

# The Obstetric Dilemma: An Ancient Game of Russian Roulette, or a Variable Dilemma Sensitive to Ecology?

Jonathan C.K. Wells,<sup>1\*</sup> Jeremy M. DeSilva,<sup>2</sup> and Jay T. Stock<sup>3</sup>

<sup>1</sup>Childhood Nutrition Research Centre, UCL Institute of Child Health, London WC1N 1EH, UK

<sup>2</sup>Department of Anthropology, Boston University, Boston, MA 02215

<sup>3</sup>Department of Archaeology and Anthropology, Cambridge CB2 3DZ, UK

**KEY WORDS** Birth; phenotypic plasticity; obstetric dilemma; encephalization; pelvis; cephalo-pelvic disproportion; nutrition; growth

**ABSTRACT** The difficult birth process of humans, often described as the “obstetric dilemma,” is commonly assumed to reflect antagonistic selective pressures favoring neonatal encephalization and maternal bipedal locomotion. However, cephalo-pelvic disproportion is not exclusive to humans, and is present in some primate species of smaller body size. The fossil record indicates mosaic evolution of the obstetric dilemma, involving a number of different evolutionary processes, and it appears to have shifted in magnitude between *Australopithecus*, Pleistocene *Homo*, and recent human populations. Most attention to date has focused on its generic nature, rather than on its variability between populations. We re-evaluate the nature of the human obstetric dilemma using updated hominin and primate literature, and then consider the contribution of phenotypic plasticity to variability in its magnitude. Both maternal pelvic dimensions

and fetal growth patterns are sensitive to ecological factors such as diet and the thermal environment. Neonatal head girth has low plasticity, whereas neonatal mass and maternal stature have higher plasticity. Secular trends in body size may therefore exacerbate or decrease the obstetric dilemma. The emergence of agriculture may have exacerbated the dilemma, by decreasing maternal stature and increasing neonatal growth and adiposity due to dietary shifts. Paleodemographic comparisons between foragers and agriculturalists suggest that foragers have considerably lower rates of perinatal mortality. In contemporary populations, maternal stature remains strongly associated with perinatal mortality in many populations. Long-term improvements in nutrition across future generations may relieve the dilemma, but in the meantime, variability in its magnitude is likely to persist. *Yrbk Phys Anthropol* 55:40–71, 2012. ©2012 Wiley Periodicals, Inc.

The notion that maternal pelvic dimensions are subject to powerful competing demands from reproduction and locomotion is widely accepted in the biomedical and anthropological literature (Krogman, 1951; Washburn, 1960; Schultz, 1969; Rosenberg, 1992; Rosenberg and Trevathan, 1996; Wittman and Wall, 2007; Walsh, 2008; Franciscus, 2009; Trevathan, 2011). The maternal pelvis is frequently considered to be subject to two counteracting evolutionary forces: decreased height and increased mediolateral breadth in order to optimize the biomechanics of locomotion, and increased anteroposterior dimensions in order to enable birth of the unusually encephalized human infant. The compromise imposed by these antagonistic demands manifests as a difficult passage of the fetal head through the birth canal (Wittman and Wall, 2007), resulting in the birth process being a more complex and lengthy procedure in humans than in closely related species of ape (Tevathan, 1988; Rosenberg, 1992; Rosenberg and Trevathan, 2002). The antagonistic interaction of bipedalism and encephalization has been assumed to have followed the emergence of the large *Homo* brain within the last 2 million years (Martin, 1983).

In addition to shaping the unusual mechanism of birth, however, the “tug-of-war” exerted on maternal pelvic dimensions by bipedalism and encephalization is widely assumed to have increased the risks of delivery. Krogman (1951) described human birth as a “scar” of our evolutionary history, while Washburn (1960) coined the term “obstetric dilemma,” which has been widely used subsequently.

The obstetric dilemma is widely considered a key ultimate factor underlying high levels of maternal and neonatal mortality in contemporary populations, though various other proximate factors are also relevant. One might, however, question why natural selection should favor the persistence of an obstetric dilemma with such apparent adverse effects on either mother or offspring, or indeed both. For the mother, death during childbirth is clearly terminal for fitness, and stillborn offspring and perinatal mortality are significant fitness penalties. For the offspring, poor growth is similarly detrimental, with low birth weight the single strongest predictor of mortality in early life (Kramer, 1987), the period of the life-course when mortality risk is greatest. Although Trivers’ parent-offspring conflict theory (Trivers, 1974) may apply to many aspects of human growth and development (Haig, 1993; Wells, 2003; Crespi, 2011), this is one scenario where the two parties must surely favor a compromise that substantially constrains the penalties on each party. Rather than comprising a long-standing game of “Russian roulette,” where either mother or offspring

\*Correspondence to: Jonathan C.K. Wells, Childhood Nutrition Research Centre, UCL Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK. E-mail: Jonathan.Wells@ucl.ac.uk

Received 6 September 2012; accepted 7 September 2012

DOI 10.1002/ajpa.22160

Published online 8 November 2012 in Wiley Online Library (wileyonlinelibrary.com).

might receive the “fatal bullet” arising from the risks of obstructed labor, it seems logical that the “bipedalism-encephalization conflict” hypothesis is either insufficient as an explanation of perinatal mortality, or overstated.

Over time, many different versions of the obstetric dilemma have been emphasized by different authors. Two decades ago, Rosenberg argued that there was no single dilemma, and that instead, different aspects of the biology of human birth evolved in a complex mosaic way over time (Rosenberg, 1992). The evolution of each of encephalization and bipedal locomotion is best considered as a lengthy process, which in turn interacted with diverse ecological stresses including energy availability and the thermal environment. Subsequent work has considered how the obstetric dilemma differs between hominin species (Weaver and Hublin, 2009; Ruff, 2010) and among contemporary human populations (Ruff, 1994; Weaver and Hublin, 2009; Kurki, 2007, 2011).

In this review, we build on extensive work on the unusual human birth mechanism by others, to consider how the obstetric dilemma is further shaped by phenotypic plasticity, potentially leading to time periods during which the difficulty of birth increases or decreases. The prevalence of caesarian section may vary with trends in diet, exercise, and stature (Liston, 2003), casting doubt on the extent to which the contemporary magnitude of the obstetric dilemma is a useful model for its manifestation in our evolutionary history. However, it also should be noted that caesarian rates may not provide an objective index of the magnitude of the obstetric dilemma in contemporary populations. For example, unusually high rates of cesarean sections in a few countries such as Brazil seemingly indicate cultural preference, although recent work has demonstrated that the main factor underlying such high rates comprises clinician/hospital preference, especially in private patients, as opposed to maternal preference for this form of delivery (Potter et al., 2001; Barbosa et al., 2003; McCallum, 2005).

### EVIDENCE FOR THE PERILS OF HUMAN CHILDBIRTH

The inherent riskiness of human delivery is supported by worldwide data on maternal and neonatal mortality, defined respectively as maternal death during pregnancy, childbirth, or the subsequent 42 days (Hogan et al., 2010) and neonatal death within 28 days of birth (World Health Organization, 2006).

Maternal mortality has been substantially reduced by medical progress in recent decades, and the estimated annual global total fell from ~530,000 in 1980 to ~340,000 in 2008 (Hogan et al., 2010). Nevertheless, this decline indicates the importance of medical and public health expertise in addressing a critical health issue. The majority of maternal deaths occur in nonindustrialized settings, with >50% of the total in just six countries—Afghanistan, India and Pakistan in Asia, and Ethiopia, the Democratic Republic of Congo and Nigeria in Africa (Hogan et al., 2010), though these levels are related to high fertility rates (Diamond-Smith and Potts, 2011). A high proportion of maternal mortality occurs in the immediate postpartum period (Li et al., 1996), and obstructed labor is one of the four most common causes, amounting to ~12% of maternal mortality worldwide (World Health Organization, 2005). For example, obstructed labor is the leading cause of maternal mortality in Afghanistan (AbouZahr, 1998).

For the offspring, annual mortality worldwide currently comprises ~3.3 million stillbirths (indicating fetal death), of which one third are attributable to the delivery process itself, and ~4 million neonatal deaths, of which ~3 million occur in the first week of life and hence are classified as perinatal deaths (World Health Organization, 2006). The annual total of perinatal deaths around the time of birth is thus ~6.3 million. Again, 98% of the deaths occur in nonindustrialized settings, highlighting the role of medical facilities and public health programs in reducing this source of mortality. In nonindustrialized settings, only ~50% of births occur with the help of a doctor, midwife, or qualified nurse (World Health Organization, 2006), although a variety of other individuals (often referred to as “traditional birth attendants”) assist at births in such societies (Leedam, 1985; Lefebvre and Voorhoever, 1997), and the norm of delivering alone is extremely rare (though see Sargent, 1982, 1989).

The recent historical record offers little evidence that such high mortality rates are a new phenomenon. Most indices demonstrate falling rates of both maternal and neonatal mortality through the last century, suggesting a more serious problem in the recent past. The mortality rate within the first year of life among 22 foraging populations averaged 20.3% (Kelly, 1995), although few data are available for either maternal or perinatal mortality in such populations.

Looking further back into the past, the scenario is much less clear, as only indirect evidence is available. Data from historical cemeteries or recent prehistoric populations show a typical younger age at death of women compared with men, with reproduction potentially contributing to this sex-difference. In a sample of populations ranging from the Neolithic to the middle ages, women died on average 5 years earlier than men (Wells, 1975), while a similar conclusion was reached based on the analysis of 24,848 epitaphs from the Roman era (MacDonell, 1913). More detailed investigations, however, cast strong doubt on the notion that these sex differences arise directly from increased female reproductive mortality, as discussed below.

Archaeological excavations have periodically revealed evidence of fetuses within the maternal pelvis, often referred to as “obstetric death.” Examples have been recorded from ancient Egypt (Elliot-Smith and Wood Jones, 1910), ancient Greece (Liston and Papadopoulos, 2004), Neolithic Vietnam (Willis and Oxenham, 2011), 1300 BC to 1400 AD Chile (Arriaza et al., 1988), prehistoric and medieval Britain (Hawkes and Wells, 1975; Roberts and Cox, 2003), medieval Denmark (Moller-Christensen, 1958) and 16th to 19th century Amerindian populations in the US (Owsley and Bradtmiller, 1983). A recent study also identified pelvic stress injuries in a small-bodied female forager from Southern Africa (Pfeiffer, 2011), with obstetrical stress a plausible explanation. In Britain, the archaeological record includes 5 possible cases of “obstetric death” from the Iron Age, 6 from the early medieval period, and 10 from the late medieval period (Roberts and Cox, 2003). While some of these examples may be reliable evidence of obstetric complications, with the fetus clearly situated in the pelvis, many merely comprise co-burial of an adult female and fetus and infant, offering no evidence that the individuals were related, or that death occurred through difficulties during delivery (Wells, 1975; Roberts and Cox, 2003). Infectious diseases are an alternative likely cause of such deaths; for example puerperal fever was a major

factor in both maternal and neonatal deaths during the 18th to 19th centuries (Wells, 1975).

Although fetal/infant skeletal material preserves less well than that of adults, the existing archaeological evidence for “obstetric deaths” has been proposed by several authors to indicate a surprisingly “empty” record of problematic childbirth (Wells, 1975; Pfeiffer, 2011; Willis and Oxenham, 2011). The only evidence of “obstetric death” being relatively common is from ancient Andean populations of the Azapa valley in northern Chile, where analysis of mummified females suggested around 25% of females may have died from childbirth-complicated deaths in the early agricultural period, but a much smaller proportion (~6%) from AD 700 to 1400 (Arriaza et al., 1988). Average adult female height of this population was ~149 cm (Pomeroy and Stock, 2012), and this relatively low stature may be an important factor contributing to the high prevalence of obstetric death, as discussed in detail in the second half of this review.

In general, however, the tendency for higher rates of female relative to male mortality in the recent archaeological record appears to be linked with poorer bone health in general, and to indicate greater female exposure to malnutrition (Wells, 1975; Willis and Oxenham, 2011), and hence potentially to infectious disease, rather than a major specific impact of childbirth complications. Consistent with that conclusion, excess female mortality in these datasets continued after the likely cessation of the ability to give birth, at around 40 years (Wells, 1975). However, this scenario has not necessarily persisted into more recent times: as the burden of infectious disease has declined, the sex difference in mortality risk has both reversed and increased in association with economic development (Kruger and Nesse, 2006). Rickets, a vitamin deficiency disease common in urban populations and detrimental to pelvic development, has been considered the most common cause of obstructed labor from the 17th to early 20th centuries, but this condition is presumed to have been very rare before the second millennium AD (Wells, 1975), and hence not to have been a major evolutionary stress.

This brief review of evidence for maternal and perinatal mortality has aimed simply to raise the issue that contemporary data on maternal and perinatal mortality arising from the challenges of childbirth need not necessarily provide a reliable estimate of longer-term obstetric complications throughout human evolutionary history. The puzzle of why the obstetric dilemma might be both universal in quality and yet variable in quantity needs reappraisal, from the perspectives of primatology, the hominin fossil record, and human anthropometric history.

## MATERNAL Pelves AND OFFSPRING SIZE

There is ample evidence across many mammalian species that the female pelvis has relatively larger internal dimensions than that of the male, indicating the fundamental role of pregnancy and birth in shaping female skeletal dimensions. Such dimorphism has been reported for example for rodents (Dunmire, 1955; Berdnikovs et al., 2007), bats (Crelin and Newton, 1969; Chapman et al., 1994), grey fox (Schutz et al., 2009b), sheep (Cloete et al., 1998), and primates (Schultz, 1949; Leutenegger, 1974; Steudel, 1981) including humans (LaVelle, 1995; Tague, 2000; Kurki, 2007). Among primates, the size of the maternal pelvis is associated with neonatal

brain size (Schultz, 1949; Leutenegger, 1974; Rosenberg, 1992), a conclusion that holds up when the analysis is controlled for phylogenetic relationships and body size (Ridley, 1995). However, the fit between pelvic and neonatal dimensions is by no means perfect in many species. In free-tailed bats, the disparity between the traits is so great that the interpubic ligament must stretch to ~15 times its original length during birth to allow delivery of the offspring (Crelin, 1969).

The obvious role of reproduction in shaping female pelvic dimensions might suggest that the male pelvis, lacking exposure to such selective pressures, might be predominantly shaped by locomotor mechanics. However, some pelvic dimorphism might arise through allometric effects of body size (Tague, 2000), or through more general secondary sexual differentiation (Leutenegger, 1974; Tague, 2005). Other analyses indicate a powerful effect of the thermal environment on variability in torso dimensions, and specifically the pelvis (Ruff, 1994; Weaver, 2003). Such studies indicate that a number of different selective pressures affect the pelvis of each sex, and that reproduction is not the only stress contributing to pelvic dimorphism. For example, pelvic structure is also associated with body posture (Reynolds and Hooton, 1936; Abitbol, 1987), and may therefore respond to the greater mass of the upper body in males, a secondary sexual signal.

Furthermore, such pelvic variability need not only reflect genetic adaptation, as increasing evidence indicates that diverse environmental factors such as dietary quality and sunlight (Dick, 1922; Merewood et al., 2009), maturational status (Brabin and Brabin, 1992), and parity (Cox and Scott, 1992; Schutz et al., 2009a) can also affect pelvic phenotype, potentially giving rise to variability within and across populations in the magnitude of the obstetric dilemma.

Before addressing this variability in detail, we first reconsider how the generic obstetric dilemma characteristic of our species arose, through a review of the literature on the hominin fossil record, and maternal and neonatal data from primates.

## EVOLUTIONARY HISTORY OF THE HOMININ OBSTETRIC DILEMMA

The challenges of delivery in humans have attracted extensive interest from anthropologists, and a number of features where humans appear to contrast with other primates have been identified. In comprehensive reviews, Trevathan (1988) and Rosenberg (1992) observed that humans have an unusually lengthy typical duration of labor; a tendency for other individuals to assist mothers during delivery, due to the unusual position of emergence of the offspring relative to the mother; and an unusual rotational mechanism of birth, as summarized below.

While fetal head size and maternal pelvic size appear to be key determinants of the potential for obstetric complications, the shape of the pelvic inlet and outlet is a central determinant of interspecific variation in the birth process. The pelvic inlet of nonhuman primates is generally elongated in the anteroposterior dimension, while narrower mediolaterally. The human pelvis is unique amongst primates, as a result of the evolution of bipedalism, in being very broad transversely, relatively short in the cranial-caudal dimension, and shallower anteroposteriorly (Lovejoy, 2005). These morphological characteristics result in a differently shaped inlet of the obstetric

canal, which is relatively shallow anteroposteriorly, and broader in the mediolateral dimension. The relationship between breadth and depth changes, however, between the pelvic inlet which is more broad and shallow, and the midplane and outlet in which there is greater depth and less breadth.

Whereas the birth canal is relatively straight in nonhuman primates, it manifests in humans as a cylinder of varying diameters and angles, as the inlet, midplane, and outlet have different cross-sectional shapes and areas (Rosenberg, 1992; Abitbol 1996). A number of movements of head and torso of the fetus are generally required in order to pass through these three sections (Rosenberg, 1992). Along with shifts in alignment of the body, plasticity in the fetal cranium is another important means of accommodating the tight fit. The open sutures and pliability of the neonatal head cause both the length and biparietal breadth to reduce during vaginal delivery (Sorbe and Dahlgren, 1983; Pu et al., 2011). Recent work has shown that this pliability of the fetal skull was already present in *Australopithecus* (Falk et al., 2012).

The limited available evidence suggests that the dimensions of the obstetric pelvis increase only marginally during labor through loosening of the ligaments maintaining the rigidity of the pubic symphysis and sacroiliac joints (Bjorklund et al., 1997), and although the hormone relaxin is implicated in this mechanism, the magnitude of the effect appears modest likewise (Johnson et al., 1994; Goldsmith and Weiss, 2009). The main implication of these studies for the obstetric dilemma is that the neonatal head appears substantially more pliable during delivery than the maternal pelvis.

