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Chapter 27: There Is No Evolutionary “Obstetrical Dilemma”

Holly Dunsworth

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Introduction

Even for those who have never heard of the evolutionary hypothesis known as the “obstetrical dilemma” (or just “obstetric dilemma” or OD), its logic may ring a bell and can make a lot of sense given how difficult human childbirth can be. It is especially familiar to students and practitioners of anthropology, biology, evolution, and anatomy as it has been a widespread narrative in these disciplines for decades, influencing evolutionary medicine and evolutionary psychology, as well as the pregnancy, childbirth, and parenting industries.

Here is a synopsis of the OD: *Natural selection for ever-increasing brain size and for narrow bipedal hips has created a dilemma that humans solve by ending gestation early before the big-brained neonate outsizes the bipedal birth canal. The difficult dangerous births of helpless, underdeveloped infants serve as evidence of this evolutionary dilemma and its evolutionary solution.* For many who hear it, the OD has been a welcome antidote to the *Book of Genesis*. Difficult labor, dangerous childbirth, and helpless babies are no longer Eve’s fault, they’re evolution’s (Dunsworth 2016a).

Though the ideas were percolating early in the history of physical anthropology, like in Wilton Krogman’s paper “The Scars of Human Evolution” (1951), Sherwood Washburn (1960) is usually credited for conceiving of the OD. In his wake but without using the term “obstetrical dilemma,” highly influential evolutionary biologist Stephen Jay Gould relied on and argued for the OD both in professional and popular contexts (Gould 1977; 2007). Perhaps more than anyone else, Gould is responsible for the widespread assumption, within and beyond academia, that humans are born especially “early” and deserve a unique explanation: the bipedal pelvis.

In 1995, Karen Rosenberg and Wenda Trevathan wrote the classic article, “Bipedalism and human birth: The obstetrical dilemma revisited.” In it, they described how the twisting of the fetus helped a great deal in navigating birth but also in contributing to its difficulty. The twist added a new kind of issue that is mitigated with assistance. Along with the human propensity to birth socially, Rosenberg and Trevathan also pointed out a weakness in the OD’s narrow focus on neonatal brain size; it’s not just the baby’s head, but the overall large size, including especially its shoulders, contributing to childbirth difficulty. Rosenberg and Trevathan’s perspectives, steeped in midwifery experience and life experience, complicated the evolutionary narrative of human childbirth and weakened the OD. And then in 2012, two independent teams (Dunsworth et al., 2012; Wells et al., 2012) published papers that provided additional arguments against the classic OD, followed by additional biomechanical studies of pelvic morphology and bipedalism (Warrener et al., 2015), which was sufficient evidence to lead a third, independent group to declare the “falsification of Washburn’s obstetrical dilemma hypothesis” (Ponce de León et al., 2016). Since the initial offering in 2012, I have made contributions that deconstruct the “logic” of the OD (Dunsworth and Eccleston 2015; Dunsworth 2016a, 2016b, 2016c, 2018). Recent papers on the evolution of human childbirth and neonatal development have dropped the OD as a framework, using new language to refer to childbirth difficulty like “the obstetric conundrum” (Grunstra et al 2019), while others, like Nowell and Kurki (2020) have titled a paper in a way that signals a shift in the field: “Moving beyond the obstetrical dilemma

hypothesis...” Still, the OD remains. Researchers continue to refer to difficult childbirth as “the obstetrical dilemma” and they do so explicitly or implicitly within the OD framework (e.g. Wells 2017; Wells 2020). That is, when “obstetrical dilemma” is employed as a synonym for childbirth difficulty or the tightness of fit between bony birth canal and neonate, the authors are typically assuming that the pelvis shortened human pregnancy and that our neonates are born early (a.k.a. the OD).

Peering at it from the present, it is clear that preference, bias, and evidence-free assumptions (a.k.a. untested and unsupported hypotheses) help to sustain the OD. Here we journey through current knowledge built in diverse fields within and beyond anthropology that fails to support the existence of an “obstetrical dilemma.” When it comes to human evolutionary narratives, the sociocultural consequences are as important as the scientific truth. So, the aim here is not just to approximate a true reconstruction of human evolutionary history, but to approach one that fails to discourage (a.k.a. supports) physiologic birth (American College of Nurse-Midwives 2013; Rutherford et al. 2018) for those birthers who wish to engage in it. As of now, the most widespread evolutionary story around childbirth is the OD, which perpetuates the false impression that women’s bodies are evolutionarily compromised and that medical interventions are inevitable or imperative.

