for the patriarchy.

447 448 **Acknowledgments**

449 I am grateful to seven anonymous reviewers and Jason Kamilar for providing feedback that
450 improved this paper. I am grateful to Lynn Copes and the Frank H. Netter School of Medicine
451 for allowing me to investigate some of the anatomy described here. For what I learned, I am
452 grateful to all I have cited. For their relevant influence, I thank Kevin Stacey, Cynthia Taylor,
453 Sharon DeWitte, Cara Wall-Scheffler, Joan Richtsmeier, Max Lambert, T. Ryan Gregory, Anna
454 Warrener, and Sarah Hrdy. Thanks to Anonymous for naming these pelvic ideas the VAGGINA
455 (Virile, Active Gonads and Genitals...) Hypothesis. Any shortcomings or errors are mine and
456 there are no conflicts of interest to declare.

457
458
459
460
461
462
463
464
465
466
467
468
469
470
471
472
473
474
475
476
477
478
479
480
481
482
483
484
485
486
487
488
489
490
491
492
493
494
495
496
497
498
499
500
501

References

1. Hill AK, Bailey DH, and DA Puts. 2017. Chapter 15: Gorillas in our midst? Human sexual dimorphism and contest competition in men. In: Tibayrenc M and FJ Ayala, editors. *On Human Nature: Biology, Psychology, Ethics, Politics, and Religion*: Academic Press, Amsterdam, Pages 235-249
2. Dunsworth HM. 2016. Chapter 2: The ‘obstetrical dilemma’ unraveled. In Trevathan W and K Rosenberg, editors: *Costly and Cute: Helpless infants and human evolution*. Santa Fe: School for Advanced Research, p 29-50.
3. Boyd R, Silk JB. 2015. *How Humans Evolved*, 7th edition. New York: WW Norton & Co.
4. Larsen CS. 2016. *Essentials of Physical Anthropology*. New York: WW Norton & Co.
5. Stanford C, Allen JS, Anton SC. 2017. *Biological Anthropology: The Natural History of Humankind*, fourth edition. Boston: Pearson.
6. Darwin C. 1871. *Descent of Man, and Selection in Relation to Sex*. London: Murray.
7. Hamlin K. 2014. *From Eve to Evolution: Darwin, science and women’s rights in gilded age America*. Chicago: University of Chicago Press.
8. Blackless M, Charuvastra A, Derryck A, Fausto-Sterling A, Lauzanne K, and E Lee. 2000. How sexually dimorphic are we? Review and Synthesis. *Am J Hum Biol* 12: 151-166.
9. Fausto-Sterling A. 2018. Why sex is not binary. *The New York Times*
<https://www.nytimes.com/2018/10/25/opinion/sex-biology-binary.html>
10. Astorino CM. 2019. Beyond Dimorphism: Sexual Polymorphism and Research Bias in Biological Anthropology. *American Anthropologist* 121(2): 489-490.
11. Mayr E. 1961. Cause and effect in biology. *Science* 134: 1501
12. Laland KN, Sterelny K, Odling-smee J, Hoppitt W, and T Uller. 2011. Cause and effect in biology revisited: Is Mayr’s proximate-ultimate dichotomy still useful? *Science* 334: 1512-1516.
13. Department of Health and Human Services. 2002. 2000 CDC Growth Charts for the United States: Methods and Development. *Vital Health Statistics* 11(246):
https://www.cdc.gov/nchs/data/series/sr_11/sr11_246.pdf
14. Bogin B. 1999. Evolutionary perspective on human growth. *Ann Rev Anthropol* 28: 109-53.