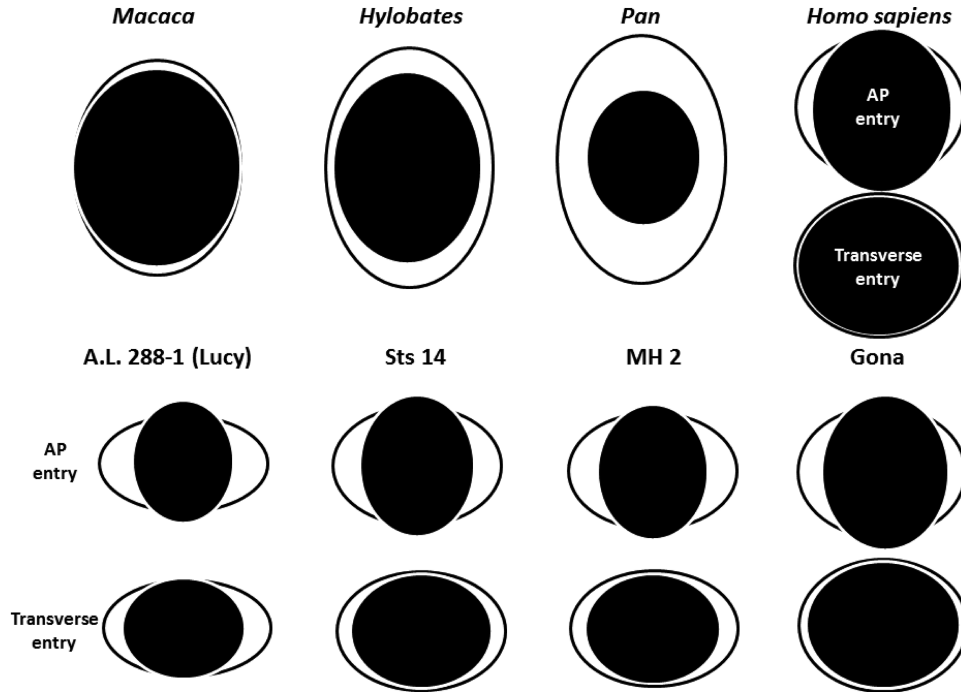
During delivery, the greater length of the neonatal cranium in the sagittal dimension, and the changing dimensions of the birth canal from superior to inferior, tends to induce the human head to enter the pelvic inlet in the oblique or transverse plane (facing to the side rather than anterior or posterior as in nonhuman primates), and rotate to pass through the pelvic midplane and outlet in the sagittal plane (facing toward the posterior, rather than the anterior side as is typical of nonhuman primates). A further rotation is required to allow the shoulders to pass through the pelvic outlet (Rosenberg, 1992; Trevathan and Rosenberg, 2000). This "rotational" birth has long been considered to be a unique characteristic of humans, reflecting the competing constraints of encephalized offspring and bipedal biomechanics (though see below). Though modern human birth can occasionally happen without these rotations, from a clinical perspective, this rotational birth mechanism is both considered the norm, and associated with lower risk than other mechanisms (Norwitz et al., 2003; though see Walrath, 2003). Importantly, the size of the neonatal head relative to pelvic dimensions is not the only factor in predicting obstetric challenges (Ferguson et al., 1998), though it still remains an important variable, and other body proportions are relevant as discussed below.

To put the human birth process in context, it is informative to consider evidence for the birth process among earlier hominins. It has been suggested that rotational birth mechanisms may have been selected not only to accommodate large fetal craniums, but also relatively large and inflexible shoulders, which are associated with generalized ape adaptations for suspensory or brachiating locomotion (Rosenberg and Trevathan, 2002). These authors have therefore hypothesized that rotational birth may have preceded the evolution of highly encephalized

infants in the genus *Homo*, due to the interaction of bipedal pelvis and large fetal shoulders. The absence of extreme encephalization in *Australopithecus* newborns would further suggest a different rotational mechanism from that in *Homo*. Of particular interest here are several reports of 'occiput anterior' births amongst captive chimpanzees, suggesting that it may be relatively common for chimpanzee births to have a rotational pattern (Elder and Yerkes, 1936; Hirata et al., 2011). Importantly, it is the broad shoulders of these apes that may be responsible for this rotation, given that the transverse dimension of the midplane in the female chimpanzee pelvis reduces to a mere 8.3 cm (Tague and Lovejoy, 1986), while the sagittal dimensions remain large throughout. These observations of rotational birth in chimpanzees complement earlier evidence for breech birth (Rushton and McGrew, 1980), by increasing the range of birth variation observed amongst chimpanzees, and suggesting that narrow characterization of birthing patterns among species may be difficult. A similar point has been made for humans (Walrath, 2003).

The australopithecine pelvis is perhaps best understood from A.L. 288-1, (Lucy), a 3.18 million-year-old *Australopithecus afarensis* female. The dominant reconstruction of this pelvis indicated a highly derived morphology that is very broad mediolaterally, and shallow anteroposteriorly throughout the entirety of the birth canal, suggesting an unusual pattern of transverse orientation of the head throughout the birthing process (Tague and Lovejoy, 1986). While reconstructions of the Sts 14 *Australopithecus africanus* pelvis also indicate similar broad dimensions (Abitbol, 1995; Häusler and Schmid, 1995; Berge and Goullaras, 2010), other reconstructions of AL-288-1 have suggested greater anteroposterior dimensions of this specimen (Berge et al., 1984; Häusler and Schmid, 1995). A recent reconstruction of Sts 14 suggests a human-like pattern of rotational birth in these early *Australopithecus* females (Berge and Goullaras, 2010). These authors posit that asymmetrical contractions would flex and rotate the fetal skull through the birth canal, ultimately resulting in an "occiput anterior" birthing position (Berge and Goullaras, 2010). The recent discovery of skeletal material attributed to *Australopithecus sediba* (MH 2) complicates this matter. The pelvis has features associated with both *Australopithecus* and *Homo*; however, the obstetric dimensions are still small, suggesting that pelvic dimensions were not driven by fetal encephalization alone, and that some aspects of pelvic morphology attributed to *Homo* vary independently of obstetric dimensions (Kibii et al., 2011). Simulations based on ape allometry suggest that a high ratio of neonatal mass to maternal mass may have evolved early in the hominin lineage, remaining relatively consistent from australopithecines through the genus *Homo* to modern humans, suggesting that large neonatal bodies may also contribute to the obstetric dilemma in these early hominins (DeSilva, 2011).

It was initially argued that *Homo erectus* (*sensu lato*) was characterized by mediolaterally broad pelvis, but that this was accompanied by relatively shallow anteroposterior dimensions, and as such may not have required rotational mechanisms of birth (Ruff, 1995). The transverse breadth of australopithecine and early *Homo* pelvis is often interpreted as evidence for adaptations to bipedal locomotion, increasing the lever arm for the lesser gluteals during the "single-legged stance" phase of walking. The discovery of an early Pleistocene female



**Fig. 1.** Redrawn from Rosenberg and Trevathan (1996); based on Schultz (1949). Relative cranial dimensions in infant primates (filled ovals) are superimposed on pelvic openings (outer oval), with the offspring head in anterior–posterior orientation (upper row) and transverse orientation (lower row). All pelvises are scaled so that the mediolateral dimensions are equal. Notice the anteroposteriorly deep birth canal in chimpanzees (*Pan*), allowing for relatively easy passage of the neonatal head. Broad ape shoulders may require some rotation as has been observed recently (Hirata et al., 2011). Monkeys, lesser apes (*Hylobates*) and humans present more of an “obstetric dilemma” with the neonatal head close to, or even exceeding, the dimensions of the birth canal. In the bottom row are four hominid fossils illustrating the relative difficulty of birth in *Australopithecus* and early *Homo*. Modeled here are the inlet dimensions of the birth canal. As in humans, the maximum dimension of the pelvic inlet in early hominids is oriented mediolaterally, indicating that the neonatal cranial entered the pelvic inlet obliquely or transversely during birth. Based on estimates of cranial dimensions and minimum dimensions of the birth canal, birth was particularly difficult in the earliest australopithecines represented here by Lucy and Sts 14.

pelvis dated to between 1.4 and 0.9 mya in the Gona region of Ethiopia has been cited as evidence for very large pelvic size in early *Homo erectus*, which included an anteroposterior expansion of the pelvic canal, allowing for greater cranial size of offspring (Simpson et al., 2008). Though the Gona pelvis possesses a relatively spacious birth canal, it is transversely wide throughout, as is found in *Australopithecus*. It is unclear whether this morphology would result in a human-like rotational birth as suggested for some australopithecines (Berge and Goularas, 2010), a semirobational birth driven primarily by shoulder anatomy (Trevathan and Rosenberg, 2000), or a transverse, nonrotational delivery as Tague and Lovejoy (1986) suggested for Lucy. Additionally, the taxonomic attribution of this specimen has been questioned (Ruff, 2010).

The paucity of female pelvises and complete absence of fossil neonates of early hominids prevent us from making any definitive statements about the birth process in our ancestors. However, by using estimates of brain size at birth in fossil taxa (from DeSilva and Lesnik, 2008) and published dimensions of the available fossil pelvises, some interesting patterns emerge. Following the classic diagram produced by Schultz (1949) and redrawn by Rosenberg and Trevathan (1996, 2002), we added the fossils Lucy, Sts 14, MH 2, and Gona (Fig. 1). First, it is clear that the neonate must have entered the pelvic inlet in a transverse or oblique orientation since the anteroposteriorly narrowed hominid pelvic inlet is too small to

accommodate the sagittal dimensions of a fetal skull. Relative to the minimum pelvic dimension, the estimated head size in early *Australopithecus* (Lucy, Sts 14, and MH 2) and in early *Homo* (Gona) appears to present a significant obstetric dilemma. A relatively large birth weight (DeSilva, 2011), and large shoulders (Trevathan and Rosenberg, 2000) would have further complicated birth, perhaps necessitating some form of rotation in some australopithecine species (Berge and Goularas, 2010).

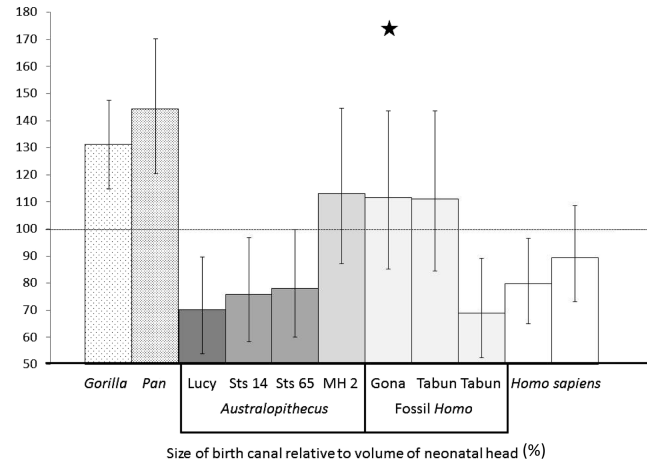
A skeleton from Jinniushan, northeastern China, dated to 260KYA, provides rare fossil evidence of female hominid pelvic morphology from the Middle Pleistocene (Rosenberg et al., 2006). This unusually large-bodied female has an extremely broad bi-iliac breadth, but her pelvic inlet shape is within the range of modern humans (Rosenberg et al., 2006). While the lack of a preserved sacrum prevents direct measurement of obstetric dimensions, estimates of an extremely broad pelvis seem reasonable, and this interpretation also suggests a transversely broad pelvic canal.

Recent reconstructions of the Tabun 1 pelvis have shed the first light on the morphology of the female Neanderthal pelvis (Ponce de León et al., 2008; Weaver and Hublin 2009). A model of Neanderthal brain growth suggests similar cranial sizes to modern humans at birth, and similar obstetric demands on pelvic morphology (Ponce de León et al., 2008). While this analysis was primarily concerned with understanding cranial ontogeny

in Neanderthals, it importantly included a virtual reconstruction of the female pelvis. The reconstruction suggests that the mediolateral dimensions of the Neanderthal pelvic inlet and midplane are considerably greater than among modern females, with mediolateral orientation of the sagittal plane at the beginning of parturition, but with larger anteroposterior dimensions in the pelvic outlet necessitating rotation of the neonatal cranium to the “occiput anterior” position. This could suggest Neanderthals had the same pattern of rotational birth as modern humans. A subsequent reconstruction of the same pelvis by Weaver and Hublin (2009) suggests mediolaterally broad dimensions of both the pelvic inlet and outlet, and a primitive nonrotational/transverse orientation of the neonate cranium throughout the birthing process. Despite this interpretation, the authors concluded that due to obstetric and cranial dimensions, Neanderthals would have experienced difficult births, though whether these approximated the modern human scenario is unknown. A further female skeleton from Palomas, Spain (Walker et al., 2011), and a partial female pelvis from Sima de los Huesos (Bonmati et al., 2010) may shed further light on the obstetric phenotype of Neanderthals.

The evolutionary history of the obstetric dilemma as currently understood in the fossil record is presented in Figure 2. Neonatal cranial dimensions that could pass through the minimum pelvic dimensions of African apes, fossil hominins, and modern humans were calculated using the methods presented in Simpson et al. (2008). The values presented in Figure 2 are the ratios of the brain size at birth (from DeSilva and Lesnik, 2008) relative to these minimum pelvic dimensions. Values above 100 indicate that the neonatal head is smaller than the minimum pelvic opening, while values below 100 indicate that the neonatal head exceeds the pelvic dimensions and would require pliability of the fetal skull and, possibly, accompanying relaxation of the pelvic ligaments to allow passage of the neonate. Relative to African apes, early *Australopithecus* (Lucy, Sts 14, and Sts 65) had particularly high cephalo-pelvic disproportion. However, this apparent obstetric dilemma had eased by the early Pleistocene as seen in the nearly identical values for the MH 2 pelvis and the Gona specimen. These values in Figure 2 differ slightly from those illustrated in Figure 1 because only the inlet dimensions are shown in that latter figure. Though the inlet (anteroposterior) is the narrowest part of the birth canal in both MH2 and the Gona pelvis, the narrowest dimensions in the Lucy and Sts 14 reconstructions are in the pelvic outlet and midplane respectively, exacerbating the cephalo-pelvic disproportion illustrated in Figure 1. The reconstructions of Tabun 1 are inconsistent, one suggesting a dilemma similar to that of MH2 and Gona (Ponce de León et al., 2008) and the other a much more severe dilemma (Weaver and Hublin, 2009), illustrating the need for a more complete female Neanderthal pelvis. Modern humans (data from Tague and Lovejoy, 1998; Berge and Goularas, 2010) are more *Australopithecus*-like in the apparent fit between offspring head and maternal pelvic dimensions, though this need not imply similar difficulty or mechanism of delivery.

Overall, there is contradictory evidence of the evolution of characteristics of the modern pelvis in the fossil record. It is clear that very broad mediolateral internal and external dimensions of the pelvis were an early adaptation to bipedal locomotion found among australopithecines, while there is evidence for later anteroposterior elongation of the pelvic canal within the genus *Homo*,



**Fig. 2.** Neonatal cranial capacities (CC) accommodated by minimum pelvic dimensions in female hominoids were compared with neonatal brain size at birth in *Australopithecus*, extinct *Homo*, and modern apes and humans. Pelvic dimensions were converted to neonatal CC following Simpson et al. (2008), with minimum values obtained by assuming that (1) the biparietal dimensions of the neonatal head were 8% smaller than the minimum pelvic dimension, and (2) the cranial height is 60% the biparietal breadth. Maximum values were obtained by assuming that (1) the biparietal dimensions were 5% smaller than the minimum pelvic dimension, and (2) the cranial height is 70% the biparietal breadth. These ranges were compared with the average and 95% confidence intervals for neonatal CC calculated for extinct hominins and modern hominoid values from DeSilva and Lesnik (2008). A.L. 288-1 is “Lucy,” a female *Australopithecus afarensis* [values from Tague and Lovejoy (1996)]. Sts 14 and Sts 65 are female *Australopithecus africanus*. Values for Sts 14 from Berge et al. (2010) and Sts 65 (personal observation). MH2 is a female *Australopithecus sediba* pelvis—values from Kibii et al. (2011). Gona is a female *H. erectus* pelvis [values from Simpson et al. (2008)]. The star indicates the position of the average pelvic dimension to neonatal CC value if this pelvis is from *Paranthropus boisei* (Ruff et al., 2010). Tabun is a Neanderthal female—values are (left to right) from the minimum values reported by Ponce de León et al. (2008) and Weaver and Hublin (2009). Unfortunately, these values are different enough that little can be said about the obstetric dilemma in late Pleistocene humans. Human values are from Berge et al. (2010) on left and Tague and Lovejoy (1998) right, and African ape values from Tague and Lovejoy (1998).

and differing interpretations of Neanderthal pelvic morphology. The long-held notion that anatomically modern humans are the only species offering unambiguous evidence for rotational birth is challenged by the recent chimpanzee observations, though rotational mechanisms are likely to differ between species. We suggest that while long-term trends in encephalization and bipedalism have undoubtedly shaped the obstetric dilemma over hominin evolution, the relative importance of these two traits for variability in the magnitude of the dilemma within the genus *Homo* remains less certain. One possibility is of a recurring pattern, whereby the obstetric dilemma first intensified, then was relieved by evolution of a rotational birth mechanism, and then reintensified in response to new ecological stresses.

Unfortunately, the near absence of data on the shape of the birth canal in late Pleistocene *Homo* hinders our ability to characterize birth in our immediate ancestors. It should not go unnoticed that we probably know more about birth in australopithecines than in our mid-late

Pleistocene ancestors. In the absence of more concrete data on the obstetric dilemma of hominins, re-evaluation of the primate evidence may therefore be informative.