Humans Are not Born Early and Gestation Is not Truncated

There are fundamental problems with the “dilemma” aspect of the OD, but we will begin by questioning the basis for its “solution.”

The long human pregnancy and the large human neonate defy OD expectations that a dilemma has been, however imperfectly, solved by birthing human babies early. Of all the primates, the great apes have the longest pregnancies (ranging across species from roughly 30 to 39 weeks), and among them humans lie at the long end, with maybe a few weeks more. Human babies have the most fat of the baby primates (Kuzawa 1998) and this contributes to our absolutely largest neonatal body size of all baby primates. After a long gestation, the human newborn brain is the absolute largest of the newborn primates and is already nearly as large as an adult chimpanzee’s (reviewed in Dunsworth et al. 2012).

Despite the absolutely large newborn human brain, our scientific tradition in these matters—led by Adolph Schultz (1949), Adolf Portmann (1969), and Stephen Jay Gould (1977)—emphasizes its *relatively* small size. Gould’s book *Ontogeny and Phylogeny* and his popular essays (1977; 1977/2007) were read beyond anthropological and human evolutionary biology circles and famously argued that human gestation is truncated. In the tradition of Gould’s wide and compelling influence, scientists and non-scientists continue to assume that fetuses really should be gestating for longer.

For a baby primate, humans are indeed relatively small-brained at birth; that is, the human newborn has the smallest fraction of its adult brain size of the great apes (DeSilva and Lesnik 2006). But there is little else about human pregnancy or neonatal developmental status that supports a designation of “early” birth, and especially the implication that it holds a unique status among primates.

The more we learn about human neonates and as they compare to other primates, the harder it becomes to agree with arguments, like his and others’, that humans are exceptionally “secondarily altricial” (Portmann 1969). Deeming humans “secondarily altricial” suggests our newborns share significant traits in common with wolves and rats to hold us apart from the rest of the primates which fall on the precocial end of the developmental spectrum (with horses).

Gould titled a popular essay, “Human babies as embryos” about our first nine months of life (1977/2007). He also claimed that human “babies are as helpless and undeveloped at birth as those of most altricial mammals” (Gould 1977/2007, 71). Though Gould did not use the term “obstetrical dilemma” in these discussions, Gould’s perspective was influenced by and incorporated into the OD. Echoing Washburn, Gould asserted, effortlessly, that our early birth and our altriciality were caused by our bipedal pelves that shortened our gestation.

However, contrary to Gould’s claims, we have only superficialities, like our naked skin, in common with actual altricial newborn mammals like rat pups, which have closed eyes and ears. Altricial mammals are born prior to peak brain growth rate, but precocial mammals including humans, like the rest of the primates, are born after that peak (Halley 2017). As toddlers, humans develop bipedalism at a pace expected for a mammal of our brain mass (Garwicz et al. 2009).

Human newborns are odd and deserve intense scientific investigation, but we are not “altricial” or “secondarily” so (for much more detail on this topic see Trevathan and Rosenberg 2016a,b). Our relative helplessness at birth is influenced by our developing newborn brains and its relationship with motor-neuronal development. Humans have an extraordinarily long developmental period during which we grow and develop our extraordinarily large brains, but our condition does not require an exceptional explanation like the OD’s where our uniquely human bipedal pelves are the cause.

Primates with big adult brains have smaller relative brain sizes as newborns and take longer to develop their big adult brains than their relatives with smaller adult brains. Human newborns, infants, and toddlers experience this primate pattern of development, but with the additional burden of a massive head and without the aid of grasping feet. Our perception of newborn helplessness may not always be scientifically objective, either. It may reflect attitudes and physical conditions of WEIRD (Western, educated, industrialized, rich, democratic) parents of WEIRD children (Dunsworth and Eccleston 2015). Rather than assuming human newborns are embryonic or are developmentally equivalent to rat pups, one could marvel at what is increasingly understood about the sophisticated cognition employed by human babies as they navigate and manipulate their world (see, for example, Hrdy 2009; Gopnik et al 1999; Trevathan and Rosenberg 2016b).