- 502 15. Bogin B, Varea C, Hermanussen M, and C Scheffler. 2018. Human life course biology: A
503 centennial perspective of scholarship on the human pattern of physical growth and its place in
504 human biocultural evolution. *Am J Phys Anthropol* 165: 834-854.
505
- 506 16. Cabrera SM, Bright GM, Frane JW, Blethen SL, and PA Lee. 2014. Age of thelarche and
507 menarche in contemporary US females: a cross-sectional analysis. *J Pediatr Endocrinol Metab*
508 *27(0): 47-51.*
509
- 510 17. Madimenos FC, Snodgrass JJ, Liebert MA, Cepon TJ, and LS Sugiyama. 2012. Reproductive
511 effects on skeletal health in Shuar females of Amazonian Ecuador: A life history perspective.
512 *Am J Hum Biol* 24: 841-852.
513
- 514 18. Workman M and K Kelly. 2017. Heavier birth weight associated with taller height but not
515 age at menarche in US females born 1991-1998. *Am J Hum Biol* 29(5). doi: 10.1002/ajhb.22999.
516
- 517 19. Cutler GB. 1997. The role of estrogen in bone growth and maturation during childhood and
518 adolescence. *J Steroid Biochem Molec Biol* 61(3-6): 141-144.
519
- 520 20. Rogol, AD, Roemmich JN, and PA Clark. 2002. Growth at puberty. *J Adolescent Health* 31:
521 192-200.
522
- 523 21. Rochira V, Kara E, and C Carani. 2015. The endocrine role of estrogens on human male
524 skeleton. *Intl J Endocrinol* 2015: 165215.
525
- 526 22. Weise, M., De-Levi, S., Barnes, K. M., Gafni, R. I., Abad, V., & Baron, J. (2001). Effects of
527 estrogen on growth plate senescence and epiphyseal fusion. *Proceedings of the National*
528 *Academy of Sciences of the United States of America*, 98(12), 6871-6.
529
- 530 23. Singh, Divya & Sanyal, Sabyasachi & Naibedyia, Chattopadhyay. (2010). The role of
531 estrogen in bone growth and formation: Changes at puberty. *Cell Health and Cytoskeleton*. 3.
532 10.2147/CHC.S8916.
533
- 534 24. Nilsson, O., Weise, M., Landman, E. B., Meyers, J. L., Barnes, K. M., & Baron, J. (2014).
535 Evidence that estrogen hastens epiphyseal fusion and cessation of longitudinal bone growth by
536 irreversibly depleting the number of resting zone progenitor cells in female rabbits.
537 *Endocrinology*, 155(8), 2892-9.
538
- 539 25. Baron J, Sävendahl L, De Luca F, Dauber A, Phillip M, Wit JM, and O Nilsson.2015. Short
540 and tall stature: a new paradigm emerges. *Nat Rev Endocrinol* 11: 735-746.
541
- 542 26. Nilsson O, Marino R, De Luca F, Phillip M, and J Baron.2005. Endocrine regulation of the
543 growth plate. *Horm Res* 64: 157-165.
544
- 545 27. Katchadourian H. 1977. *The Biology of Adolescence*. San Francisco: Freeman.
546

- 547 28. Schulster M, Bernie AM, and R Ramasamy. The role of estradiol in male reproductive
548 function. *Asian J Androl* 18: 435-440.
549
- 550 29. Carreau S, Wolczynski S, and I Galeraud-Denis. 2010. Aromatase, oestrogens and human
551 male reproduction. *Phil Trans R Soc B* 365: 1571-1579.
552
- 553 30. Solomon SJ, Kurzer MS, and DH Calloway. 1982. Menstrual cycle and basal metabolic rate
554 in females. *Am J Clin Nutr* 36: 611-616.
555
- 556 31. Dalvit SP. 1981. The effect of the menstrual cycle on patterns of food intake. *Am J Clin Nutr*
557 34: 1811-15.
558
- 559 32. Johnson WG, Corrigan SA, Lemmon CR, Bergeon KB, and AH Crusco. 1994. Energy
560 regulation over the menstrual cycle. *Phys and Behav* 65(3): 523-527.
561
- 562 33. Strassmann B. 1996a. Energy economy in the evolution of menstruation. *Evol Anthropol* 5:
563 157-164.
564
- 565 34. Strassmann B. 1996b. The evolution of endometrial cycles and menstruation. *Q Rev Biol* 71:
566 181-220.
567
- 568 35. Clancy KBH. 2009. Reproductive ecology and the endometrium: Physiology, variation, and
569 new directions. *Am J Phys Anthropol* 140(S49): 137-154
570
- 571 36. Reiches M, et al. 2013. The adolescent transition under energetic stress: Body composition
572 tradeoffs among adolescent women in The Gambia. *Evol Med Public Health* 2013(1):75-85.
573
574
- 575 37. Lange IG, Hartel A, and HHD Meyer. Evolution of oestrogen functions in vertebrates. *J*
576 *Steroid Biochem & Molec Biol* 83: 219-226.
577
- 578 38. Leigh SR and BT Shea. 1995. Ontogeny and evolution of adult body size dimorphism in
579 apes. *Am J Primatol* 36: 37-60.
580
- 581 39. Ellison P, et al. 2012. Puberty as a life history transition. *Annals of Human Biology* 39(5):
582 352-360.
583
- 584 40. Darwin C. 1871. *Descent of Man, and Selection in Relation to Sex*. London: Murray.
585
- 586 41. Plavcan JM. 2001. Sexual dimorphism in primate evolution. *Yrbk Phys Anthropol* 44:25-53.
587
- 588 42. Plavcan JM. 2012. Body size, size variation, and sexual size dimorphism in early Homo.
589 *Curr Anthropol* 53(S6): S409-S423.
590
- 591 43. Slatkin M. 1984. Ecological causes of sexual dimorphism. *Evolution* 38(3): 622-630.
592