**NEONATAL BRAIN SIZE: THE PRIMATE EVIDENCE**

The use of the word “encephalization” is increasingly critiqued, in recognition that it is neuron number rather than sheer size of the brain that determines relative cognitive capacity (Herculano-Houzel, 2011). The estimation of encephalization by assessing brain mass relative to body mass is also considered problematic, with some arguing that absolute brain size is more relevant to cognitive capacity (Marino, 2006). In the following section, the word encephalization is used to refer simply to the physical association between brain mass and body mass, i.e. the brain: body ratio, without intending to indicate functional or cognitive correlates of this property. This allows discussion of the relative physical contributions of brain and other body components to the obstetric dilemma.

The fact that humans are extremely encephalized relative to other primates in adult life has led to the perception that encephalization is the key challenge for human delivery posed by the infant. Leutenegger challenged this view, however, by considering relationships between maternal and neonatal brain and body size across primates (Leutenegger, 1982). This approach now merits reappraisal, due to the increased amount of data available. Data on brain mass, total body mass, and duration of gestation were obtained for neonates, and for either the mother (body mass) or for adults averaged across the two sexes (brain mass), in a sample of up to 92 primate species including humans (Harvey et al., 1987; Smith and Jungers, 1997; Smith and Leight, 1998; Begun and Kordos, 2004; Drews et al., 2011) and unpublished data from Yerkes, Southwest, and Oregon National Primate Research Centers. For regression analysis, data were natural log-transformed, and associations tested using reduced-major axis regression analysis (Smith, 2009). Full details of the regressions are given in Table 1. Regression residuals were calculated in sympercent format as  $\text{Ln}(O/E) \times 100$  (Cole, 2000a), where  $O$  is the observed value for humans and  $E$  is the value predicted from the relevant regression equation.

These data are first analyzed to provide an interprimate perspective, and then repeated for catarrhines only. This two-stage approach allows appreciation of the extent to which catarrhines as a group show adaptations between maternal and primate dimensions, and illustrates the extent to which humans fit or contradict these trends among more closely related species.

Plotting Ln neonatal mass against Ln adult female mass, Leutenegger noted first that the value of the regression slope was below 1, and second, that humans have a highly positive regression residual (Leutenegger, 1982). Using an expanded dataset, these conclusions remain supported. The updated primate (Fig. 3a) and catarrhine regression equations respectively are:

$$\text{Ln Neonate mass} = 4.329 + (0.913 \times \text{Ln Adult female mass}) \tag{1a}$$

$$\text{Ln Neonate mass} = 4.918 + (0.691 \times \text{Ln Adult female mass}) \tag{1b}$$

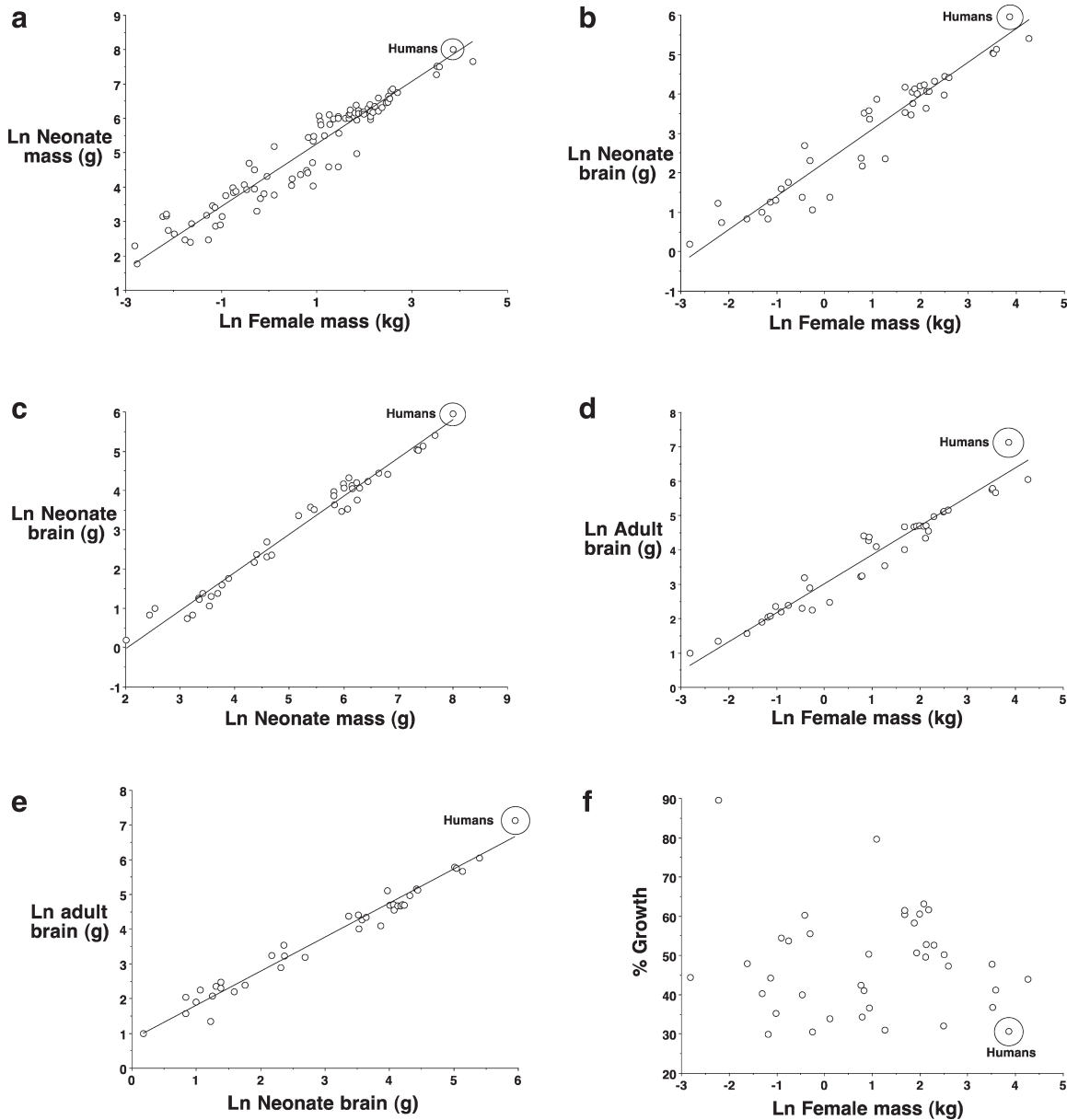
The negative slopes imply that smaller primates produce relatively larger neonates in proportion to adult female mass. The regression residual for humans indi-

TABLE 1. Reduced-major axis regression statistics for associations between maternal and offspring traits relevant to the obstetric dilemma in primates

| Outcome <sup>a</sup> | Predictor <sup>a</sup> | Sample       | n  | k <sub>1</sub> | SE    | 95% CI           | k <sub>2</sub> | SE    | 95% CI         | Residual (%) <sup>b</sup> | r <sup>2</sup> |
|----------------------|------------------------|--------------|----|----------------|-------|------------------|----------------|-------|----------------|---------------------------|----------------|
| Neonate mass         | Adult female mass      | All primates | 92 | 4.329          | 0.058 | 4.220 to 4.435   | 0.913          | 0.026 | 0.865 to 0.970 | 15.0                      | 0.917          |
| Litter mass          | Adult female mass      | All primates | 85 | 4.530          | 0.065 | 4.393 to 4.643   | 0.835          | 0.028 | 0.789 to 0.897 | 25.0                      | 0.906          |
| Neonate mass         | Adult female mass      | Catarrhines  | 43 | 4.918          | 0.107 | 4.711 to 5.097   | 0.691          | 0.051 | 0.595 to 0.778 | 41.8                      | 0.892          |
| Neonate brain mass   | Adult female mass      | All primates | 43 | 2.238          | 0.09  | 2.058 to 2.401   | 0.851          | 0.037 | 0.786 to 0.931 | 42.2                      | 0.915          |
| Litter brain mass    | Adult female mass      | All primates | 43 | 2.384          | 0.091 | 2.185 to 2.529   | 0.799          | 0.038 | 0.738 to 0.887 | 47.7                      | 0.910          |
| Neonate brain mass   | Adult female mass      | Catarrhines  | 19 | 2.512          | 0.305 | 1.975 to 2.991   | 0.753          | 0.122 | 0.573 to 0.964 | 52.7                      | 0.793          |
| Neonate brain mass   | Neonate mass           | All primates | 42 | -1.979         | 0.151 | -1.708 to -2.264 | 0.972          | 0.026 | 0.927 to 1.025 | 14.5                      | 0.976          |
| Neonate brain mass   | Neonate mass           | Catarrhines  | 18 | -1.826         | 0.531 | -0.740 to -2.593 | 0.949          | 0.078 | 0.787 to 1.054 | 17.7                      | 0.909          |
| Adult brain mass     | Adult female mass      | All primates | 39 | 2.998          | 0.061 | 2.868 to 3.102   | 0.844          | 0.036 | 0.792 to 0.915 | 87.2                      | 0.947          |
| Adult brain mass     | Adult female mass      | Catarrhines  | 16 | 2.843          | 0.414 | 2.071 to 3.376   | 0.905          | 0.181 | 0.683 to 1.216 | 79.1                      | 0.809          |
| Neonate mass         | Gestation length       | All primates | 35 | -1.541         | 0.806 | -0.241 to -3.227 | 0.039          | 0.004 | 0.033 to 0.049 | -78.8                     | 0.593          |
| Neonate mass         | Gestation length       | Catarrhines  | 16 | 2.842          | 0.38  | 1.946 to 3.471   | 0.019          | 0.002 | 0.016 to 0.024 | 12.9                      | 0.791          |
| Adult brain mass     | Neonate brain mass     | All primates | 39 | 0.815          | 0.114 | 0.586 to 1.020   | 0.983          | 0.032 | 0.922 to 1.040 | 46.8                      | 0.970          |
| Adult brain mass     | Neonate brain mass     | Catarrhines  | 16 | -0.495         | 0.405 | -1.111 to 0.388  | 1.267          | 0.850 | 1.075 to 1.389 | 8.9                       | 0.946          |

All equations in the format: outcome = k<sub>1</sub> + (k<sub>2</sub> × predictor).<sup>a</sup> Both outcome and predictor natural log-transformed except for gestation length as predictor.

<sup>b</sup> Residual for humans expressed as percentage of expected value.



**Fig. 3.** Associations between maternal and neonatal phenotype in 43 primate species. (a) Neonatal mass and adult female mass; (b) Neonatal brain mass and adult female mass; (c) Neonatal brain mass and neonatal mass; (d) sex-averaged adult brain mass and adult female mass; (e) sex-averaged brain mass and neonatal brain mass; (f) percentage of sex-averaged adult brain size achieved at birth.

ates that human offspring are 15.0% larger than expected for their adult female mass relative to primates in general (or 25.0% if litter mass is predicted), but 41.8% larger relative to other catarrhines.

Plotting Ln neonatal brain mass against Ln adult female mass, a similar scenario was observed by Leutenegger (1982). The updated primate (Fig. 3b) and catarrhine regression equations respectively are:

$$\text{Ln Neonatal brain mass} = 2.238 + (0.851 \times \text{Ln Adult female mass}) \quad (2a)$$

$$\text{Ln Neonatal brain mass} = 2.512 + (0.753 \times \text{Ln Adult female mass}) \quad (2b)$$

Again, smaller primates produce offspring with relatively larger brains in proportion to adult female mass,

but humans contradict this trend and produce neonates with brain mass 42.2% larger than expected for their maternal size (47.7% if litter brain mass is the outcome). Considering only catarrhines, the human contrast is slightly greater, at 52.7%.

However, plotting Ln neonatal brain mass against Ln neonatal body mass, Leutenegger (1982) reported a slope of 0.94, and found that humans lay exactly on the regression line. He therefore concluded that neonatal brain mass scaled broadly isometrically with neonatal body mass across primates in general, and that humans did not deviate from the trend. Using more recent data, Figure 3c illustrates that his conclusion regarding isometrical scaling remains supported for primates in general, with the updated regression equation being:



$$\begin{aligned} \text{Ln Neonatal brain mass} &= -1.979 \\ &+ (0.972 \times \text{Ln neonatal mass}) \quad (3a) \end{aligned}$$

However, the human regression residual indicates a 14.5% greater brain mass than expected for neonatal body mass, showing a modest degree of neonatal encephalization compared with other primates. This discrepancy is greater if only catarrhines are considered:

$$\begin{aligned} \text{Ln Neonatal brain mass} &= -1.826 \\ &+ (0.949 \times \text{Ln neonatal mass}) \quad (3b) \end{aligned}$$

Using this equation, humans have 17.7% greater brain mass than expected for neonatal body mass. This degree of encephalization is still substantially less than the magnitude of encephalization in adulthood. Figure 3d plots adult sex-average brain size against maternal weight, where the primate regression equation is:

$$\begin{aligned} \text{Ln adult brain mass} &= 2.998 \\ &+ (0.844 \times \text{Ln adult female mass}) \quad (4a) \end{aligned}$$

Again, the negative slope indicates that in relative terms, smaller adult primates have larger brains; however, humans strongly contradict this trend and have 87.2% greater adult brain mass than expected for their adult female mass. Similar findings emerge if the regression is restricted to catarrhines:

$$\begin{aligned} \text{Ln adult brain mass} &= 2.843 \\ &+ (0.905 \times \text{Ln adult female mass}) \quad (4b) \end{aligned}$$

Considering only catarrhines, humans have 79.1% greater adult brain mass than expected. Only a minority of this extreme encephalization is therefore evident at birth, although more so relative to catarrhines than to primates in general.

From his analyses, Leutenegger concluded that humans do not produce unusually encephalized neonates, rather they produce unexpectedly large neonates relative to adult female mass, and these large neonates have large brains, but not disproportionately so compared with other primates (Leutenegger, 1982). Our updated analyses suggest that Leutenegger slightly underestimated the magnitude of encephalization in the human neonate, but was correct in concluding that increased body mass was the main outlying characteristic of our species.

Species can be further compared by assessing the production of neonatal mass in relation to the duration of gestation, measured in days. The updated regression equations for primate and catarrhine species respectively are:

$$\begin{aligned} \text{Ln birth mass} &= -1.541 + (0.039 \times \text{Gestation length}) \\ & \quad (5a) \end{aligned}$$

$$\text{Ln birth mass} = 2.842 + (0.019 \times \text{Gestation length}) \quad (5b)$$

Humans produce offspring with almost 80% less body mass than expected for the duration of gestation relative to primates in general, suggesting that humans achieve their large neonatal mass in spite of, not because of, their gestation period of 267 days (Harvey et al., 1987). However, considering only catarrhines, humans achieve 12.9% greater neonatal mass than expected, and in doing so, also achieve greater than expected rates of fetal brain growth [Eqs. (2b and 3b)]. How humans

might manage to achieve these high rates of growth is discussed in the following section.

Independent of their fetal growth pattern, Figure 3e shows that humans achieve greater adult brain mass than expected for their brain mass at birth. The regression equations for primates and catarrhines respectively are:

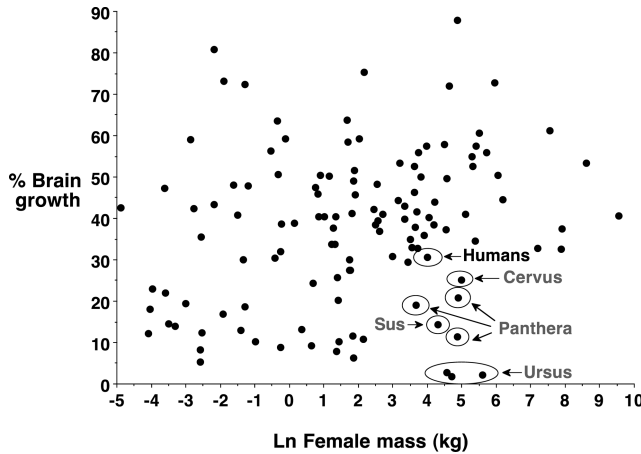
$$\begin{aligned} \text{Ln Maternal brain} &= 0.815 + (0.983 \times \text{Ln Neonatal brain}) \\ & \quad (6a) \end{aligned}$$

$$\begin{aligned} \text{Ln Maternal brain} &= -0.495 + (1.267 \times \text{Ln Neonatal brain}) \\ & \quad (6b) \end{aligned}$$

The slope of 0.983 for Eq. (6a) shows that the overall scaling is near-isometric in primates in general, however humans achieve 46.8% greater adult brain mass than expected, despite also having 14.5% greater brain mass than expected for body mass at birth. Considering only catarrhines, however, the slope is greater than isometric, and humans are much smaller outliers, having only 8.9% greater brain mass than expected. This reflects the greater average encephalization of catarrhines at birth. These findings can be squared with the results from Eq. (4) above, by considering that high adult encephalization in humans occurs in part by rapid brain growth in both pre-natal and postnatal life, and in part through slower rates of linear growth relative to brain growth in post-natal life. This has been argued previously by Deacon (1997).

Given that primates vary substantially in both adult brain size, and the rate at which this size is attained, a further comparison is the proportion of the adult brain grown during fetal life. Figure 3f plots the percentage of adult brain mass attained by birth against Ln adult female mass. This graph shows that across a wide range of body weight, the majority of primates grow between 30 and 60% of the adult brain in utero, with humans right at the lower limit. This suggests a minimum ceiling of "viable primate brain growth," i.e. at least one third of the adult primate brain appears to be required at birth, and supports Leutenegger's suggestion that a key trait under selection was achieving sufficient brain growth in utero to protect against excessive (i.e. unviable) altriciality (Leutenegger, 1982).

If, however, the same plot is produced for a larger sample of mammals, incorporating both primates and nonprimates (Barton and Capellini, 2011), then a number of other species can be seen to complete lower percentages of brain growth in fetal life (Fig. 4). The majority of these are rodents or carnivores, which give birth to altricial young underground or in nests. For larger mammals, humans once again are toward the lower limits of completed brain growth at birth. The other large-bodied species with low levels of brain growth by birth include three species of *Ursus*, who produce especially small neonates during hibernation, and four species (*Panthera*, *Sus*) producing young in nests. This larger sample supports the notion that primates in general have a higher minimum brain proportion at birth than mammals in general, and that humans lie at the very lower limit of this range. Indeed, human neonates born premature have very low survival rates in the absence of modern technology, having inadequate brain development and gastrointestinal function for effective breast-feeding (Klein, 2002). They also have relatively high metabolic



**Fig. 4.** Percentage of adult brain mass achieved at birth in a sample of mammals. Based on the data of Barton and Capellini (2011).

needs per unit weight, and inadequate fat stores to buffer energy requirements (Olhager and Forsum, 2003).