Gould offers an explanation for humans by comparing us to chimpanzees, and if one only looks to chimpanzees, his arguments are compelling. Chimpanzees are born with about 40% of adult brain size, while we are only born with about 30% (or less). According to Gould and OD-thinking, humans should be born with just as much growth accomplished towards their adult-sized brain as those apes, and that we are not means something unique in our evolutionary history must be preventing it: the bipedal pelvis. After all, chimpanzees don’t have such a tight fit between bony birth canal and neonate. Our pelvis must be preventing us from birthing babies with as much brain growth accomplished as chimpanzees. So the thinking goes.

Gould’s claims about a truncated human gestation implicitly assume that the timing of birth is mechanistically or biologically similar to the timing of tooth eruption. That is, that the event of birth is comparable to developmental milestones like first molar eruption, the initiation of puberty, or menarche (first menstruation). And so, when birth happens earlier or later in the general primate developmental schedule, Gould is assuming that is a shift in some hypothetical clock-like mechanism that governs birth along with other developmental milestones. However, that approach to the timing of birth is not consensus.

Chimpanzee brain growth, birth, and neonatal development reveal bias in OD thinking when it comes to humans. Chimpanzees are the second most encephalized primate. Newborn chimpanzees need special care and attention from their mothers. With a small brain that is only about 40% of its future adult size, they have a long way to grow. They are immature in their ability to thermoregulate, benefiting greatly from mother's body as a heat source. They are completely reliant on their mother's body for nourishment through milk for many months before they routinely taste otherwise. With their developing motor skills, they are incapable of grasping on to their mother for significant periods of time. Chimpanzee mothers hold, carry, and cuddle their newborns and infants during these crucial early days, weeks, and months, extending into years of intense parental investment. Chimpanzees require high investment from their mothers and we assume this has a great deal to do with their relatively small brain size at birth and their stretched out postnatal developmental life history, associated with growing a large brain.

Perhaps because chimpanzees lack a tight fit between birth canal and neonate, their helpless babies have begged for no special explanation, like the OD. Further, it is never assumed that chimpanzees should be born with 50% of their adult brain growth achieved, like capuchin monkeys have at birth (Fragaszy 2004), or that because they are not larger brained at birth that their evolution requires a special explanation like the OD. Again, perhaps this is because chimpanzees do not have the difficult childbirth that humans do, and so there is nothing conspicuous, behaviorally or physically, that is constraining their newborn brain size. It has long been taken for granted that as monkeys, apes, and hominins encephalized, their postnatal brain growth increased and so their relative brain size at birth decreased. No one has ever asserted that increased encephalization should occur during gestational days alone. And yet, this is what Gould's argument implies—that in order to grow larger brains, our ancestors should have *reversed* primate tradition and accomplished increased brain growth *in utero*. To be clear, this is an assumption that human encephalization should have been unlike any other primate's. However, if one approaches human encephalization from a comparative primate perspective, one expects that with increasing adult hominin brain size there must have been decreasing relative brain size at birth, regardless of childbirth difficulty. Whatever is causing nonhuman primate gestation to end is likely similar to what ends human pregnancy, just with less birthday drama.

If the pelvis didn't limit newborn brain size or gestation length in chimpanzees and other primates too, then why should we believe that the tight fit in humans is anything more than a coincidence? Why should we believe that the human pelvis uniquely limited our gestation length and newborn brain size?

Women's Hips Are Not Compromised

The OD assumes that the bony birth canal is at its upper limit in volume and that human femaleⁱ pelvises—being more capacious than men's, but barely bigger than a neonate—are evidence that they are up against a threshold. Men's hips have been traditionally assumed to be ideal for bipedalism and assumed to be released from selection for successful childbirth. Women's hips have traditionally been assumed to be locked in a compromise between selection for bipedalism and birth. So many of these assumptions are based in an outdated approach to human sex differences where there are hypothetical "male genomes" and "female genomes" influencing differences in morphology between the hypothetical "female skeleton" versus the "male skeleton."

Gould summed up the OD's take on the evolutionary impact of the human female pelvis this way: "When one contemplates the radical redesign that human females would have to

undergo in order to give birth to year-old babies, the link of ‘early’ birth to difficult parturition seems quite reasonable” (Gould 1977, 369).