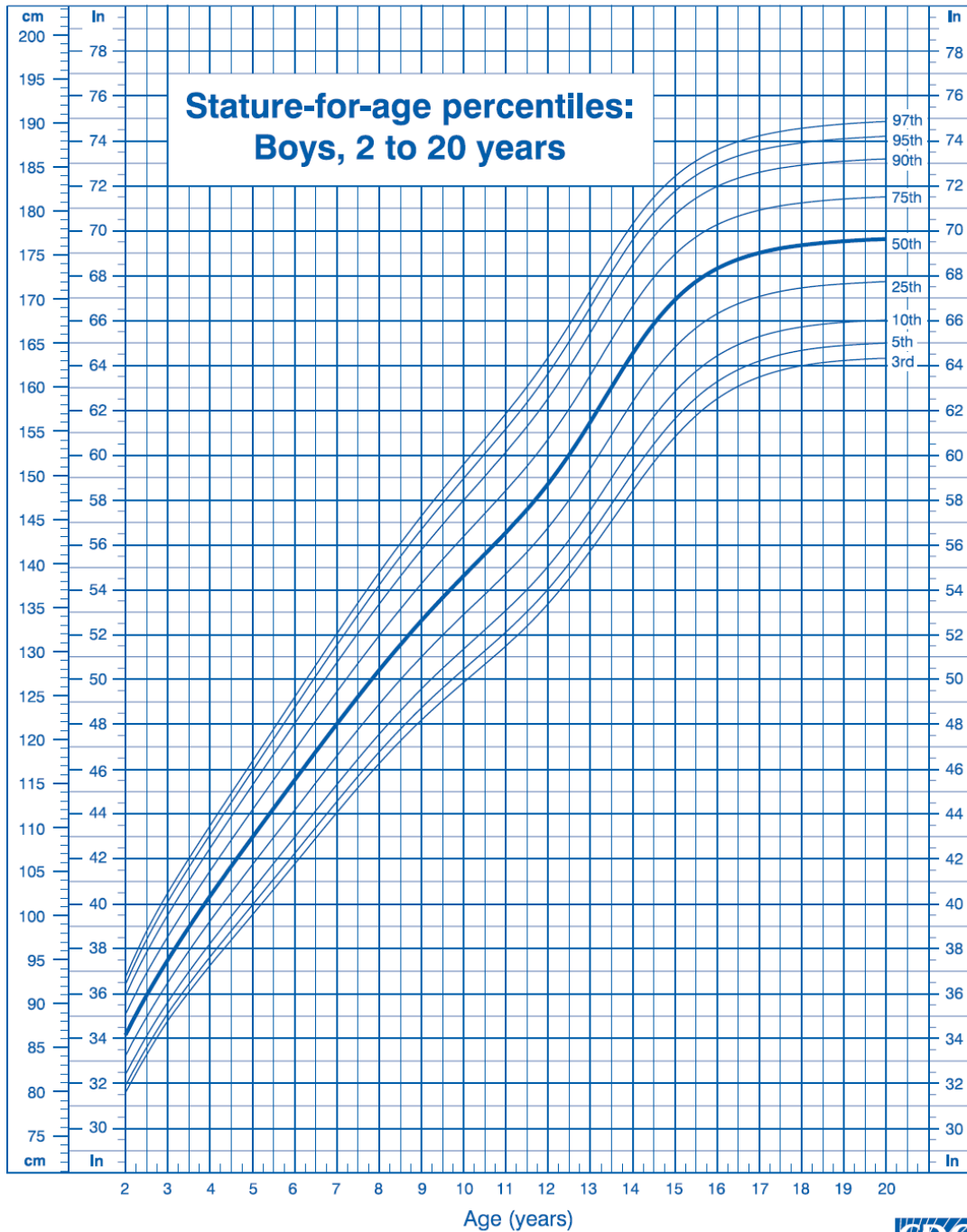
- 593 44. Gaulin SJC and LD Sailer. 1985. Are females the ecological sex? *American Anthropologist*
594 87:111-119.
595
- 596 45. Gordon AD, Johnson SE, and EE Lewis. 2013. Females are the ecological sex: Sex-specific
597 body mass ecogeography in wild sifaka populations (*Propithecus* spp.). *Am J Phys Anthropol*
598 151: 77-87.
599
- 600 46. Dunsworth HM, Warrener AW, Deacon T, Ellison PT, and H Pontzer. 2012. Metabolic
601 hypothesis for human altriciality. *Proc Natl Acad Sci USA* 109 (38) 15212-15216.
602
- 603 47. Wall-Scheffler CM and MJ Myers. 2017. The biomechanical and energetic advantages of a
604 mediolaterally wide pelvis in women. *Anat Rec* 300: 764-775.
605
- 606 48. Clutton-Brock TH, Harvey PH, and B Rudder. 1977. Sexual dimorphism, socioeconomic sex
607 ratio and body weight in primates. *Nature* 269: 797-800.
608
- 609 49. Stearns SC. 1992. *The evolution of life histories*. Oxford: Oxford University Press.
610
- 611 50. Wood JW. 1994. *Dynamics of Human Reproduction*. New York: Aldine de Gruyter.
612
- 613 51. Stulp G and L Barrett. 2016. Evolutionary perspectives on human height variation. *Biol Rev*
614 91: 206-234.
615
- 616 52. Wood AR, Esko T, Yang J, Vedantam S, Pers TH, Gustafsson S, et al. 2014. Defining the
617 role of common variation in the genomic and biological architecture of adult human height. *Nat*
618 *Genet* 46(11): 1173-1186.
- 619 53. Stulp G, Buunk AP, Verhulst S, Pollet TV (2015) Human Height Is Positively Related to
620 Interpersonal Dominance in Dyadic Interactions. *PLoS ONE* 10(2): e0117860.
621 <https://doi.org/10.1371/journal.pone.0117860>
622
- 623 54. Moffett EA. 2017. Dimorphism in the size and shape of the birth canal across anthropoid
624 primates. *Anat Rec* 300: 870-889.
625
- 626 55. Walrath D, Glantz M. 1996. Sexual dimorphism in the pelvic midplane and its relationship to
627 Neandertal reproductive patterns. *Am J Phys Anthropol* 100(1): 89-100.
628
- 629 56. Walrath D. 2003. Rethinking Pelvic Typologies and the Human Birth Mechanism. *Curr*
630 *Anthropol* 44(1): 5-31.
631
- 632 57. Betti L and A Manica. 2018. Human variation in the shape of the birth canal is significant
633 and geographically structured. *Proc R Soc B* 285: 20181807.
634
- 635 58. Delprete H. 2017. Pelvic inlet shape is not as dimorphic as previously suggested. *Anat Record*
636 300: 706-715.
637
638