Collectively, these analyses offer some support for the hypothesis that the offspring's contribution to the obstetric dilemma derives in part from the need to grow a viable proportion of the adult brain by birth. However, a large part of this stress devolves to the larger neonatal body mass. It is likely that the relatively large body size at birth is closely related to the brain size that is only slightly larger than that of other primates in relative terms, but substantially larger in absolute terms. First, increased fetal adiposity may be important for buffering the obligatory energy demands on the brain in early life (Kuzawa, 1998). Second, the large head may require the early development of large shoulders to provide anatomical support. Third, increased mass of some vital organs may also be required to support the high neonatal brain BMR (Holliday, 1978). For each of these reasons, the encephalized brain may favor larger neonatal size overall, which may then impact the obstetric dilemma. Growing such neonates of larger size is negligibly accommodated by lengthening the gestation period, as the 267 days period of humans is similar to that of orang-utans (261 days) and gorillas (256 days) although slightly longer than that of chimpanzees (~228 days) (Brandt and Mitchell, 1971), suggesting that faster fetal weight gain per unit time is the primary mechanism. However, the constraints on offspring brain mass are not necessarily the same as those on total offspring mass. The minimal level of fetal brain growth results in human offspring being semialtricial at birth, and it has been suggested that they continue fetal rates of brain growth "extra-uterine" in early postnatal life (Portmann, 1941; Montagu, 1961).

If achieving a minimal level of viable brain mass, and an unusually large body mass and wide shoulders, by birth constitute the offspring's contribution to the obstetric dilemma, from where does the maternal contribution derive?

#### MATERNAL ACCOMMODATION OF DELIVERING LARGE INFANTS

There is increasing appreciation that whatever novel stresses bipedal locomotion placed on the maternal pel-

vis in humans, the obstetric dilemma is not a new evolutionary phenomenon. Leutenegger cited evidence for a variety of smaller primates experiencing difficulties giving birth (Leutenegger, 1982) while Lefebvre and Carli (1985; p 315) observed "mild or severe discomfort, in the form of straining, stretching, arching, grimacing, writhing, shaking, doubling up, eye closure, and restlessness" during delivery in 69 of 88 individuals from 29 species of nonhuman primate. In smaller primate species, delivery requires either the dilation of pelvic ligaments during parturition, or the temporary deformation of the fetal cranium, or both (Leutenegger 1982).

Leutenegger (1982; p 90) noted that "abortions, stillbirths, and miscarriages are especially frequent in those species with particularly large neonatal crania," and cited evidence that mortality from such causes exceeded 50% in squirrel monkeys (Goss et al., 1968). However, closer inspection of these mortality data suggests that other factors besides the obstetric dilemma are also relevant. Amongst marmosets, high mortality rates were inflated by a high proportion of second-generation mothers being primiparous (Phillips, 1976), an established risk factor for neonatal mortality (Brandt and Mitchell, 1973). In squirrel monkeys, of the 12 neonatal deaths (41%) amongst 29 births reported by Goss et al. (1968), only three were listed as obstetric death and the cause was not stated in the other nine deaths. The average birth weight of those dying (98 g) was negligibly higher than that of those surviving (94 g). Both social and dietary factors have been suggested to contribute to high neonatal mortality rates in captive primates (Hampton et al., 1966; Bowden 1967; Lehner 1967). Another reason for high neonatal death rates in marmosets may be their tendency to produce twins (Hampton and Hampton, 1965). Leutenegger (1973) suggested that producing multiple offspring in callithrichids was favored by selection against over-sized offspring to reduce the obstetric dilemma, but an alternative hypothesis is that multiple births are favored because callithrichids are colonizing animals. Thus, further data specifically on the obstetric dilemma are required in small primates, and particularly in wild populations, to re-evaluate the magnitude of the problem, although such data are notoriously difficult to obtain (Brandt and Mitchell, 1971).

The main implication of these primate analyses is that bipedalism may be less fundamental to the clash of maternal pelvic size and offspring brain size than is often assumed. This does not mean that bipedalism has not acted as a major stress on the structure and morphology of the human pelvis. In fact, Figures 1 and 2 suggest that the obstetric dilemma in early hominins was probably the result of obligatory bipedality in *Australopithecus*. However, as the comparative evidence demonstrates, bipedalism is merely one amongst a wide variety of locomotory systems that can contribute to obstetric tension (Rosenberg and Trevathan, 2002; Trevathan, 2011). Humans therefore face a delivery problem similar to that of many smaller primates, but have evolved a novel suite of birth mechanisms to address it (Rosenberg, 1992; Rosenberg and Trevathan, 2002).

The striking contrast in normal delivery mechanism between humans and nonhuman primates indicates that birth did indeed become a more complex process during human evolution, characterized by several adaptations. Yet, none of these adaptations need necessarily translate into high obstetric mortality rates in mothers and offspring. Rather, successful adaptations should arguably

achieve the opposite, by overcoming these risks in the majority of individuals. The evidence discussed above indicates that these adaptations seem less successful in many human populations than might be expected following hundreds of generations of selection in our species, suggesting that the obstetric dilemma might have become exacerbated more recently.

### RE-EVALUATING BIPEDALITY AS THE KEY CONSTRAINT

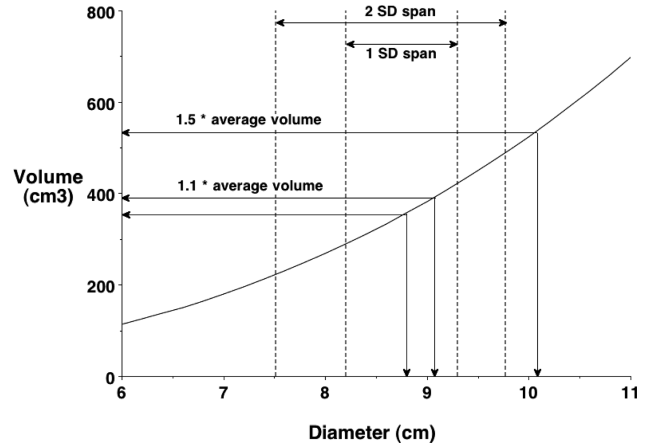
According to one perspective (Epstein, 1973), the magnitude of the biomechanical problem may have been over-estimated, due to misrecognition of the small-scale changes required to “resolve” the obstetric dilemma.

Epstein (1973) argued that brain volume (and hence approximately weight) scales with the cube of skull circumference. A value of human neonatal brain mass of 368 (SD 66) g, based on 1,024 measurements (from sources in DeSilva and Lesnik, 2006), was used to repeat Epstein’s simulations. Assuming a density of brain tissue of 1.036 (Blinkov and Glezer, 1968), this equates to 355.2 cm<sup>3</sup>. Since the volume ( $V$ ) of a sphere is given by the formula:

$$V = 4/3 \times \pi \cdot r^3 \quad (7)$$

where  $r$  is the radius, the diameter ( $2r$ ) of a 355 cm<sup>3</sup> spherical brain is 8.8 cm. Figure 5 plots brain volume against diameter, again assuming a spherical form. It can be seen that the range of brain volumes spanned by  $\pm 2$  standard deviations equates to a head diameter range of 7.5 to 9.8 cm. The equivalent range of brain volumes spanned by  $\pm 1$  standard deviations equates to a head diameter range of 8.2 to 9.3 cm. A 10% increase in brain volume requires a very small change in diameter of 0.28 cm, or 3.2%. Even a 25% increase in brain volume requires only +0.68 cm of diameter, equivalent to a 7.7% increase, and a 50% increase in brain volume is only marginally outside the range given by two standard deviations. These percentage increases also apply to maternal pelvic circumference.

These simulations suggest that actual variability in neonatal brain volume and in maternal pelvic dimensions between individuals is massive compared with trivial changes in the same dimensions within mothers or offspring that could potentially reduce the magnitude of the obstetric dilemma. This conclusion is supported by data on population variability in internal dimensions of the human pelvis. Table 2 presents comparative data from a range of contemporary and past populations on the anterior–posterior (AP) and transverse ( $T$ ) dimensions of the pelvic inlet and outlet, along with the pelvic brim index, calculated as  $[(\text{inlet AP}/\text{inlet } T) \times 100]$  (Angel, 1978). Although methodological inconsistency undoubtedly contributes, these data indicate a substantial degree of between-population variability, equivalent for some outcomes to differences greater than 2 within-population standard deviations. The coefficient of variation for all data is  $\sim 7\%$  for inlet values, but  $\sim 14$  and  $\sim 11\%$  for outlet AP and  $T$  values respectively, and  $\sim 9\%$  for the pelvic brim index. The greater variability in outlet  $T$  versus AP dimensions is of particular interest, and discussed later in this review. In contrast to such pelvic variability, the between-population coefficient of variability for neonatal head girth is  $< 3\%$  (Leary et al., 2006a). The conclusion from these contrasting magnitudes of variability is consistent with Epstein’s arguments, in that locomotion alone is unlikely to be the key maternal



**Fig. 5.** Brain volume against diameter, assuming a spherical form. The range of brain volumes spanned by  $\pm 2$  standard deviations equates to a head diameter range of 7.5 to 9.8 cm. The equivalent range of brain volumes spanned by  $\pm 1$  standard deviations equates to a head diameter range of 8.2 to 9.3 cm. These ranges indicate that very modest changes in head diameter, and by extrapolation maternal pelvic diameter, can generate large percentage changes in brain volume. Data from DeSilva and Lesnik (2006).

factor relevant to the obstetric dilemma in contemporary humans, given the reasonable assumption that these populations are all equally adept at bipedalism.

An elegant study by Kurki (2007) demonstrated that among Later Stone Age foragers of Southern Africa, with characteristically small body size, the female pelvis possesses an enlarged pelvic canal, as a compensatory mechanism to accommodate childbirth (Kurki, 2007). High levels of terrestrial mobility are characteristic of this population (Stock and Pfeiffer, 2001), which indicates that obstetric accommodation in the female pelvis need not negatively affect locomotor efficiency. Assuming no significant population variability in maternal locomotor mechanics regarding the data in Table 2, a relatively large neonatal head in one population could be easily accommodated by the average maternal pelvis of another population. Indeed, while the human pelvis is clearly adapted for upright posture and bipedalism, evidence of biomechanical penalties for variability in pelvic shape has not been presented, and the hypothesis appears unsupported (Lewton, 2012). The striking conclusion from this analysis is that constraints other than bipedalism are required to explain the close fit between neonatal head girth and pelvis dimensions within individual mothers.

Only one study appears to have examined the heritability of human pelvic dimensions directly (Sharma, 2002). Comparing 30 monozygotic and 30 dizygotic twin pairs, Sharma concluded that although growth and development of pelvic traits were “under the control of similar biological determinants . . . the environmental factors affecting these traits in the two zygositys are not similar,” with dizygotic twins significantly more variable than monozygotic twins for several outcomes. The analyses indicated that 60 to 80% of the variability was genetic, however, these estimates are unreliable due to the data invalidating some of the assumptions on which the classic twin study design is based (Sharma, 2002). Furthermore, twin studies routinely overestimate genetic heritability (Wells and Stock, 2011) although the magnitude of over estimation remains unclear, and the

TABLE 2. Pelvic dimensions across contemporary and recent populations

| Population                   | n       | Date              | Inlet AP (cm) |     | Outlet AP (cm) |     | Inlet T (cm) |     | Outlet T (cm) |     | BI   | Reference                     |
|------------------------------|---------|-------------------|---------------|-----|----------------|-----|--------------|-----|---------------|-----|------|-------------------------------|
|                              |         |                   | Mean          | SD  | Mean           | SD  | Mean         | SD  | Mean          | SD  |      |                               |
| <b>In vivo data</b>          |         |                   |               |     |                |     |              |     |               |     |      |                               |
| China                        | 300     | 1956              | 11.9          | -   | 11.3           | -   | 12.6         | -   | -             | -   | 94.4 | Chen et al., 1956             |
| England                      | 375+    | 1940              | 11.8          | 1.0 | 12.0           | 0.9 | 13.1         | 0.7 | -             | -   | 90.1 | Ince and Young, 1940          |
| Nigeria                      | 51      | 1978              | 11.1          | 1.0 | 7.8            | 1.1 | -            | -   | -             | -   | -    | Kolawole et al., 1978         |
| Philippines                  | 500     | 1954              | 11.8          | -   | 7.3            | -   | 12.3         | -   | -             | -   | 95.9 | Manahan et al., 1954          |
| Saudi Arabia                 | 88-93   | 1995              | 10.9          | 1.0 | 11.4           | 1.1 | 13.2         | 0.8 | -             | -   | 82.6 | English and Alcoair, 1995     |
| Sweden                       |         |                   | 12.2          |     | 11.9           |     | 13.8         |     |               |     |      | Ohlsen 1980                   |
| Uganda                       |         |                   | 10.0          | 0.8 | -              | -   | 11.2         | 0.7 | 10.0          | 0.7 | 89.3 | Albrook, 1962                 |
| United States                |         |                   | 10.5          |     | 11.5           |     | 13.5         |     | 10.0          |     | 77.8 | Pritchard and Macdonald, 1985 |
| Wales                        | 50      | 1978              | 12.1          | 1.0 | 8.2            | 1.3 | -            | -   | -             | -   | -    | Kolawole et al., 1978         |
| Mean                         |         |                   | 11.4          |     | 10.2           |     | 12.7         |     | -             |     | 88.4 |                               |
| Coefficient of variation (%) |         |                   | 6.8           |     | 19.9           |     | 6.6          |     | -             |     | 7.9  |                               |
| <b>Skeletal data</b>         |         |                   |               |     |                |     |              |     |               |     |      |                               |
| African pygmy                | 6       | 20th century      | 10.2          | 0.7 | 11.3           | 0.9 | 10.9         | 0.8 | 8.8           | 0.6 | 93.6 | Kurki, 2011                   |
| Andaman Islands              | 6       | ca 1860-1900      | 10.4          | 0.6 | 10.8           | 1.0 | 10.5         | 0.5 | 10.6          | 0.9 | 99.0 | Kurki, 2011                   |
| Australia                    | 8       | 19th century      | 10.6          | 1.1 | 11.5           | 1.4 | 11.8         | 0.9 | 10.7          | 0.9 | 84.8 | Kurki, 2011                   |
| Denmark                      | 8       | Medieval          | 10.3          | 1.0 | 11.1           | 0.8 | 13.3         | 0.7 | 12.2          | 0.9 | 77.4 | Kurki, 2011                   |
| India                        | 64      | 1929              | 10.1          | 0.7 | 7.9            | 0.7 | 11.9         | 0.7 | 8.6           | 0.9 | 84.9 | Pan, 1929                     |
| Inuit                        | 27      | 800-1900 AD       | 10.4          | 0.9 | 12.1           | 1.3 | 13.1         | 0.8 | 11.7          | 1.0 | 79.4 | Kurki, 2011                   |
| Northern Africa              | 17+     | 2000 BC-550AD     | 11.1          | 0.9 | 11.2           | 1.0 | 12.2         | 0.7 | 11.6          | 0.9 | 91.0 | Kurki, 2011                   |
| Nubia                        | 36      | 550-1550 AD       | 10.3          | 0.7 | 11.1           | 0.7 | 11.6         | 0.7 | 9.7           | 0.9 | 88.8 | Sibley et al., 1992           |
| Philippines                  | 8       | 19th century      | 9.9           | 0.6 | 11.0           | 1.1 | 11.4         | 0.6 | 10.2          | 0.8 | 86.8 | Kurki, 20121                  |
| Portugal                     | 40      | 19th-early 20th C | 11.0          | 1.0 | 11.1           | 1.6 | 12.6         | 0.9 | 11.3          | 1.2 | 87.3 | Kurki, 2011                   |
| South Africa                 | 67      | 20th century      | -             | -   | 11.6           | 0.8 | 12.0         | 0.8 | -             | -   | -    | Heynus, 1944                  |
| Southern Africa              | 16+     | 240-5370 BP       | 10.1          | 1.0 | 10.2           | 0.8 | 11.2         | 0.9 | 9.9           | 1.0 | 90.2 | Kurki, 2011                   |
| Tierra del Fuego             | ca 1880 |                   | 10.6          | 0.7 | 12.9           | 1.1 | 13.0         | 0.6 | 11.9          | 0.7 | 81.5 | Kurki, 2011                   |
| US Europeans                 | 40      | 19th-20th C       | 11.3          | 1.2 | 11.5           | 0.9 | 13.2         | 0.8 | 11.8          | 1.1 | 85.6 | Kurki, 2011                   |
| US Amerindians—Knoll         | 4+      | 3000-2000 BC      | 10.8          | 0.7 | 11.4           | 0.9 | 13.4         | 0.7 | -             | -   | 80.6 | Tague, 1986                   |
| US Amerindians—Pecos Pueblo  | 6+      | 16th century      | 9.1           | 0.8 | 11.4           | 0.9 | 13.4         | 0.6 | -             | -   | 67.9 | Tague, 1986                   |
| US Amerindians—Libben        | 11+     | 800-1100 AD       | 9.8           | 0.8 | 12.1           | 0.6 | 13.4         | 0.7 | -             | -   | 73.1 | Tague, 1986                   |
| US Amerindians—Haida         | 8+      |                   | 11.2          | 1.0 | 13.5           | 1.0 | 13.5         | 0.8 | -             | -   | 83.0 | Tague, 1986                   |
| Mean                         |         |                   | 10.1          |     | 11.3           |     | 12.4         |     | 10.7          |     | 84.7 |                               |
| Coefficient of variation     |         |                   | 5.4           |     | 10.1           |     | 7.9          |     | 11.2          |     | 9.0  |                               |
| <b>All data</b>              |         |                   |               |     |                |     |              |     |               |     |      |                               |
| Mean                         |         |                   | 10.7          |     | 11.0           |     | 12.5         |     | 11.2          |     | 85.5 |                               |
| Coefficient of variation     |         |                   | 7.2           |     | 13.6           |     | 7.7          |     | 10.8          |     | 8.6  |                               |

AP, anterior-posterior diameter; T, transverse diameter; BI, brim index = (AP/T) × 100 for inlet.

environmental influence on pelvic variability is likely to be greater than these calculations imply. Finally, extrapolating these data to singletons is difficult, because twins experience different conditions in utero, and the study was also restricted to external pelvic dimensions, and unable to address pelvic inlet and outlet dimensions, of most relevance here. Further work on the contribution of genetic factors to pelvic variability is therefore required, using family-study designs rather than twin studies.