We can counter Gould’s contemplation with some of our own. For humans to birth our babies with 40% of adult brain size, as chimpanzees do, we would have to lengthen gestation seven more months to a pregnancy of 16 months. At seven months of age, we have 40% of our adult brain size (DeSilva and Lesnik 2006). Such a lengthened gestation would result in a newborn head diameter increase of 3-4 cm. Women already vary by this magnitude in dimensions of the bony birth canal (Simpson et al. 2008) and no one has correlated this to meaningful variation in their walking or running ability. Further, no one has yet demonstrated that increasing the present average in bony birth canal dimensions by 3-4 cm (slowly over deep evolutionary time so as to include soft tissues co-evolution and accommodation) would ruin bipedalism. Certainly the size of the shoulders and the overall size of the neonate contributes to the tight fit at birth, however, this thought experiment was focused on the brain because that has been the narrow focus of OD thinking.

As Gould demonstrates, the OD has benefited from compelling hypothetical assertions that are difficult if not impossible to test. Because humans have a bony birth canal, it is surely an upper limit on fetal size in the context of any unfortunate mother-neonate dyad. But, the existence of a tight fit between bony birth canal and neonate is not logical support for the idea that our pregnancies end early or that they end because of pelvic constraints. It is also not logical support for the assertion that something is preventing the pelvis from expanding to reduce the tight fit and the risks that come with it.

The traditional OD assumes that bipedal efficiency or economy or performance (or a combination of all three) is what keeps men’s hips narrow and is therefore what keeps women’s pelves too narrow to make childbirth easier. Small differences between the sexes in pelvic anatomy are assumed to correlate to small differences in performance and health, which are hypothesized to have been on selection’s radar, keeping women’s pelves from expanding and forcing a shortened gestation, so the thinking goes (Ruff, 2017).

One way to investigate these issues has been to test if women’s hips are less efficient or costlier or somehow worse at bipedalism than men’s in a biomechanical or kinematic context. Although it is not a direct test of natural selection, linking variation in pelvic anatomy to some measure of efficiency, or cost, or performance remains one of the only ways to investigate OD assumptions. Researchers in biomechanics and kinematics who are actively asking these questions have not demonstrated that wide or women’s hips are significantly worse than narrower ones or men’s (Warrener et al. 2015; Whitcome et al. 2017; Warrener 2017; Wall-Scheffler and Myers 2013; Wall-Scheffler and Myers 2017). Theoretical arguments that female human pelvic anatomy is constrained by bipedalism are still alive (Ruff 2017), but experimentally it has not worked out. What is more, careful studies of variation in pelvic anatomy of human skeletal remains have not found evidence that selection has especially stabilized (or constrained) females’ compared to males’ due to some compromise between bipedalism and birth (Kurki 2013; Kurki and Decrausaz 2016).

Bony pelvic anatomy is highly variable around the world, not just between the sexes (Betti and Manica 2018), and a growing hominin fossil record suggests that pelvic variation has been the norm for bipedalism over the last seven or so million years (Dunsworth and Eccleston 2015; Laudicina et al. 2019). Pelvic variation has long supported successful bipedalism, including that of actively foraging, parenting, and pregnant bipeds. Women’s bodies are highly

adapted for complex functionality, and they are not compromised (see also Wall-Scheffler 2012 and Kurki 2013).

Bipedalism's Pelvic Correlates Did Not Limit Pregnancy or Fetal Brain Growth

So far we have countered OD thinking that we are born early. And we have questioned the assumption that women's hips, which prevent our lineage's extinction, are somehow worse than men's. Partnered with that bias against women's bodies is the assumption that the pelvis could not expand to accommodate a larger neonate, or a present-day-sized neonate more easily. Because it is challenging to think of a test of that assumption, it is perhaps more productive to consider what could limit gestation and fetal growth if *not* the bony pelvis? That is, if we reject the premise that the pelvis is the cause, as we did above, we are still left without an evolutionary explanation for the timing of human birth. We can consider something less conspicuous than difficult childbirth and a tight fit between pelvis and neonate.

The larger the mother's body, the longer the pregnancy, which explains why the great apes have the longest pregnancies of all the primates (Dunsworth et al. 2012). Body mass is a useful proxy for metabolic rate, which enables and constrains a species' gestation length and fetal growth. Maternal mass (and, thus, metabolic rate) is just as fundamental to pregnancy in whales and dolphins (Sacher and Staffeldt 1974), which lack bony birth canals for constraining neonatal size and gestation length.