- 639 59. Cunningham C, Scheurer L, and S Black. 2016. *Developmental Juvenile Osteology*, 2nd
640 Edition. Amsterdam: Academic Press.
- 641
- 642 60. LaVelle M. 1995. Natural selection and developmental sexual variation in the human pelvis.
643 *Am J Phys Anthropol* 98(1):59-72.
- 644
- 645 61. Greulich WW and H Thoms. 1944. The Growth and Development of the Pelvis of Individual
646 Females Before, During, and After Puberty. *Yale J Biol Med* 17(1): 91–98.8.
- 647
- 648 62. Huseynov A, Zollikofer CP, Coudyzer W, Gascho D, Kellenberger C, Hinzpeter R, and MS
649 Ponce de León. 2016. Developmental evidence for obstetric adaptation of the human female
650 pelvis. *Proc Natl Acad Sci USA* 113(9): 5227-5232.
- 651
- 652 63. Ellison PT. 2017. Endocrinology, energetics, and human life history: A synthetic model.
653 *Horm Behav* 91:97-106.
- 654
- 655 64. Polese B, Gridelet V, Araklioti E, Martens H, Perrier d’Hauterive S, and V Geenen. 2014.
656 The endocrine milieu and CD4 T-lymphocyte polarization during pregnancy. *Front*
657 *Endocrinol* <https://doi.org/10.3389/fendo.2014.00106>
- 658
- 659 65. Best KC, Garvin HM, and LL Cabo. 2018. An investigation into the relationship between
660 human cranial and pelvic sexual dimorphism. *J Forensic Sci* 63(4): 990-1000.
- 661
- 662 66. Smith P, Heimer G, Norgren A, and U Ulmsten. 1990. Steroid hormone receptors in pelvic
663 muscles and ligaments in females. *Gynecol Obstet Invest* 30: 27-30.
- 664
- 665 67. Smith P, Heimer G, Norgren A and U Ulmsten. 1993. Localization of steroid hormone
666 receptors in the pelvic muscles. *Eur J Obstet Gynecol Reprod Biol.*50: 83-85.
- 667
- 668 68. Grigoriadis C, Hassiakos D, Bakas P, Tympa A, Panoulis C, Cretsas G, Kondi-Pafiti A,
669 Liapis A. 2015. Effect of gonadal steroid receptors alterations on the pathophysiology of pelvic
670 organ prolapse and urinary incontinence. *Minerva Ginecologica* 68(1) 37-42.
- 671
- 672 69. Lang JH, Zhu L, Sun ZJ, Chen J. 2003. Estrogen levels and estrogen receptors in patients
673 with stress urinary incontinence and pelvic organ prolapse. *International Journal of Obstetrics*
674 *and Gynecology* 80(1): 35-39.
- 675
- 676 70. Ewies AA, Thompson J, Al-Azzawi F. 2004. Changes in gonadal steroid receptors in the
677 cardinal ligaments of prolapsed uteri: immunohistomorphometric data. *Hum Reprod* 19(7):
678 1622-8.
- 679
- 680 71. Smith P, Heimer G, Norgren A and U Ulmsten. 1993. The round ligament: a target organ for
681 steroid hormones. *Gynecol Endocrinol* 7: 97-100.
- 682
- 683 72. Ferlin A, Pepe A, Facciolli A, Giancesello L, and C Foresta. 2010. Relaxin stimulates
684 osteoclast differentiation and activation. *Bone* 504-513.