However, a number of studies have provided indirect evidence for the intergenerational transmission of pelvic dimensions, which may arise through genetic or environmental mechanisms. Several studies have shown a familial occurrence of dystocia or caesarean section. Amongst non-Hispanic white women in the US, compared with those with birth weight in the range 2,500 to 3,999 g, those with higher or lower birth weight had ~3.2 times the risk of delivering by caesarean (Shy et al., 2000). The risk of caesarean delivery was 1.8 in US women whose own birth had been complicated by cephalo-pelvic disproportion (Varner et al., 1996). Among Norwegians, mothers delivered by caesarean had ~1.5 times the risk of delivering their own offspring in the same way, whereas no such intergenerational association was apparent for the offspring of fathers delivered by caesarean section (Tollanes et al., 2008). In Sweden, the risk of dystocia was 1.7 for firstborn women themselves experiencing dystocia as a neonate, 3.5 for sisters whose elder sister had experienced dystopic labor, and 24 amongst twins if the other twin had previously experienced dystopic labor (Berg-Lekas et al., 1998).

The mechanisms underlying these intergenerational effects remain unclear. On the one hand, in a sample of Indian women from Mysore, maternal head circumference and adiposity were associated with placental shape and efficiency (Winder et al., 2011), suggesting nutritional effects. On the other hand, a recent study suggested several genetic loci may be involved (Algovik et al., 2010). Furthermore, both paternal and maternal contributions to the obstetric dilemma may be important. In a UK birth cohort, the risk of caesarean delivery in women of average height was increased for tall relative to short fathers (Stulp et al., 2011), most likely indicating a paternal genetic contribution to offspring size.

Rejecting the argument of that biomechanical factors were the primary constraint on maternal pelvic dimensions and variability therein, Epstein (1973) therefore proposed that metabolic factors were the key constraint on neonatal brain size, and this issue has subsequently been addressed in greater detail.

### OFFSPRING BRAIN METABOLISM AS A NUTRITIONAL STRESS

In an influential article published in 1983, Martin argued that neonatal brain size was a function of maternal metabolic turnover. Across a range of mammal species, both neonatal brain mass and maternal basal metabolic rate (BMR) scale with maternal weight to the power 0.75 (Martin, 1983). This pair of relationships thus indicates an isometric association between maternal BMR and neonatal brain mass. A slightly different argument was made by Hofman that the BMR-brain association was due to an association between maternal BMR and gestation time, such that lower BMR, by constraining gestation, would then constrain brain growth (Hofman, 1983).

In adult mammals, there is now robust evidence linking BMR with brain mass. In an analysis of 347 mammal species, controlling for phylogenetic relationships, a positive association was found between brain mass adjusted for body mass, and BMR adjusted for body mass (Isler and van Schaik, 2006). Subsequent studies suggested that this scenario might be specific to placental mammals, as marsupials do not differ systematically in the ratio of brain to body mass, despite having lower BMR (Weisbecker and Goswami, 2010). However, the relationship between maternal BMR and neonatal brain mass proposed by Martin has remained more controversial.

Martin's argument was critiqued by Pagel and Harvey, who found that a given maternal BMR value produced very different levels of brain mass in species with altricial versus precocial offspring, and that the isometric association of maternal BMR and neonatal brain mass was not supported if brain mass of the total litter, rather than the individual offspring, was considered (Pagel and Harvey, 1988). These authors favored a life-history approach, arguing that gestation length is the key variable influencing neonatal brain mass, and that shorter gestations and smaller neonatal brains occur in species with high risk of mortality (Pagel and Harvey, 1988; Pagel and Harvey, 1990).

Using a larger sample of mammals in combination with more powerful phylogenetic comparative methods, Barton and Capellini recently re-examined the maternal BMR offspring brain mass association. After removing effects of maternal body mass and neonatal body mass, neonatal brain mass remained correlated with gestation length, supporting the notion that relatively larger brains require a longer pregnancy (Barton and Capellini, 2011). Taking this association into account, a correlation between neonatal brain mass and maternal BMR was also evident, indicating that higher maternal BMR is an independent factor promoting fetal brain growth for a given gestation length (Barton and Capellini, 2011).

The large absolute brain mass of the human neonate certainly translates into high energy requirements. Both brain tissue, and that of other organs closely associated with brain metabolism such as the liver, have relatively high rates of energy expenditure (Holliday, 1978). At birth, human brain metabolism approximates 80% of total basal metabolism (Holliday, 1978). Such a high-energy demand might be expected to require a complementary increase in maternal BMR.

Although humans are typically portrayed as having the expected BMR for their body weight, on the basis of Kleiber's equation (Kleiber, 1961), it has been proposed that this ignores the unusually high level of adiposity in female humans. Few adult mammals have the high body fat content typical of human females, hence humans may conceal a relatively high BMR per kg lean mass through having a more conventional BMR per kg total body mass (Aiello and Wells, 2002). The magnitude of this effect has yet to be determined, due to the lack of data on basal metabolism and body composition in mammals. Given such an association, the maternal energy budget emerges as a plausible factor constraining neonatal brain mass at a relatively consistent level found across human populations. This can be addressed by modeling the effect of increasing fetal brain mass on fetal energy demand.

From Eq. (7), a 1% increase on neonatal head diameter equates to a 3% increase in brain volume. The increase

in cerebral energy expenditure may not be isometric with this increase in brain volume, as the energy cost of an organ tends to decline as the organ mass increases (Wells et al., 2004; Heymsfield et al., 2007); nevertheless it is clear that even modest increases in neonatal head girth would rapidly inflate fetal energy demand. Thus, it can be assumed that energetic limitations deriving from maternal metabolism contribute to the difficulty of increasing fetal brain growth, and hence push back in the opposite direction to the offspring's drive to grow a minimum viable neonatal brain mass.

However, high maternal metabolism is not the only possible adaptation whereby the high fetal energy demands can be met, for example the characteristics of the placenta may also be important (Leutenegger, 1973). For example, higher mass of individual offspring relative to mass of the mother in anthropoid versus prosimian primates was attributed by Leutenegger (1973) to improved placental efficiency. Consistent with this hypothesis, humans have the relatively more invasive "hemochorial" type of placenta, where the fetal tissues are bathed in maternal blood (Mossman, 1987; Wooding and Burton, 2008), favoring the transfer of nutrients (Haig, 1993; Elliot and Crespi, 2008). Taking phylogenetic relationships into account, different slopes for the scaling of brain size and growth rate were found for different types of placenta (Crespi, 2011), supporting the notion that hemochorial placentas are one adaptation for faster fetal growth. Similarly, the degree of placental interdigitation may also be important, as it affects the surface area of exchange between mother and fetus (Wooding and Burton, 2008). This could, in principle, affect nutrient transfer rates because a higher degree of interdigitation increases the surface area for exchange (Wooding and Burton, 2008). The most efficient form of interdigitation was found in labyrinthine placentas, which have a higher surface area of exchange between mother and fetus, and which were associated with significantly shorter gestation durations (Capellini et al., 2011), however, humans have relatively villous placentas with less surface area of exchange. Humans do not therefore have a type of placentation that systematically favors shorter gestation, instead they appear to have a biology favoring large neonates, and this in turn supports the notion of higher maternal BMR being important.

The termination of gestation may therefore have been determined by energetic rather than pelvic anatomical criteria, with the dimensions of the maternal pelvis then responding to the magnitude of fetal brain mass resulting from this selective pressure. Consistent with that approach, Ellison has argued that human birth occurs at the time-point at which fetal energy demand exceeds the capacity of maternal metabolism through placental nutrition (Ellison, 2001, 2008). In late pregnancy, some maternal skinfold thicknesses decline, indicating that the energy demands of fetal growth exceed the supply of energy directly available from maternal dietary intake (Forsum et al., 1989; Homko et al., 1999; Sivan et al., 1999). At this stage of pregnancy, maternal metabolism shifts to fat utilization, which may prioritize the offspring's access to carbohydrate substrates (Butte et al., 1999). Such carbohydrate prioritization is favored in part because glucose is the preferred substrate for fetal brain metabolism, and in part because it also allows offspring lipid deposition, whereas fatty acids have poor capacity to cross the placenta (Morris and Boyd, 1988; Aldoretta and Hay, 1995).

According to Ellison (2008), the cascade of signals that leads to the initiation of labor begins with the inability of the mother to adequately meet the fetus' metabolic requirements. For example, fetal cortisol stimulates prostaglandin production in the placenta, liberating essential fatty acids required by the fetal brain directly into the fetal circulation, and initiating contractions in the uterus (Majzoub et al., 1999; McLean and Smith, 2001). This "metabolic cross-over hypothesis" therefore treats the initiation of labor as a consequence of fetal metabolic requirements exceeding the maternal ability to meet them (Ellison, 2001, 2008). Following parturition, the offspring's increasing energy requirements can better be met by lactation, as lipids and fatty acids are more readily transferred by this route than by the placenta. This is further consistent with the conclusion of Barton and Capellini (2011), that lactation represents an independent developmental period during which rapid brain growth may be achieved.

The importance of maternal metabolism for fetal growth is indicated by the patterns of growth observed in neonates whose growth is not constrained, due to pathological alterations in maternal metabolism. Even in nondiabetic pregnancies, there is a direct association between maternal fasting glucose and neonatal weight, head circumference, and ponderal index (Hill et al., 2005). Diabetic pregnancies, where poor glycemic control results in high circulating glucose levels, are associated with macrosomic offspring with increased adiposity and body proportions (Nasrat et al., 1997; Catalano et al., 2003), including shoulder width (McFarland et al., 1998). Some studies indicate that macrosomic infants also have an increased head circumference at birth (Brans et al., 1983; Greene et al., 1995; Falhammar et al., 2010), although other studies found no such association (Nasrat et al., 1997; Falhammar et al., 2010), and one study found an increased risk of small head circumference in the offspring of diabetic mothers (Betti et al., 2011). Longer-term follow-up of macrosomic offspring demonstrates that increased head girth remains in childhood (Hediger et al., 1998; Evagelidou et al., 2006).

These data indicate that maternal metabolism is a key determinant of fetal growth, and that increased maternal fuel availability translates into offspring with larger body and brain mass at birth. Yet if energy supply is the primary constraint on fetal growth, what is it that prevents the pelvic dimensions from being larger, aiding the ease of delivery?

The fact that human delivery is difficult even when successful is due to the offspring routinely growing close to the maximum size permitted by the mother. It may be selected to adopt this approach because life in utero is relatively low-risk, particularly in relation to infectious diseases that are the primary cause of postperinatal mortality. Rather than maternal energy reserves being limited in absolute sense, given that fat reserves remain for lactation, there may be a maternal mechanism which transmits signals of inadequate energy supply to the fetus as it approaches maximum occupation of the uterine space. Such a dynamic mechanism would help explain the tendency for a close match between maternal and offspring dimensions, however several different factors, reviewed below, might disrupt this match, either by depressing the dimensions of the maternal pelvis, or by increasing the capacity of the offspring to grow in utero. In this context we consider first thermodynamic effects, then plasticity in maternal stature, long-term trends in

the human diet, and finally the stress of infectious disease which may have favored greater infant adiposity.

### MATERNAL SHAPE AS A THERMAL STRESS

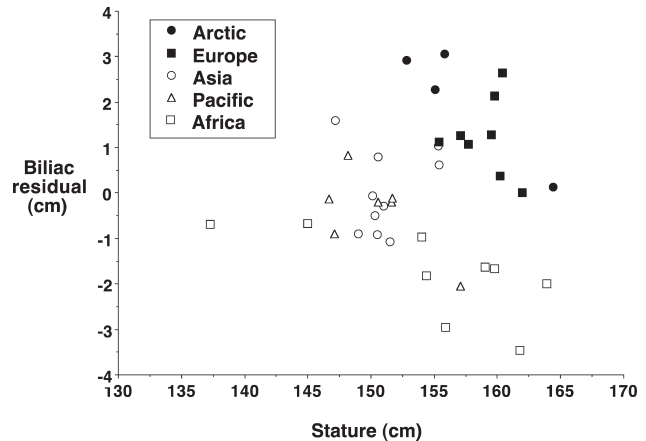
The thermal environment has been considered a key ecological pressure in the evolution of the *Homo* genus (Wheeler, 1992, 1993), and pregnancy adds significantly to heat stress in hot environments (Wells, 2002). The relationship between human body proportions and thermal environment is a function of fundamental physical laws. Initially expressed as ecogeographical “rules” in the 19th century (Bergmann, 1847; Allen, 1877), the heat production of biological organisms is assumed to be proportional to body mass, or body volume, whereas heat loss is assumed to be proportional to surface area (Ruff, 1994; Paterson, 1996; Katzmarzyk and Leonard, 1998). Human populations in hot environments are therefore predicted to maximize heat loss by a linear physique, maximizing the area: mass ratio, whereas populations inhabiting cold environments are predicted to minimize this ratio through a stocky physique, whilst further increasing total mass to increase heat production. Classic work by Roberts demonstrated associations between the thermal environment and body mass (Roberts, 1953), and further work has replicated his findings for body mass and physique (Hiernaux, 1968; Roberts, 1973; Hiernaux and Froment, 1976; Crognier, 1981; Ruff, 1994), and more recently for body composition (Wells, 2012a).

More recently, Ruff focused specifically on the implications of variability in torso breadth for thermoregulation. Treating the body as a cylinder, and treating cylinder volume as a proxy for body mass, he showed that increasing its length (equivalent to taller stature) whilst holding its breadth constant changed body mass in isometric proportion, but made no effect on the area: mass ratio (Ruff, 1994). Conversely, increasing cylinder breadth (equivalent to more stocky physique) whilst holding length constant altered both mass and area: mass ratio. This simple model indicates that from a thermodynamic perspective, variability in human body size should be targeted at stature for populations within a given thermal environment, but should be targeted at breadth for populations distributed across different thermal environments. Data from the literature on males and females supported this model, showing a relatively constant ratio of bi-iliac breadth to stature across a wide range of stature in populations from similar thermal environments, but increased bi-iliac-breadth:stature ratios at colder temperatures (Ruff, 2010). To re-evaluate this association, data on stature (cm) and bi-iliac breadth (cm) from female populations was collated from the literature. Bi-iliac breadth was regressed (using reduced major axis regression) on stature, to give the following equation:

$$\text{Bi-iliac breadth} = -20.619 + (0.304 \times \text{height}) \quad (8)$$

$$n = 38; r^2 = 0.237$$

Figure 6 plots the regression residuals against height, categorizing the populations as Arctic, European, Asian, Pacific, or sub-Saharan African. The Arctic and European populations have systematically more positive residuals, while the other three populations have more negative residuals. In each grouping, the bi-iliac residual falls with increasing stature, as observed by Ruff.



**Fig. 6.** Association between bi-iliac breadth, expressed as the residual of the regression of bi-iliac breadth on stature, against stature in five groupings of female populations. In all groupings, the residual declines with increasing stature. Arctic and European populations have higher residuals than Asian, sub-Saharan and Pacific populations.

Ruff (1994) proposed that thermodynamic issues are therefore highly relevant to the obstetric dilemma, in that narrower pelvises are assumed to be favored in warmer climates. However, his arguments are here taken further, by incorporating the effect of variability in fetal energy demand. During pregnancy, fetal size contributes directly to maternal heat production (Wells, 2002). In hot environments, fetal weight therefore constitutes one source of heat stress, particularly since maternal physiology is the only pathway whereby fetal heat may be dissipated to the environment. Any increase in fetal energy demand therefore generates two related effects on maternal energy metabolism: the demand for more energy supply, and the demand for increased heat dissipation. Since these demands are necessarily antagonistic, increasing fetal energy expenditure must rapidly increase maternal heat stress, hence increasing the pressure to maintain body proportions that maximize heat loss.

This antagonistic tug-of-war between heat production and heat loss may therefore be one factor contributing to the relatively tight fit between maternal pelvic dimensions and offspring brain size, and its variability between populations. According to this approach, variability in energy availability would be assumed to impact stature rather than pelvic inlet area (Ruff, 1994), and the obstetric dilemma would be predicted to be more severe in hot environments.

In a study of a small-bodied forager population from the Later Stone Age, Kurki found that despite narrow bi-iliac breadth, pelvic dimensions were similar to those of larger-sized humans, indicating that small maternal body size need not necessarily constrain fetal growth (Kurki, 2007). Further investigation suggested that this population was not strongly adapted to thermal stress (Kurki et al., 2008), suggesting that the small stature of the population might reflect limitations on energy availability rather than thermodynamic constraint. Other small-bodied populations vary substantially in bi-iliac breadth, many being relative broad and others (e.g. Andaman Islanders) being narrow (Kurki et al., 2008), yet extrapolating from the study of Kurki (2007), narrow bi-iliac breadth need not necessarily imply narrow obstetric

dimensions. It thus remains unclear how severely thermal load impacts directly on pelvic dimensions.

In general, pregnancy increases thermal load through weight gain, although among Gambian farmers inhabiting a highly seasonal environment, pregnancy could proceed during a net loss of fat (Lawrence et al., 1987). Intriguingly, in this population basal metabolism was found to decrease during pregnancy (Poppitt et al., 1994), thus reducing internal heat production. Whilst this was interpreted by the authors as “energy-sparing,” and hence adaptive in the face of limited energy supply, it might also be considered as ‘heat-sparing’, and this issue merits further research.