What Ellison (2001; Dunsworth et al. 2012) outlined to explain variation in gestation length and fetal growth within our species is a useful perspective for explaining differences between species. Ellison's "metabolic crossover hypothesis" proposes that human pregnancy ends when the fetus can no longer continue growing inside its mother, constrained by the maternal-fetal-placental metabolism. Maternal metabolic rate, energetic needs and fetal energetic needs increase throughout pregnancy. Then, just as the fetal needs are overshooting what the mother can provide, the pair are hitting the 9-10 months mark of pregnancy. Leading up to the end of pregnancy, mothers reach what looks like a metabolic ceiling, or limit on sustained, daily elevated metabolic rate, at about 2.1 times the basal metabolic rate—suggesting that mothers can only sustain a growing fetus at this constantly elevated metabolic rate for so many weeks, let alone while a fetus continues to increase in costliness. A mother is producing and pumping an additional 50% blood volume during pregnancy. She is feeding the growth of the fetus and placenta through her own diet. She is also breathing for the fetus, embodying the limit to the fetus's available oxygen supply and, thus, its energy. Constraints on fetal growth during pregnancy cannot be lifted by merely increasing a mother's dietary intake of calories or glucose. Once the infant is born they can breathe on their own and, paired with a diet of milk and eventually much more, they can grow to surpass what was physiologically possible *in utero*. In what we nicknamed the "EGG hypothesis" (energetics of gestation and fetal growth), we proposed that these phenomena we described in humans are similar across primates and placental mammals and help explain the constraints on their gestation and fetal growth as well (Dunsworth et al. 2012). These EGG constraints explain why primate encephalization is achieved with the help of additional postnatal brain growth. Perhaps our higher metabolic rate and penchant for metabolic endurance compared to our closest primate relatives (Pontzer et al. 2016; Thurber et al. 2019) explains the human capacity for long pregnancies that produce such large-bodied, large-brained babies.

Birthing Big Babies Did Not Cause Sex Differences in Pelvic Dimensions

In the course of my research on the obstetrical dilemma, I have helped to perpetuate the assumption that human females have big pelves due to selection for birthing big babies, and that otherwise they'd have narrower hips like males. I wrote: "Many participating in OD discussions are often left wondering why the birth canal does not widen to make childbirth easier or less dangerous. Perhaps it already has. Sexual dimorphism in the human pelvis is considered to be the result of natural selection for successful childbirth. A mother with too-narrow pelvic genes is not likely to pass those genes onto her children who could not pass through her pelvis" (Dunsworth 2016a). My thinking echoes a pithy quote from Tague: "Females have big pelves because they give birth to big babies" (Tague 2005). Here we will question whether sex differences in the human pelvis are caused by big babies and, thus, lend any support to the OD.

There are consistent and patterned sex differences in human pelvic morphology pertaining to the size of the space inside the pelvic cavity. The dimensions of the "true pelvis" or "birth canal" are relatively larger in females than in males. That is, on average, human female pelves have longer pubes, more laterally flaring ischial spines and tuberosities, and relatively shorter and wider sacra. Thus, they often have inlets (often measured from sacral promontory to the superior pubic symphysis; see **Figure 1**) and outlets (often measured from coccyx to inferior pubic symphysis or measured as the distance between ischial tuberosities; see **Figure 1**) that are relatively larger in diameter than those of males (Moffett 2017; Walrath and Glantz, 1996; Walrath 2003). These patterns persist globally despite geographic variation in human pelvic morphology (Betti and Manica 2018) and despite the fact that typical female pelvic inlet shape may be "android" which is to compare it to a male's (contra historical expectations that female pelves be distinctly "gynecoid" (Delprete 2017)). Fetal pelves, between seven months and birth, display sex differences in size and shape that already hint at those in adults (Cunningham et al. 2016). Sex differences in pelvic morphology become pronounced during the transition to adulthood and into adulthood (LaVelle 1995; Greulich and Thoms 1994; Huseynov et al. 2016).

It is not uncommon for scientists to assume that sex differences in human pelvic morphology are due to genetics. For example, Ponce de Leon et al. (2016) wrote that "we do not yet understand which mechanisms of sex-biased autosomal gene expression govern human pelvic development." As discussed above, this sort of assumption can spark thoughts of pre-programmed "female" as opposed to "male" skeletal morphology, as if such hypothetical genes build sex differences in the size and shape of the *space* between pelvic bones. It's as if the bony birth canal is an empty room in a house under construction, where the walls are built around the space that people plan to fill with furniture, or in the case of the OD, with a baby. But the blueprint is a bad metaphor for the genome and there is never an empty space between the pelvic bones until they're lying in a museum drawer. In life, and for their entire construction, the pelvic bones are always occupied by organs and tissues, and frequently with bodily waste. Recently, I have proposed that we consider how the sex differences in human pelves are influenced by the development and functioning of the organs and tissues within those pelves (Dunsworth 2020) and I briefly present those ideas here.