685
686 73. Tague RG. 1988. Bone resorption of the pubis and preauricular area in humans and
687 nonhuman mammals. *Am J Phys Anthropol* 76: 251-267.
688
689 74. Sulak O, Cosar F, Malas MA, Cankara N, Cetain E, and SM Tagil. 2007. Anatomical
690 development of the fetal uterus. *Early Hum Devel* 83: 395-401.
691
692 75. Martins W, Leite SP, and CO Nastri. 2009. Pelvic ultrasonography in children and
693 teenagers. *Radiol Bras* 42(6): <http://dx.doi.org/10.1590/S0100-39842009000600014>
694
695 76. Herter LD, Golendziner E, Flores JAM, Becker E, and PM Spritzer. 2002. Ovarian and
696 uterine sonography in healthy girls between 1 and 13 years old: Correlation of findings with age
697 and pubertal status. *AJR* 178: 1531-1536.
698
699 77. Cacciatore B, Apter D, Alftan H, and U-H Stenman. 1991. Ultrasonic characteristics of the
700 uterus and ovaries in relation to pubertal development and serum LH, FSH, and estradiol
701 concentrations. *Adolesc Pediatr Gynecol* 4:15-20.
702
703 78. Farage M and H Maibach. 2006. Lifetime changes in the vulva and vagina. *Arch Gynecol*
704 *Obstet* 273: 195-202.
705
706 79. Merz E, Miric-Tesanic D, Bahlmann F, Weber G, and S Wellek. 1996. Sonographic size of
707 uterus and ovaries in pre- and postmenopausal females. *Ultrasound Obstet Gynecol* 7:38-42.
708
709 80. Piironen O and HL Kaihola. 1075. Uterine size measured by ultrasound during the menstrual
710 cycle. *Acta Obstet Cyneec Scand* 54: 247-250.
711
712 81. Mazloomdoost D and RN Pauls. 2015. A comprehensive review of the clitoris and its role in
713 female sexual function. *Sex Med Rev* 3: 245-263.
714
715 82. Xia S-J, Xu X-X, Teng J-B, Xu C-X, and X-D Tang. 2002. Characteristic pattern of human
716 prostatic growth with age. *Asian J Androl* 4: 269-271.
717
718 83. http://w-radiology.com/female_pelvis_mri.php; http://w-radiology.com/male_pelvis_mri.php
719
720 84. Richtsmeier JT and K Flaherty. 2013. Hand in glove: brain and skull in development and
721 dysmorphogenesis. *Acta Neuropathol* 125: 469-489.
722
723 85. Moss ML and RW Young. 1960. A functional approach to craniology. *Am J Phys Anthropol*
724 18:281-92.
725
726 86. Andrew GM, Becklake MR, Guleria JS, and DV Bates. 1972. Heart and lung functions in
727 swimmers and nonathletes during growth. *J Appl Physiol* 32(2): 245-251.
728
729 87. Armour J, Donnelly PM, and PTP Bye. 1993. The large lungs of elite swimmers: an
730 increased alveolar number? *Eur Respir J* 6: 237-247.