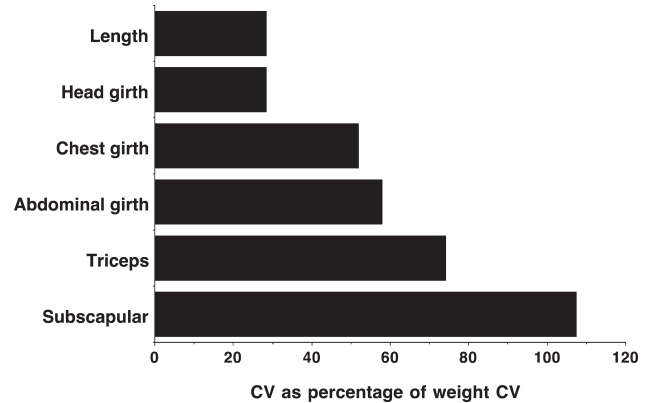
So far, in this review we have attempted to update our understanding of what constitutes the obstetric dilemma. The primary conclusions are that the tension derives on the one hand from the need of the offspring to grow a certain viable brain mass, supported by an appropriate body mass, and on the other hand from the maternal constraints arising from limits on energy availability or possibly thermal stress, rather than directly from maintaining efficient bipedalism. In the remainder of the review we consider how each of maternal pelvic dimensions and neonatal dimensions may vary over time, in part through transgenerational phenotypic plasticity, and hence consider how variable degrees of plasticity between the two parties can introduce variability in the magnitude of the dilemma.

### PLASTICITY IN THE OFFSPRING

Examining the components of size at birth amongst human populations, it is clear that traits differ markedly in their degree of variability. Mean birth weight itself varies substantially across populations, with a coefficient of variation (CV) for 140 populations of 7.1% (Wells and Cole, 2002), while within populations the CV is typically ~14%. In a more detailed study of neonatal anthropometry in samples from England, Scotland, Finland, DR Congo, Nigeria, Jamaica, India, Sri Lanka, and China, between-population CVs were 8.1% for birth weight, 8.7% for subscapular skinfold, 6.0% for triceps skinfold, 4.7% for abdominal girth, 4.2% for chest girth, and 2.3% for each of head girth and length (Leary et al., 2006a). Thus, plasticity in weight, adiposity, and lean tissue appears to buffer more tightly regulated fetal growth of length and the brain. Figure 7 shows CVs for various body proportions expressed as a percentage of the CV in weight.

The brain is not the only neonatal characteristic challenging pelvic dimensions. In contemporary human populations, macrosomic infants induce substantial elevations in the risk of a caesarian delivery, and also in the risk of shoulder dystocia, other birth injuries and neonatal asphyxia (Berard et al., 1998; Ju et al., 2009; Ezegwui et al., 2011; Vidarsdottir et al., 2011). The risk of shoulder dystocia increases linearly with birth weight (Gherman et al., 2006). It has been suggested that humans would require a rotational birth mechanism purely on account of the unusually broad neonatal shoulder width (Trevathan, 1988), and that this scenario may have preceded a more complex rotational mechanism addressing large head girth subsequently (Rosenberg and Trevathan, 2002).

The greater variability in nonbrain versus brain components of neonatal phenotype supports the notion that achieving a viable minimum brain mass is the primary



**Fig. 7.** Between-population coefficients of variation for anthropometric variables in newborns. Based on data of Leary et al. (2006a).

selective pressure acting on fetal growth. Compared with European neonates, Indian neonates are characterized by a “thin-fat” phenotype, comprising substantial deficits in indices of lean mass, but relatively preserved adiposity (Yajnik et al., 2003). Despite a ~24% reduction in average birth weight (2.66 vs. 3.49 kg), the equivalent reduction in head circumference was only ~6% (33.1 vs. 35.2 cm), indicating that other components were sacrificed to preserve brain mass (Yajnik et al., 2003). A slight reduction in head girth would also be anticipated, as Indian infants have very slightly shorter typical gestation than European infants (Meire and Farrant, 1981; Patel et al., 2004). Although the reduction in head girth would not appear so small if converted from circumference (linear dimension) to volume (cubic dimension), these data nevertheless indicate that in response to chronic under-nutrition, fetal brain growth is protected, a key tenet of the “thrifty phenotype” hypothesis (Hales and Barker, 1992).

What is most notable here is the relative reduction in neonatal brain variability compared with the maternal pelvic variability described in Table 2. The relatively low level of plasticity in fetal brain growth supports the notion that it has played a key role in the long-term evolutionary emergence of the obstetric dilemma, responsible for the rotational birth mechanism and lengthy duration of labor. However, it is potentially the greater plasticity of fetal body proportions, interacting with similar plasticity in maternal phenotype, that may contribute to short-term variability in the magnitude of the obstetric dilemma across populations and over time.

### PLASTICITY IN THE MOTHER

Plasticity in the mother may relate to a variety of components of size, with clear implications for obstetric difficulties. In contemporary populations, narrow pelvic proportions increase the risk of caesarean delivery. Compared with a control group of 30 women delivering vaginally, both pelvic outlet (328 vs. 346 mm,  $P = 0.0024$ ) and inlet (245 vs. 255 mm,  $P = 0.0038$ ) were reduced in 30 Swedish women delivering by caesarean section (Stalberg et al., 2006). What is also clear, however, is that a variety of other maternal dimensions are relevant, and are associated with variability in pelvic dimensions and the risk of complications during delivery. For example, foot length  $\leq 23$  cm, and intertrochanteric diameter



TABLE 3. Measurements of stature and pelvic dimensions in women categorized by pelvic type

|                       | Type of pelvis |              |              |             |
|-----------------------|----------------|--------------|--------------|-------------|
|                       | Dolichopellic  | Mesatipellic | Brachypellic | Platypellic |
| Clinic women          |                |              |              |             |
| <i>n</i>              | 18             | 58           | 51           | 5           |
| Stature (cm)          | 158.5          | 159.3        | 155.6        | 158.3       |
| Sitting height (cm)   | 84.6           | 84.5         | 83.1         | 84.4        |
| Pelvic inlet AP       | 12.8           | 11.8         | 10.7         | 9.9         |
| Pelvic inlet <i>T</i> | 12.1           | 12.4         | 12.8         | 13.2        |
| Cephalic index        | 79.7           | 81.1         | 81.6         | 78.9        |

Data from Greulich et al., 1939.

≤30 cm are both associated with increased risk, indicating that smaller body dimensions may index a reduced pelvic capacity (Benjamin et al., 2012). While the pelvis represents the direct constraint against which neonatal head size is pitted, substantial work has demonstrated that the magnitude of this constraint is strongly mediated by maternal stature.

The importance of stature for birth complications was well recognized by early 20th century obstetricians, who noted high rates of perinatal mortality in the main urban centers of 19th century industrialization (Baird, 1949; Illsley, 1966). Illsley identified strong north-south gradients in both perinatal mortality, and in maternal height. Within each geographical region, and within social classes, perinatal mortality and maternal height remained correlated. A large proportion of this association (e.g. 1,058 of 1,282 caesarean sections performed in 1911) could be attributed to rickets, which involved significant flattening of the pelvis in response to nutritional deficiencies during early maternal development (Dick, 1922). The majority of these caesarean sections were undertaken in the main urban conurbations, with 383 in London, 304 in Glasgow, 155 in Manchester, 96 in Liverpool, and 92 in Sheffield (Dick, 1922), where living conditions in previous generations had been very poor.

In these early industrial populations, short stature and pelvic deformation had common origins in early-life undernutrition, exacerbated by lack of adequate sunlight. While rickets is not documented widely amongst the world's population, and was considered relatively absent outside western industrializing cities until recently (Wells, 1975), a more general association between maternal stature and perinatal mortality is evident from data from numerous populations. Both perinatal mortality and caesarian delivery index the obstetric dilemma in contemporary populations, albeit imperfectly.

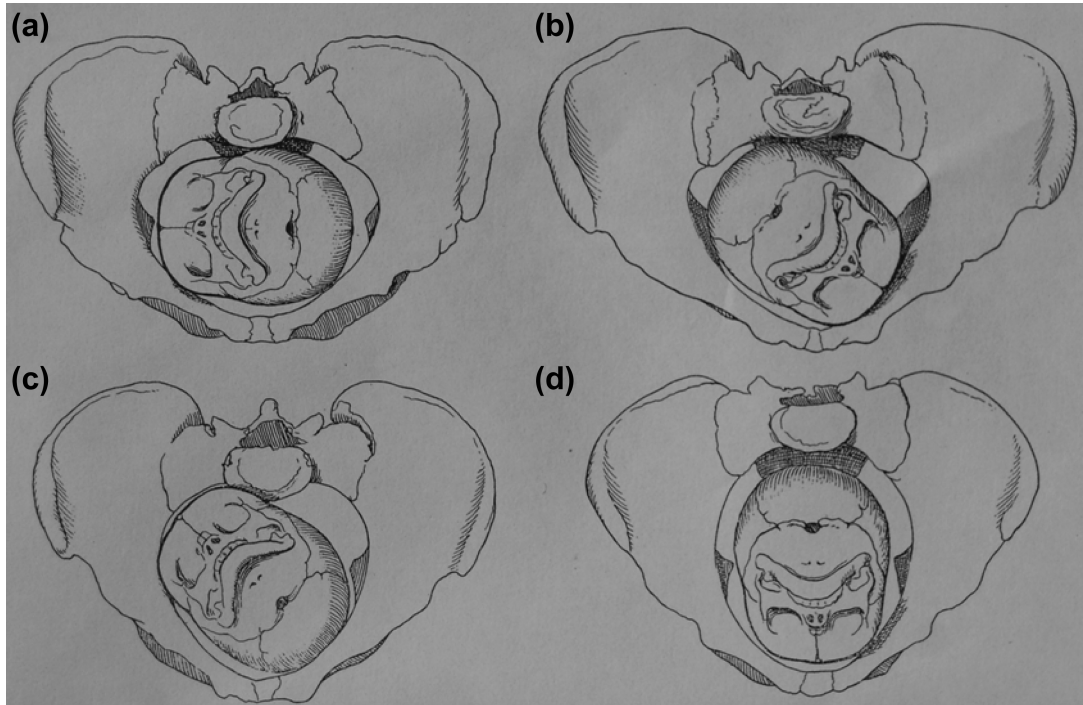
In contemporary populations, short stature remains a major risk factor for caesarian delivery. Numerous studies have noted that tall maternal stature is protective against conditions leading to caesarean section (Mahmood et al., 1988; Sokal et al., 1991; Kwawukume et al., 1993; Read et al., 1994; Moller and Lindmark, 1997; Cnattingius et al., 1998; Khunpradit et al., 2005; Smith et al., 2005; Stulp et al., 2011) and birth injury (Gudmundsson et al., 2005; Rozenholc et al., 2007). In a study of nulliparous women delivering singleton births in Sweden, the odds ratio for caesarean delivery for women 155 to 164 cm was 2.0, and 4.5 for short women (<155 cm), compared with the reference group of tall women (>174 cm) (Cnattingius et al., 1998). Likewise in Western Australia, the odds ratio of caesarean delivery was 2.4 for women 160 to 164 cm and 5.3 in women <160 cm, compared to those >164 cm (Read et al., 1994). In Malawi and Nigeria, the odds ratio of caesarean delivery was 3.8 for women maternal height ≤154

cm, compared with those ≤155 cm (Brabin et al., 2002). In rural Nepal, maternal stunting and large infant head circumference each increased the risk of neonatal asphyxia, categorized by failure to cry at birth (Lee et al., 2009). There is a potential circular argument, in that clinicians may, on the basis of prior evidence, elect to perform caesarean deliveries in women of shorter stature; however, other studies show an increased risk of birth complications in general in shorter women, especially when the offspring is macrosomic (Gudmundsson et al., 2005; Gupta et al., 2010; Monasta, 2011).

Variability in pelvic dimensions has further been linked with stature variability. For example, Bernard (1950) showed in a sample of women from Aberdeen, UK that shorter stature was associated with a flatter pelvis, with this association exacerbated if the individual was also of poor nutritional status. Several categorizations of pelvic shape have been proposed (Caldwell and Moley, 1933; Thoms, 1937), though such categorizations have also been critiqued, in part because they reflected antiquated racial perspectives on skeletal variability, and in part due to the artificiality of defining groups from a continuous range of variability (Walrath, 2003). One commonly used categorization is as follows: dolichopellic pelvises have anteroposterior greater than transverse diameter; brachypellic pelvises have transverse diameter greater than anteroposterior diameter; mesatipellic pelvises have relative similar anteroposterior diameters, and platypelloid pelvises have a significantly flattened oval shape due to reduced anteroposterior diameter (Thoms 1937). In a sample of primigravida white women from New Haven, CT, Greulich et al. (1939) found that these pelvic types were associated with variability in both height and shape of the maternal head (cephalic index) (Table 3). If these characteristics were reproduced across generations, then the shape of the offspring's head would vary systematically with the shape of the mother's birth canal. However, this has yet to be demonstrated empirically and merits investigation.

In turn, early work suggested inter-population variability in pelvic shape (Martin, 1928), supported by the data in Table 2. Caldwell and Moley (1933) provided diagrams suggesting how different pelvic shapes affected the passage of the neonatal head, reproduced in Figure 8. Flattening of the pelvis may change the alignment of the head as it passes through the outlet.

Similarly, stature was found to be associated with inlet AP and T diameters and the pelvic brim index in British women (Ince and Young, 1938). A study of Ghanaian women demonstrated that those undergoing caesarean section for cephalo-pelvic disproportion had both shorter height (152.7 vs. 157.2 cm) and shorter pelvic inlet AP diameter (9.54 vs. 10.61) than those without disproportion (Adadevoh et al., 1989). However, in the Indian sample (*n* = 36) collected by Pan (1920), the outlet AP



**Fig. 8.** Drawings suggesting effects of pelvic shape variability on the position of the fetal head in the birth canal, from Caldwell and Moley (1933). Note that the categorization of discrete “pelvic types” has been criticized (Walrath, 2003), and the aim here is simply to illustrate potential variability in fetal alignment in relation to pelvic shape. (a) Platypelloid pelvis; (b) android pelvis; (c) gynecoid pelvis; (d) anthropoid pelvis. This article was published in *American Journal of Obstetrics and Gynecology*, Vol 26, WE Caldwell and HC Moley, Anatomical variations in the female pelvis and their effect in labour with a suggested classification, 479–505, Copyright Elsevier (1933).

diameter was the only pelvic dimension significantly associated with stature. Further work is required to establish with greater confidence the association between stature and pelvic dimensions, and potential variability in these associations between populations. Nevertheless, the limited available data on growth and change over time within populations suggests that increasing height is associated with increasing pelvic dimensions and reduced risk of cephalo-pelvic disproportion (Holland et al., 1982; Harrison et al., 1988).

In contrast to the association of maternal height with pelvic dimensions, however, any link between maternal height and offspring head circumference appears negligible. The association between maternal height (in cm) with neonatal head circumference (HC; in cm) was examined in a sample of Ethiopian mother-infant dyads (unpublished data). The regression equation is as follows:

$$\text{Ln Neonatal HC} = 3.267 + (0.055 \times \text{Ln maternal height}) \quad (9)$$

$$n = 600; r^2 = 0.000$$

Although these data are cross-sectional, the analysis suggests that decreases in maternal height may not have any significant effect on neonatal HC. This is consistent with the geographical studies of neonatal body proportions, in which neonatal HC was found to vary substantially less between populations than birth weight (Leary et al., 2006a). The strongest predictor of neonatal head circumference is maternal head circumference (Leary et al., 2006b), suggesting an intergenerational

association that may reflect a strong genetic component.

Given the association between maternal height and risk of cephalo-pelvic disproportion, Guegan et al. considered whether variability in maternal reproductive mortality rate might be associated with the degree of sexual dimorphism in stature across populations. Consistent with their mathematical model, sexual dimorphism was decreased in highly fecund populations in which the effect of stature-related mortality would be assumed to be stronger (Guegan et al., 2000). Within populations, parental height differences also influence the risk of caesarean delivery (Stulp et al., 2011). However, whilst sexual dimorphism is often assumed to reflect genetic adaptation, its magnitude also varies within populations over time (Guntupalli and Moradi, 2008), suggesting plasticity also contributes. In turn, plasticity in the magnitude of pelvic variability is suggested both indirectly, by evidence for secular trends in stature, and also directly by evidence on changes in pelvic dimensions.

Analyzing data from African-America populations, Kelley and Angel concluded that slavery was associated with depression of both stature and pelvic proportions, as well as exacerbating other indices of under-nutrition (Kelley and Angel, 1987). In a study of women from Northern Ireland, taller females were observed to have larger pelvic proportions, and the secular trend in height was associated with a similar trend in pelvic width (Holland et al., 1982). Due to the paucity of evidence on trends in pelvic shape, this would be a valuable area for further work.

These studies indicate that longer-term nutritional trends contribute significantly to maternal physical

proportions, and hence to the magnitude of the obstetric dilemma (Liston, 2003). Although height is a polygenetic trait and therefore has a component of genetic heritability, the magnitude of this heritability remains uncertain, due to the current approach to heritability quantification incorporating both genetic effects, and also nongenetic environmental effects involving epigenetic adaptations (Wells and Stock, 2011). Secular trends in maternal height are widely assumed to derive primarily from phenotypic plasticity, and to be distributed across successive generations, as demonstrated in primates (Price et al., 1999).