Beyond assumptions about hypothetical genes, estrogen is the established explanation for ontogenetic changes to female pelvic anatomy compared to what happens in males (Ellison 2017). But if estrogen is a primary driver of long bone growth and fusion in all humans, regardless of sex (reviewed in Dunsworth 2020), then we still need an explanation for how it acts locally just on the bones of the pelvis and only in females.

The muscles of the pelvic floor contain estrogen receptors, which suggests they are targets for estrogen, and they also contain estrogen receptors in their connective tissue cells.

Thus, the muscles and ligaments of the female pelvis likely influence the bones to which they are adjacent or anchored in ways that differ from other (non-pelvic) muscle- and ligament-bone interfaces, given the known effects that estrogen and relaxin (produced by ovaries and placentas) have on bone growth and remodeling. Furthermore, the additional pelvic volume occupied by female gonads and genitals may be causing the expansion of the true pelvis. The increase in size of internal gonads and genitals (throughout life and periodically and episodically during intercourse, the menstrual cycle, and pregnancy) may be spurring changes to the bones that form the cavity they occupy similar to the ways that organs and bones expand together elsewhere in the body, as with the brain inside the skull (Richtsmeier and Flaherty 2013), the eye inside the orbit, and the heart and lungs inside the ribcage. In sum, sex differences in the dimensions of the true pelvis are influenced by localized effects of estrogen and relaxin and also, potentially, arise due to the plasticity of the pelvic bones to accommodate the greater volume of developmentally and functionally dynamic gonads and genitals housed within the typical female pelvis.

Sex differences in pelvic dimensions are common across primates, not just humans (Moffett 2017), and so this phenomenon is based in phylogeny. Even chimpanzees, which have small enough neonates to fit through the male pelvis (inferred from measures published in Moffett 2017 and Schultz 1949), still have sex differences, with female pelvises being more capacious than males'. Still, humans seem to have more sex differences than expected. Moffett (2017) investigated whether primates with greater cephalopelvic proportions (size of neonatal head compared to pelvic inlet) had greater pelvic sex differences and they did, but humans have even more than is explained by cephalopelvic proportions. That is, primates like *Hylobates* have similar cephalopelvic proportions (a.k.a. tightness of fit) to humans, but exhibit smaller sex differences in the pelvis than we do.

It is possible that differences in type and magnitude of sex differences in primate pelvises (including humans) reflect differences in soft tissue anatomy (e.g. presence/absence of os clitoris/penis, uterus shape and flexion, descended testes), reproductive physiology and function (e.g. estrus swellings), and effects/amounts of estrogen and relaxin and their receptors in tissues of the pelvic region. Human sex differences in the pelvis could be more pronounced than expected compared to other primates' because of allometry, or because of the more tubular construction of the hominin pelvis, and how pelvises with and without internal female organs and greater estrogen exposure develop in the context of bipedal posture and gait. But currently there is no evidence to support the assumption that our big babies *caused* the existence, or level, of sex differences in our pelvises. It may be that our capacious pelvises *allowed for* the evolution of big newborns.

There Is No “Obstetrical Dilemma”

To sum up, maternal metabolism, physiology, and related energetic conditions are the major limiting factors of gestation and fetal growth. Though metabolic traits and processes vary among species, this is likely to be true across placental mammals. There is no scientific reason to assume that humans are born especially early. There is no evidence to support that human gestation is shortened or truncated. That is, there is no evidence that human metabolism/energetics is especially (uniquely) cutting gestation short, or that the pelvis is either. As far as we know now, we have evolved birth canals that can accommodate the size of the fetus that human metabolism has evolved to grow. Even if one could convincingly argue that human neonatal growth or gestation are cut short or that they have been limited over evolutionary history, there is still no evidence that the pelvis has been the cause. Helpless newborns are not evidence of the

OD. Even if someone does demonstrate that humans are at a maximum pelvic size, we still could not automatically assume that the pelvis has impacted human gestation length or neonatal size. Humans endure a tight fit at birth; it has not shaped the evolution of pregnancy length and fetal growth, it is not evidence that women's hips are compromised, it is not evidence that the birth canal is at an upper limit in size, and it is not evidence that the OD explains difficult childbirth, helpless neonates, or both. The presence of sex differences in pelvic anatomy are not evidence in support of the OD and they did not result from birthing big babies. That is, we cannot say big human babies caused sex differences in the pelvis.