731
732 88. Tague RG. 2005. Big-bodied males help us recognize that females have big pelves. *Am J*
733 *Phys Anthropol* 127: 392-405.
734
735 89. Schultz A. 1949. Sex differences in the pelves of primates. *Am J Phys Anthropol* 7(3): 401-
736 424
737
738 90. Hafez ESE and S Jaszczak. 1972. Comparative anatomy and histology of the cervix uteri in
739 non-human primates. *Primates* 13(3): 297-316.
740
741 91. Van Der Schoot P. 1996. Human (and some other primates') uterine teres ligament
742 represents a mammalian developmental novelty. *Anat Rec* 244: 402-415.
743
744 92. Kurki HK. 2007. Protection of obstetric dimensions in a small-bodied human sample. *Am J*
745 *Phys Anthropol* 133(4): 1152-1165.
746
747 93. Fischer B and P Mitteroecker. 2015. Covariation between human pelvis shape, stature, and
748 head size alleviates the obstetric dilemma. *Proc Natl Acad Sci* 112: 5655-5660.
749
750 94. Smith RJ. 2016. Explanations for adaptations, just-so stories, and limitations on evidence in
751 evolutionary biology. *Evol Anthropol* 25: 276-287.
752
753

754 **Figure 1.** Stature of boys (a) and girls (b) in America.¹³ See also Bogin¹⁴ and Bogin et al.¹⁵
 755
 756 (a)



Published May 30, 2000.