A valuable illustration of the importance of matrilineal nutritional history on the obstetric dilemma is given by a study of mixed-ethnic unions (Nystrom et al., 2008). Compared with the offspring of two European parents, offspring of a European father and Asian mother had an increased risk (OR 1.3) of caesarian delivery, whereas offspring of an Asian father and European mother had reduced risk (OR 0.84) despite very similar mean birth weight in the two mixed-ethnic groups (Nystrom et al., 2008). This implicates a tendency for inadequate maternal pelvic size in the Asian mothers relative to the offspring's growth potential contributed by larger European fathers, but not in the European mothers relative to the offspring's growth potential contributed by the Asian fathers. However, whether the reduced size of the Asian maternal pelvis relative to the European paternal height represents contrasting genetic or plastic adaptations is not clear from this analysis.

In animal studies, more direct evidence of plasticity in pelvic proportions comes from studies of parity. In mice where the timing of breeding and food availability were controlled, the production of offspring affected skeletal architecture (size and shape of the os coxae) of the female pelvis (Schutz et al., 2009a). These changes were furthermore correlated with the relative size of individual offspring. In humans, parity has been associated with femoral neck size (Specker and Binkley, 2005), and with subtle changes in bone morphology (Cox and Scott, 1992), but more direct evidence for parity affecting pelvic proportions is currently lacking.

Perhaps the most striking evidence for pelvic plasticity comes from the study of a goat born without forelimbs. This animal showed a marked narrowing of the internal dimensions of the pelvis, allowing the increased load of the upper body to be supported (Slijper, 1942). Such dramatic remodeling of the pelvis early in the life-course indicates substantial plasticity, however, this issue remains relatively unexplored in humans, and the intergenerational associations discussed above suggest that adult phenotype has substantial plasticity across rather than within generations.

The final part of this review considers how the emergence of agriculture may have exacerbated the human obstetric dilemma, in two counter-balancing ways: first, changes in dietary quality and population density (impacting infectious disease risk) may have reduced maternal height; second, the same factors may have promoted offspring size through both plastic and genetic mechanisms.

### LONG-TERM TRENDS IN STATURE

In recent decades, secular trends in stature have been widely reported, often upwards in response to improving nutrition and decreasing risk of infectious disease

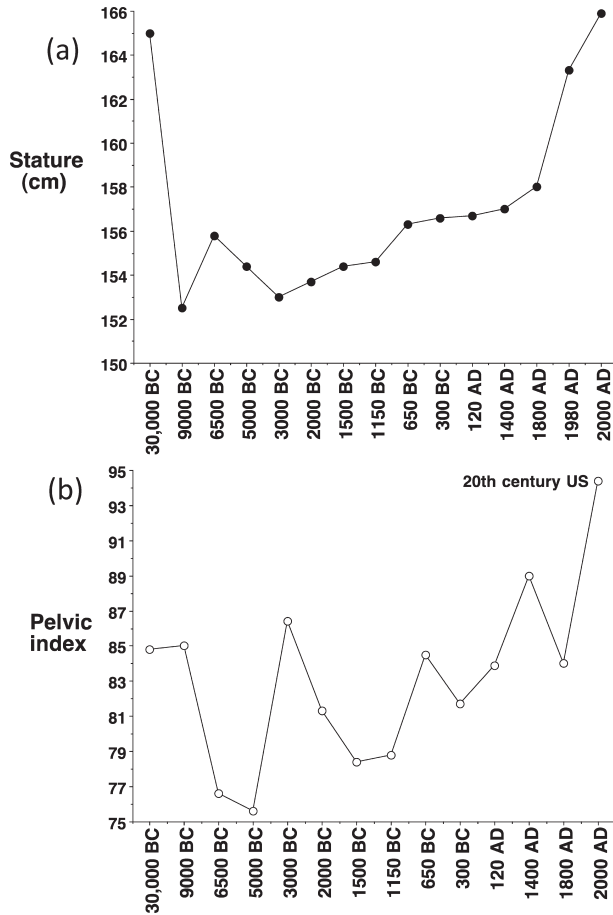
(Malina, 1990; Cole, 2000b), but occasionally negative secular trends have been noted in response to declining living standards (Tobias, 1985; Akachi and Canning, 2007; Stock and Migliano, 2009). There is considerable variation in human body size, with adult CVs being approximately 50% for body weight, 10% for stature, and 25% for body breadth (Ruff, 2002). In general, if the recent obesity epidemic is discounted, modern human body weight is lower today than it has been since the middle Pleistocene, and we see more variation within the Holocene, on account of culture increasingly working in concert with environmental factors that influence physique.

Body size variation following the transition to agriculture has received considerable attention. The most comprehensive analysis of body size in Pleistocene and Holocene Europe demonstrates that there was a significant reduction in stature from the Upper Paleolithic to the Mesolithic, with slight further reductions occurring with the origins of agriculture (Mieklejohn and Babb, 2011). A similar reduction in stature and body mass is also reported for the transition from the late Paleolithic populations of the Nile Valley, to the earliest agriculturalists, although body size rose again with the development of the Egyptian Empire (Stock et al., 2011). A recent study of subadult growth within skeletal series from the Mesolithic and Neolithic of the Danube Gorge, and the Greek Neolithic, provides evidence of growth trajectories which fall well below more recent British samples and standards from the Denver Growth Study (Pinhasi et al., 2011).

While there is now considerable evidence of a general decrease in body size and growth with the adoption of agriculture and the onset of the Holocene, there is evidence for both an increase in body size with maize agriculture in North America, but also an increase in body size variance (Auerbach, 2011). Interestingly, the results of this study show considerable geographic variation in female size and bi-iliac breadth. While trends in human body size tend to generally decrease with the onset of the Holocene, and in some places with the origins of agriculture, there seems to be a considerable increase in variation in human body size within the Holocene.

The results presented by Auerbach thus suggest that pelvic dimensions may vary with the origins of agriculture. A classic study of Mediterranean populations by Angel addressed trends in both stature and pelvic dimensions, though the samples were not from a single location over time (Angel, 1975). Changes in stature are illustrated in Figure 9a, with a substantial decline of ~12 cm from the late Paleolithic, followed by a slow recovery until the 19th century, and then a rapid secular increase in the last century. Figure 9b illustrates the equivalent trend for the pelvic brim index. The pattern of change is much less clear, but broadly, a similar initial decline followed by first slow and then rapid recovery is evident, though with fluctuations along the way which may indicate different physiques of the various populations.

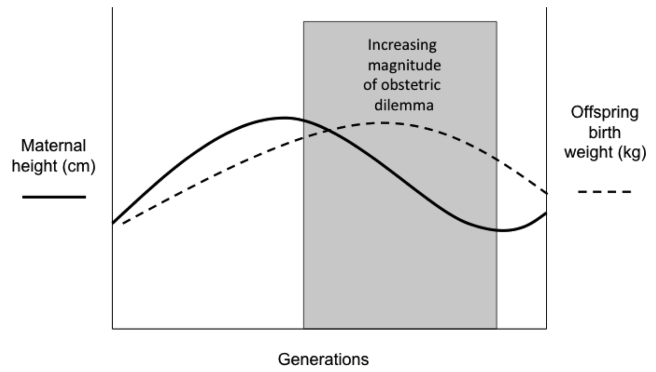
In fact, when stature falls, pelvic dimensions do not necessarily fall in exact proportion (Ruff, 1994). In studies of Japanese across three generations and United States students across two generations, secular trends in height were accompanied by minor decreases in bi-iliac breadth, suggesting an imperfect fit between trends in stature and pelvic dimensions. The protection of pelvic dimensions in Holocene Later Stone Age (LSA) foragers



**Fig. 9.** Secular trends in (a) female stature and (b) female pelvic brim index from the Paleolithic to the present, in Mediterranean populations. Based on data from Angel (1975).

of short stature (Kurki, 2007) suggests long-term adaptation to energy stress rather than climate (Kurki et al., 2008), as the archaeological evidence suggests only minor variation in consistently small body size over a lengthy period (Pfeiffer and Sealy, 2006). Amongst contemporary Khoe-San foragers, a mean birth weight of 3.08 (SD 0.46) kg was recorded in 10 infants (Howell, 1972), while skeletal data from 18 LSA perinates suggested consistency with the contemporary range of infant size (Pfeiffer and Harrington, 2012). A similar scenario may relate to the Inuit, who are likewise of relatively short stature but who do not have narrow pelvic dimensions (Kurki, 2011), though they may also have given birth to much larger neonates than those of the Khoe-San (Murphy et al., 1996; Indorf et al., 2001).

Although relevant evidence remains very limited, it suggests that small size achieved over a long time period need not necessarily increase the disparity between maternal and neonatal dimensions. The situation may, however, be very different for populations responding over shorter timescales to nutritional inadequacy, particularly if the adaptation is achieved through phenotypic plasticity rather than genetic change. Poor growth deriving from inadequate nutrition (potentially mineral as well as energy deficiencies) may lead to a flatter pelvis. This downward trend in pelvic size would thus increase the clash with the neonatal brain and shoulder dimensions, although over time a complementary downward

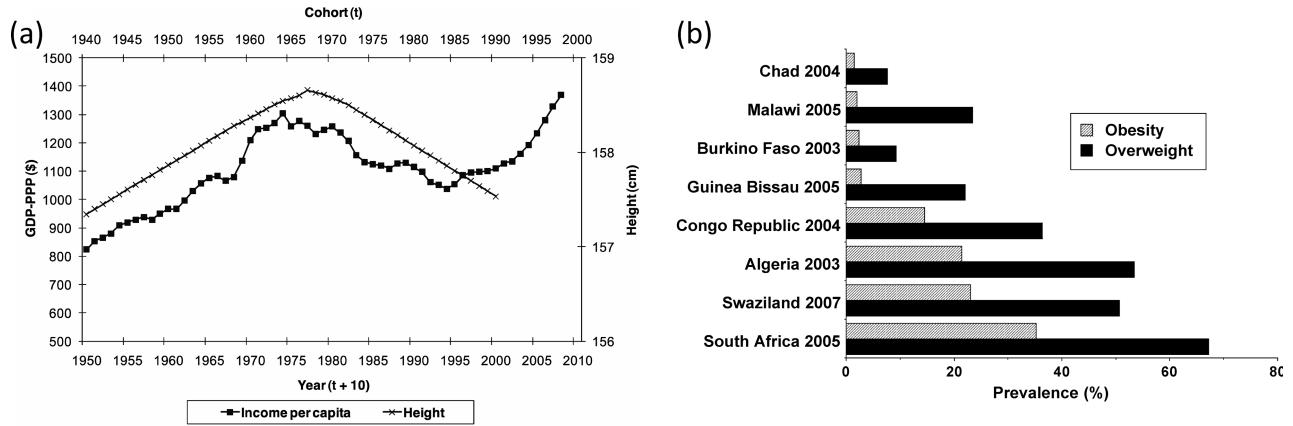


**Fig. 10.** Schematic diagram illustrating the time lag by which birth weight is assumed to track secular trends in female stature. The obstetric dilemma is assumed to increase in magnitude as declines in maternal stature precede matching declines in birth weight. Adapted with permission from Wells, *J Life Sci*, 2009, 1, 115-120, ©Korean Society of Life Science).

trend in neonatal body proportions could also occur. If upward secular trends in birth weight proceed faster than similar trends in pelvic restructuring, then for a certain period, average birth weight might exceed average pelvic proportions. In Figure 10, we illustrate schematically the effect of a time lag between intergenerational trends in maternal height, and intergenerational trends in neonatal weight (Wells, 2009). Sudden decreases in maternal stature may not immediately be followed by matching reductions in neonatal size, resulting in temporary elevation in the magnitude of the obstetric dilemma. Resolution of this disparity might require genetic adaptation of offspring size in response to maternal size, or a slow-damped response through intergenerational plasticity.

Recent nutritional experience in some global regions, driven by antagonistic economic trends, may be demonstrating this scenario. In sub-Saharan Africa, for example, maternal height rose in the mid-20th century, reflecting improvements in health and nutrition, but then declined in all social strata in the last quarter of the century following structural adjustment programs implemented in the 1970s (Garenne, 2011). This decline in height is closely linked with declines in per capita income (Fig. 11a), thereby supporting the notion that depression in living standards was responsible (Garenne, 2011). By the end of the century, however, many African countries were showing the emergence of obesity (Fig. 11b), a trend which has been increasing in the new century (Popkin et al., 2012), and which again can be attributed to adverse economic influences (Wells, 2012b; Wells et al., 2012). As discussed for India below, any increase in obesity will exacerbate the obstetric dilemma, and this effect will only be exaggerated if height is falling at the same time. Macrosomic offspring are increasing in prevalence in some African populations (Abena Obama et al., 1995; Kamanu et al., 2009; Geidam et al., 2009), although data remain sparse. This scenario therefore seemingly replicates in its overall pattern, though not necessarily in its timescale or magnitude, that discussed for the emergence of agriculture, but with globalized market forces as the underlying driving force.

Maternal nutritional deficiencies may contribute more drastically to cephalo-pelvic disproportion. For example, rickets is widely established as a risk factor for poor pelvic development (Skippen, 2009). In contemporary



**Fig. 11.** (a) Trends in maternal height in sub-Saharan Africa from 1950, illustrating a decline in height after the late 1960s, closely tracking declines in per capita income. Reproduced with permission from Garenne, Trends in nutritional status in adult women in sub-Saharan Africa. DHS Comparative Reports 27, 2011, ©Calverton, MD: ICF Macro. (b) Data on female overweight (BMI >25 kg/m<sup>2</sup>) and obesity (BMI >30 kg/m<sup>2</sup>) from a selection of African countries in the late 20th or early 21st century, showing that whilst excess weight remains rare in some countries, it has become the norm in others. These antagonistic trends may exacerbate the obstetric dilemma. Data from the WHO infobase (available at: <https://apps.who.int/infobase/>) accessed 21st June 2012.

populations, vitamin D deficiency is common in many populations lacking adequate diet or exposure to sunlight, and has been associated with increased risk of caesarean delivery (Merewood et al., 2009). No such association of vitamin D deficiency and caesarean risk was observed in a Pakistani study, however, in this population almost all women (83 of 117) were vitamin D deficient (Brunvand et al., 1998). Populations in northern latitudes, and therefore exposed to seasonal fluctuations in the opportunity for endogenous vitamin D synthesis, may have increased risk of rickets. Infants born in winter may experience constraint on early pelvic growth, with long-term effects on the risk of cephalo-pelvic disproportion (Strickland, 1993). Such a scenario has been suggested for the high, and seasonally variant, rates of maternal mortality in Mongolia, whereby seasonal fluctuations in energy supply increase birth weights, in a population characterized by high prevalences of short stature and rickets (Strickland, 1993). This cephalo-pelvic clash is therefore driven by seasonal effects in successive generations.

### THE TRANSITION TO AGRICULTURE AND MORTALITY

While the implications for the origins of agriculture are regionally variable, the transition is associated with a general deterioration in human health in many regions worldwide (Cohen, 1989; Larsen, 1995; Steckel and Rose, 2002; Larsen et al., 2008). The demographic consequences of the transition to agriculture are fairly obvious in the dramatic population expansion which followed the adoption of agricultural subsistence as a result of increasing fertility (Bocquet-Appel, 2002a,b). Despite the tremendous interest in the demographic implications of the origins of agriculture, we know little about variation in perinatal and infant mortality between foraging and agricultural populations. In Table 4, we provide a preliminary exploration of mortality profiles from skeletal samples, including five hunter-gatherer groups ranging from low to high latitudes, and three agricultural populations, derived from the literature. Hunter-gatherers include the North African Iberomarusian skeletons from

Afalou and Taforalt; Near Eastern Natufian skeletons from the sites of Rakefet, Nahal-Oren, Hayonim and Ain Mallaha; Archaic foragers from the sites of Hind, Sartori and Zimmer in Ontario, Canada; mid-Holocene foragers from the sites of Lokomotif, Raisovet, and Shamanka II in the Baikal region of Siberia; and the Sadlermiut from Native Point, in Nunavut, Canada. Data taken from the literature represent the following agricultural groups: the Arikara of the Northern Plains of the USA (Owsley and Bradtmiller, 1983); the site of Tlajinga 33, from Teotihuacan, Mexico (Storey, 1986); and the site of Kellis 2 from the Roman Period of the Dakhleh Oasis, Egypt (Tocheri et al., 2005).