Childbirth difficulty is not explained by the OD, but it still deserves an evolutionary and anthropological explanation. As Rosenberg and Trevathan (1995) have pointed out, the evolution of human shoulder morphology and its potential to obstruct labor inside a bipedal pelvis is an important part of the story alongside encephalization. Even more significant is the human habit of assisting birthers. Based on the few nearly complete and deeply ancient fossil hominin pelvises on record, it seems like the tight fit may have existed as early as three or four million years ago in some *Australopithecus* species (DeSilva 2011). However, Laudicina et al.'s (2019) recent analysis of the *Australopithecus sediba* birth process suggests that birth was not as difficult as it is for us and that neonates may not have twisted their way out of the relatively roomier birth canal.

While it is reasonable to believe that with an increase in neonatal brain and body size over hominin evolutionary history came an increase in childbirth difficulty, more recent history (over the last few thousand years) has likely contributed significantly to childbirth difficulty (for reviews and details to back up the discussion to follow, see Stone 2016; Wells et al. 2012; 2015; 2017; 2020). It is not known whether the increase in caesarean sections is realistically and positively correlated with an actual increase in cephalo- or fetal-pelvic disproportion as a cause for humans to perform those interventions. If an increase in the incidence of the impossibly tight fit is real, it may be due to a number of recent cultural phenomena. The effects of malnutrition (and other health risks during a woman's life that are exacerbated by forces like socioeconomic inequality) on pelvic under-development are often coupled with her healthy pregnancy supporting a large baby. How women have adorned, positioned, and moved their bodies during their development has affected their skeletal growth, like, for example, with the use of corsets and regarding variation in physical activity. Metabolic disease, including diabetes, gestational diabetes, and preeclampsia encourage the growth of large fetuses, but so might a glut of energy during pregnancy which is increasingly common due to lifestyle, diet, and lower stress and disease burden; that is to say, socioeconomic inequality is exacerbating childbirth difficulty. A birther's age, behavior, and position during pregnancy and labor are also contributing to childbirth difficulty. And the amount of quality experience of childbirth attendants and caregivers is a factor, especially as measured by use of medical interventions. Hospitals have been inhospitable to the time that labor frequently needs, with medical staff intervening in the birthing process before ample time has been granted to the birther-fetal-placental physiology (e.g. Bibeau 2014; Rutherford et al. 2019).

After just a brief consideration of factors contributing to childbirth difficult in extremely recent evolutionary history, it is important to consider how culture tempts us to believe that cephalopelvic and fetopelvic disproportion have been more of an obstacle than they actually are. The cultural amplification of perceived risk has lent credence to the "obstetrical dilemma" in human evolution, which has lent credence to the perception of women's bodies as being

compromised, which must be circling back and contributing to medicine's underestimation of women's bodies and over-implementation of childbirth interventions (Dunsworth 2018).

Evolution belongs in the discussion of childbirth difficulty and neonatal development, but not in terms of the OD. Evolutionary narratives have consequences. It is imperative that we get them right, even if that means complicating them beyond what is simple, intuitive, or compelling, and even if that means admitting that evolutionary cause and effect are extremely difficult to investigate scientifically. Anthropology's superpower is detecting culture's many superpowers, one of which is to masquerade as "nature." Culture (medicine, colonialism, capitalism, economic inequality, sexism, racism, etc...) helped to construct a childbirth dilemma and then packaged and sold it as a natural evolutionary "obstetrical dilemma," but there is none.

Figure 1. Superior view of a human pelvis, highlighting the "obstetric" dimensions where there are patterned sex differences.

Endnotes

ⁱ In this chapter 'female' refers to humans of all genders with anatomy that is commonly assigned to be female, and the same gender inclusivity applies to 'male'—with the understanding that neither sex nor gender divide into uniform, discrete, or binary categories, which is why "sex differences" rather than "sexual dimorphism" is employed (Blackless et al., 2000; Fausto-Sterling 2018; Astorino 2019).

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