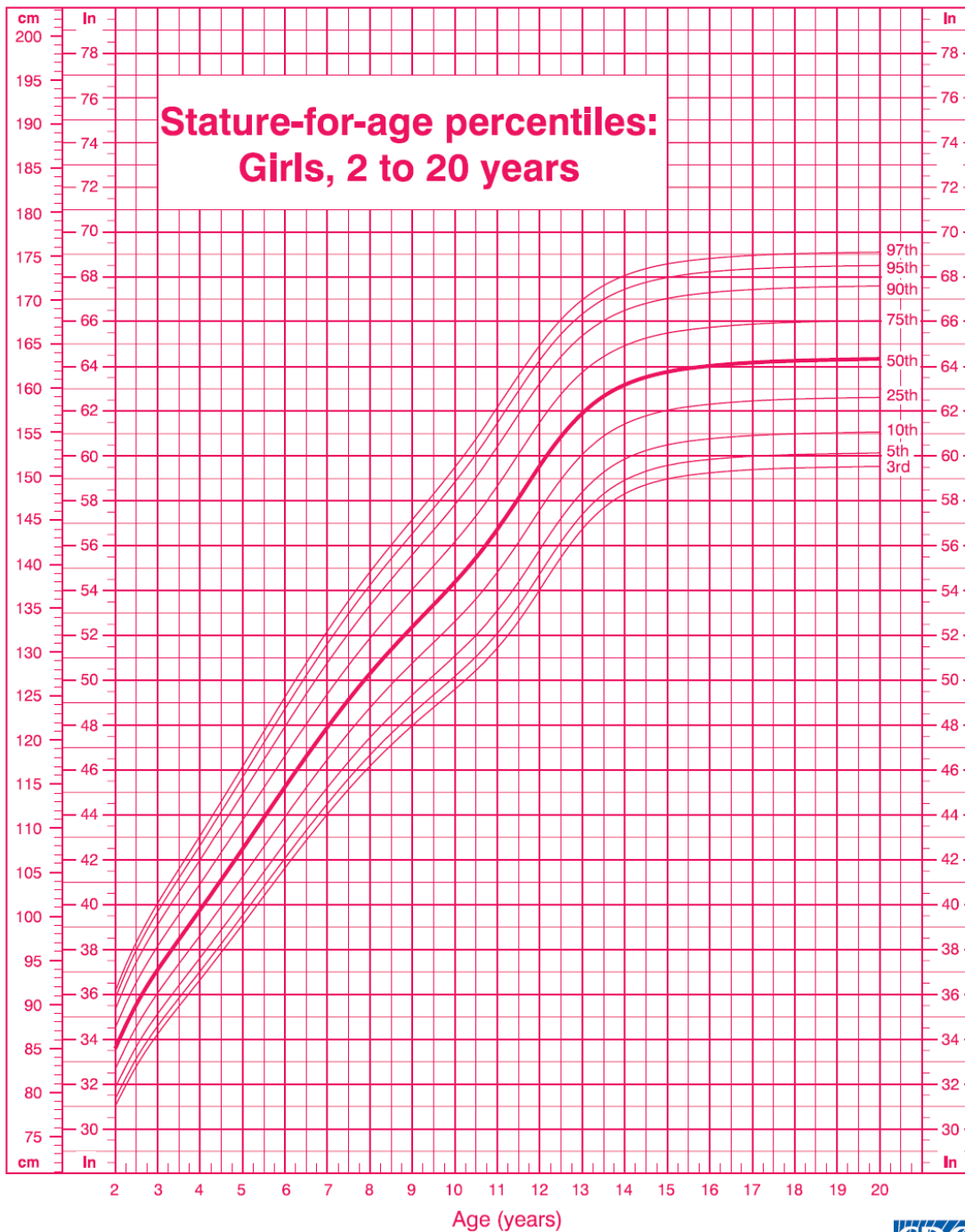
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).



SAFER • HEALTHIER • PEOPLE™

Figure 11. Individual growth chart 3rd, 5th, 10th, 25th, 50th, 75th, 90th, 95th, 97th percentiles, 2 to 20 years: Boys stature-for-age

757
 758
 759



Published May 30, 2000.
SOURCE: Developed by the National Center for Health Statistics in collaboration with
the National Center for Chronic Disease Prevention and Health Promotion



Figure 12. Individual growth chart 3rd, 5th, 10th, 25th, 50th, 75th, 90th, 95th, 97th percentiles, 2 to 20 years: Girls stature-for-age