Age estimates of subadults were based upon dental formation and eruption where possible, or estimates of age based upon long bone length. Ages were categorized into perinatal (either late-term fetal or neonate, up to 1 month), infant (2 months up to 1 year of age), 1 to 4, 5 to 9, 10 to 14 years, and adult. These age categories were used to allow maximal comparability of data between the new forager data and three published archaeological samples from the literature. Our primary interest is to investigate variation in perinatal mortality between foragers and agriculturalists. While it is impossible to determine whether perinatal mortality is the direct consequence of obstetric complications in archaeological series, it is reasonable to expect that there is a strong correlation between these parameters, such that significant differences in obstetric complications should be reflected in the proportion of perinatal skeletons within a series. It is often considered that sub-adult remains may be under-represented in skeletal assemblages due to poor preservation or cultural factors, however, the presence of perinatal and infant remains in each of the represented skeletal series suggests that cultural or taphonomic factors may have minimal impact on our demographic data in these cases. The percentage of individuals represented within each age group was calculated relative to the total number of individuals, to provide an indication of the proportion of individuals in each age category. Percentages were not calculated for the Sadlermiut sample, as the total number of the sample represents an estimate. Demographic data presented

TABLE 4. *Paleodemography of hunter-gatherer and agricultural populations, with estimates of perinatal and infant mortality<sup>a</sup>*

| Subsistence     | Population                                   | Perinatal    | 2 months to 0.9 yr | 1-4         | 5-9        | 10-14     | 15-19     | Adult       | Total  | Reference                    |
|-----------------|--|--------------|--------------------|-------------|------------|-----------|-----------|-------------|--------|------------------------------|
| Hunter-gatherer | Iberomarusian, North Africa                  | 2 (2.0%)     | 4 (4.1%)           | 7 (7.1%)    | 4 (4.1%)   | 0 (0%)    | 3 (3.1%)  | 78 (79.5%)  | 98     | This study                   |
| Hunter-gatherer | Natufian, Levant                             | 4 (6.5%)     | 0 (0%)             | 4 (6.5%)    | 5 (8.1%)   | 2 (3.2%)  | 5 (8.1%)  | 39 (62.9%)  | 62     | This study                   |
| Hunter-gatherer | Archaic, Great Lakes Ontario, Canada         | 1 (4.2%)     | 0 (0%)             | 1 (4.2%)    | 1 (4.2%)   | 3 (12.5%) | 0 (0%)    | 18 (75%)    | 24     | This study                   |
| Hunter-gatherer | Kitoi, mid-Holocene, Siberia                 | 0 (0%)       | 3 (3.4%)           | 9 (10.3%)   | 12 (13.8%) | 5 (5.7%)  | 5 (5.7%)  | 53 (60.9%)  | 87     | This study                   |
| Hunter-gatherer | Sadlermiut, Nunavut, Canada                  | 4 (4.2%)     | 18 (14.8%)         | 7 (10.3%)   | 4 (4.1%)   | 8 (4.2%)  | 1 (1.5%)  | —           | ca 183 | This study                   |
| Agricultural    | Arikara, Northern Plains, USA                | 468* (31.5%) | —                  | 235 (15.8%) | 123 (8.3%) | 62 (4.2%) | 79 (5.3%) | 520 (34.9%) | 1487   | Owsley and Bradtmiller, 1983 |
| Agricultural    | Tlajinga 33, Teotihuacan, Mexico             | 52 (31.3%)   | 7 (4.2%)           | 15 (9.0%)   | 10 (6.0%)  | 9 (5.4%)  | 9 (5.4%)  | 64 (38.6%)  | 166    | Storey, 1986                 |
| Agricultural    | Kellis 2, Roman Period, Dakhleh Oasis, Egypt | 82 (18.2%)   | 67 (14.8%)         | —           | —          | —         | —         | —           | 450    | Tocheri et al, 2005          |

<sup>a</sup> Hunter-gatherer assemblages chosen as those representing entire series of excavated skeletons from one or several sites of the same period, age estimated using dental formation and eruption where possible, otherwise from long-bone lengths, except for the Sadlermiut, which represent a subset of the entire population, a catastrophic death assemblage, here the subadults represent the subset of the total which were complete enough for age assessment and measurement of postcrania. As the total number of individuals is approximate, percentages of the total assemblage have not been calculated. Kitoi data represents individuals from the cemeteries of Shamanka II, Rakefet, and Lokomotiv, Natufian sites include Rakefet, Nahal-Oren, Hayonim, Ain Mallaha, The data from Tlajinga 33, Teotihuacan (Storey, 1986) were re-coded from slightly different age categories, Kellis 2 data represents estimates from Tocheri et al. (2005) based on the number of individuals presented in tables of gestational age estimates and the statement "Of the 450 burials excavated to date, 34% ( $n = 149$ ) fall in the less than 1-year-old category, and of these well over 50% are estimated to be fetuses or less than 1-month-old postpartum" (p 334), to provide a total infant and perinatal  $N$ .  
\*ages 0-0.9.

for the Arikara did not subdivide the infant category into perinatal remains, so only the total number of infants in the assemblage is presented here for comparison.

Amongst the forager populations, all have low frequencies of perinatal and infant skeletons, ranging from 0 to 6.5% of the skeletal assemblage, and combined perinatal and infant mortality does not exceed 6.5%. In contrast, perinatal and infant mortality among the three agricultural groups ranges from 31.3% (Arikara) to 35.5% (Teotihuacan). High levels of infant mortality amongst agricultural populations are not unexpected, and may be related to higher levels of infectious disease. The proportions of perinatal skeletons in the Teotihuacan (31.3%) and Dakhleh Oasis (18.2%, minimal estimate, based on estimates provided in the text of the article) are of particular interest, in that they might suggest high levels of obstetric mortality. Amongst the foragers, the Sadlermiut population is also of note, as there are 22 perinatal and infant skeletons, representing over 10% of the total assemblage. Here, only 4 of 22 were identified as perinatal, while 18 were determined to be over two months of age. This high rate of infant, but not perinatal, mortality might be related to infectious disease contracted following contact with European sailors (Merbs, 2004). While this leads to a higher overall frequency of infant mortality, which is more consistent with levels found amongst the agricultural populations, the level of perinatal mortality amongst the Sadlermiut is not inconsistent with other foragers. This pattern of low perinatal mortality may be the result of relatively spacious obstetric dimensions among this population (Kurki, 2011).

Overall, the pattern of perinatal and infant mortality found amongst these populations suggests that both perinatal and infant mortality are much higher among early agricultural than foraging populations. While the high levels of infant mortality amongst agriculturalists are most likely explained by higher frequencies of infectious disease, the higher levels of perinatal mortality might reflect an increased risk of obstetric complications during birth, a factor which is invoked in each of the publications from which the data were derived (Owsley and Bradtmiller, 1983; Storey, 1986; Tocheri et al., 2005). Further work on the obstetric dilemma in foragers is therefore needed.

### POTENTIAL NUTRITIONAL EFFECTS OF AGRICULTURAL DIETS

Complementary to effects on maternal height, the emergence of agriculture may have altered fetal growth patterns through dietary changes (Liston, 2003). Reconstructing Paleolithic diets is notoriously difficult (Milton, 2000), as ancestral foragers inhabited diverse ecosystems and must inevitably have adapted to the resources available. To some extent, this adaptation may have been aided by two ways of homogenizing diverse raw foodstuffs into a more fundamental diet. The consumption of each of meat and cooked vegetable foods reduces the level of physiological adaptation required by the gut. Nevertheless, the macro-nutrient content of contemporary forager diets still shows substantial variability (Milton, 2000), and this can be assumed to have been true throughout the evolutionary history of *Homo*, due to climate trends and ecosystem change (Potts, 1996). With these caveats, there are several possible ways

whereby agricultural diets may have impacted the obstetric dilemma.

Protein content is assumed to have been high in pre-agricultural populations, whereas carbohydrate content is assumed to have been low (McMichael, 2001; Colagiuri and Brand, 2002). Regardless of the actual meat content of the diet, the glycemic load is likely to have been substantially lower than that of modern agricultural diets (Milton, 2000). The emphasis on grains following the emergence of agriculture suggests that a substantial upward shift in glycemic load was initiated at this time (Eaton and Eaton, 2000; Cordain et al., 2005; Jönsson et al., 2009).

Such changes in dietary glycemic load may have affected both maternal stature and fetal growth. Dietary protein has been associated with growth in childhood (Rolland-Cachera et al., 1995; Stein et al., 2003; Hoppe et al., 2004a,b) and stature in adulthood (Galvin, 1992). A shift from high-protein to high-carbohydrate diets, as agriculture involved, may therefore have been a major contributing factor to declines in stature described above. The same dietary shift may have affected fetal weight gain. Most wild foods are high in bulk and fiber, and hence release energy into the bloodstream relatively slowly (Milton, 2000). On the one hand, higher-glycemic diets have been associated with increased birth weight (Scholl et al., 2004; Moses et al., 2006), and maternal glucose has been further associated with fetal adiposity (Catalano et al., 2003; Hill et al., 2005). On the other hand, a number of studies have associated high-protein diets with reduced birth weight (Campbell et al., 1996; Godfrey et al., 1996; Sloan et al., 2001; Kramer and Kakuma, 2003; Andreasyan et al., 2007), although other studies were not consistent (Mathews et al., 1999; Moore et al., 2004). Specific foods also have been investigated: associations between maternal seafood consumption and birth weight are inconsistent (Mendez et al., 2010), whereas maternal milk consumption during pregnancy was positively associated with birth weight and neonatal head circumference (Olsen et al., 2007).

Associations of maternal dietary intake during pregnancy with neonatal head circumference are not necessarily consistent with those for birth weight. In a small randomized trial, the offspring of mothers receiving a low glycemic index diet had similar birth weight but increased head circumference relative to those receiving a low-fat diet (Rhodes et al., 2010).

Whilst the evidence remains preliminary, these studies collectively offer support for the hypothesis that the emergence of agricultural diets, with higher glycemic load and lower protein content than typical forager diet, could have impacted each of maternal size and neonatal mass and brain size, and may therefore have exacerbated the obstetric dilemma. In very recent periods, reduced levels of physical exercise may have further exacerbated the obstetric dilemma by altering maternal glycemic control (Liston, 2003). Such effects may have been further influenced by related stresses such as population density, and an increasing infectious disease burden.

### EFFECTS OF INFECTIOUS DISEASE

The onset of agriculture was associated not only with dietary change, but also with an increased prevalence in a number of infectious diseases, many of them crossing the species barrier from newly domesticated animals and further favored by increasing population density and

trade (Cohen, 1989; Diamond, 1998). Infectious disease exerts a particular burden in contemporary populations in early life. Kuzawa has therefore argued that selection favored high levels of adiposity in our species, in order to buffer the obligatory energy requirements of the large infant brain during this vulnerable period (Kuzawa, 1998). Whilst an increased glycemic load from the agricultural diet may have made such fat stores more readily attainable by the offspring, fat as a low-density tissue would increase neonatal body volume, and potentially make delivery more difficult, as discussed above in relation to macrosomic offspring.

Most data on neonatal adiposity derive from western industrialized populations with relatively high birth weight (~3.5 kg). In non-western populations, birth weight approximates closer to ~3 kg, though substantial variability between populations also remains. In a recent study of body composition in Ethiopian neonates, mean birth weight was 3.0 kg, and mean % fat was 8% (Andersen et al., 2011). These data therefore indicate a lower level of adiposity in a non-western population, hence further data are required in order to explore variability in neonatal body composition in more detail. In relative terms, Indian infants preserve adiposity despite having low average birth weight (Yajnik et al., 2003), and this increased adiposity may contribute to the obstetric dilemma. Although in the early 20th century Pan (1929) suggested that the small size of both maternal pelvic dimensions and neonatal dimensions need not imply difficult labor in the Indian population, increases in maternal BMI and glucose intolerance, driven by urbanization (Subramanian et al., 2010), may now be increasing the magnitude of the obstetric dilemma. Consistent with this hypothesis, recent studies in India have shown that maternal gestational diabetes is associated with larger offspring and an increased risk of cephalo-pelvic disproportion, shoulder dystocia and caesarian delivery (Saxena et al., 2011; Sharma et al., 2011).

### RESOLUTIONS TO THE OBSTETRIC DILEMMA

The notion that long-term trends in nutrition may impact the magnitude of the obstetric dilemma through phenotypic plasticity prompts consideration of what beneficial effects might be achieved through nutritional intervention within the life-course. Brabin and Brabin (1992) initially speculated that adolescent girls might be especially vulnerable to cephalo-pelvic disproportion, due to their becoming pregnant before the growth process may have been completed. This scenario might be exacerbated by the fact that growth of the pelvis lags behind growth in stature (Moerman, 1982). Brabin and colleagues therefore questioned whether nutritional supplementation might exacerbate the problem in this age group. In subsequent work, however, they and others found that adolescents are if anything less likely than older age groups to experience cephalo-pelvic disproportion, due to their tendency to give birth to offspring of lower average birth weight (Larsson and Svanberg, 1983; Brabin et al., 2002; Borja and Adair, 2003; Nkwabong and Fomulu, 2009). This low birth weight is further due to continued growth in the adolescents during pregnancy, implying a trade-off between maternal and fetal energy allocation (Harrison et al., 1895a,b; Scholl et al., 1990). In general, birth weight increases with age and parity, and cephalo-pelvic disproportion is therefore commoner in older women

(Brabin et al., 2002). Thus, nutritional supplementation during adolescence is more likely to promote both maternal and offspring health without increasing the risk of cephalo-pelvic disproportion (Brabin et al., 2002), and taller height may prove protective against the obstetric dilemma both during adolescence and subsequently (Harrison et al., 1988).

In the absence of actual changes in body dimensions, other means of adaptation may be required to achieve successful birth. Extensive ethnographic research indicates that in nonindustrialized populations, the most common positions for giving birth were kneeling, squatting or sitting (Engelmann, 1882; Jarcho, 1934; Naroll et al., 1961; Rosenberg and Trevathan, 2002). Squatting has been shown to increase the size of the birth canal by up to 28% (Russell, 1969, 1982; Liu et al., 1989; Michel et al., 2002), and the widespread utilization of the supine position in industrialized populations appears not to derive from maternal preference (De Jonge et al., 2004) but rather due to its promotion by clinicians over the last two centuries, as they increasingly “medicalized” an event previously considered as normal and natural (Wertz and Wertz, 1977; Dundes, 1987; Walrath, 2003). Thus, the spread of a particular fashion for delivery position may be another sociocultural factor that has exacerbated the magnitude of the obstetric dilemma in recent decades, and increasing interest in challenging many aspects of the institutionalization of childbirth (Marland and Rafferty, 1997) may therefore have implications for management of the obstetric dilemma.

Women in Asian populations have been observed to restrict their dietary intake in the last trimester of pregnancy, specifically to avoid large babies (Brems and Berg, 1989; Choudhry, 1997; Christian et al., 2006). Unlike in Holocene southern African foragers, where short stature was not associated with reduced obstetric proportions (Kurki, 2007), the short stature of south Asian women has been associated with significantly reduced pelvic inlet and outlet dimensions, though also with small neonates (Pan, 1929). The combination of smaller uterine volume and eating down during pregnancy in south Asian populations is associated with low birth weight “thin-fat” neonates, in which indices of lean mass are substantially reduced compared to European neonates, whereas head girth and adiposity are relatively protected (Yajnik et al., 2003; van Steijn et al., 2009). The “nutrition transition” might therefore increase the magnitude of the obstetric dilemma in the Indian population, if offspring neonatal dimensions change faster than maternal pelvic dimensions.

In recent times, the development of expertise for caesarean section represents a technical solution to the dilemma. Early such operations were most likely intended to save an offspring when the mother had already died (Sewell, 1993). While the practice has only been widespread in the last century, it was observed in Uganda by a British traveller, R.W. Felkin, in 1879: “The healer used banana wine to semi-intoxicate the woman and to cleanse his hands and her abdomen before surgery. He used a midline incision and applied cautery to minimize hemorrhaging. He massaged the uterus to make it contract but did not suture it; the abdominal wound was pinned with iron needles and dressed with a paste prepared from roots. The patient recovered well, and Felkin concluded that this technique was well-developed and had clearly been employed for a long time” (Sewell, 1993). The development of such expertise itself indicates



the long-term existence of obstetric complications in this population.

Very little is known about maternal reproductive mortality, birth practices, or neonatal size, shape and adiposity in nonagricultural foraging populations, who typically consume a much higher protein diet than agricultural populations, with complementary lower glycemic load. The majority of foraging populations today occupy relatively marginal environments, and tend toward smaller body size than that found among typical late Pleistocene hunter-gatherers or most agricultural populations. This might either relieve the obstetric dilemma, as observed by Kurki (2007), or exacerbate it as we have suggested in this review was the case for agricultural populations.

For past foraging populations, the scenario remains relatively unexplored, though the data in Table 4 are provocative. In her account of a single !Kung woman, Shostak (1981) reported that her informant Nisa described her mother giving birth unaided. Such a practice of solitary delivery has also been reported from the Bariba, an agricultural population from Benin (Sargent, 1982). There is inadequate evidence to address this issue with confidence at present, yet if preagricultural populations did experience less of an obstetric dilemma than is typical of contemporary agricultural populations, the evolutionary significance of birth attendants, a scenario not unknown in other primate species (Trevathan, 2011), might also benefit from reappraisal.

## SUMMARY

We suggest that whatever ecological pressures generated the complex rotational birth process through human evolutionary history, the contemporary obstetric dilemma manifesting as maternal and perinatal mortality cannot simply be considered to derive from a long-standing and routine “clash” of offspring encephalization and maternal bipedalism. The risks of delivery derive in part from other factors, which we have shown are variable within and between populations. Hence, the magnitude of the dilemma, and the risks of delivery, appear to have waxed and waned over both longer and shorter timescales, as a variety of different ecological stresses rose and fell in importance.

With regard to contemporary *Homo sapiens*, we suggest that the magnitude of the dilemma is sensitive to several ecological pressures including the thermal environment, dietary energy availability and glycemic load, and infectious disease burden. In turn, we suggest that these ecological stresses may each have become exacerbated during the transition to agriculture, acting on both maternal and fetal phenotype, such that the obstetric dilemma may have been worse in the last few thousand years than was the case for Pleistocene *Homo*. Our analysis highlights potential differences in the capacity for maternal versus offspring plasticity, suggesting that variability in the magnitude of the obstetric dilemma fluctuates across multigenerational timescales, with phenotype of the two parties potentially changing at different rates. Whilst some populations appear to have evolved small body size without exacerbating the dilemma, others appear to have failed to adapt in this way.

Relevant ecological trends appear to continue in contemporary populations, where maternal obesity and especially high-glycemic index diets (e.g. those high in sucrose or high-fructose corn syrup) may be introducing a new complicating factor, increasing the risk of macro-

somic neonates. We have highlighted decreasing maternal height and increasing maternal obesity in sub-Saharan Africa as evidence of such trends, indicating that our perspective is not only relevant to understanding human evolution, but may also have significant public health implications.

Public health efforts cannot expect to make rapid progress in resolving a problem that emerges out of longer-term adaptive processes. Public health success is likely to emerge through long-term trends in stature, along with diets lower in glycemic index and more successful prevention of obesity. One provocative perspective is that the advent of safe caesarean delivery has removed a key constraint on the upper boundary of neonatal size, which could potentially induce genetic change favoring higher birth weight, in turn making possible upwards trends in brain size (Walsh, 2008). Such a perspective ignores the transgenerational transmission of stature, pelvic dimensions, head girth, and body mass. Given that the detrimental effects of agriculture emerged over multiple generations, resolution of the obstetric dilemma is likely to require a similar long-term approach.

## ACKNOWLEDGMENTS

The authors are very grateful to Yerkes, Southwest and Oregon National Primate Research Centers for making available unpublished primate data. Unpublished data on maternal and offspring anthropometry was provided by Henrik Friis and colleagues from the University of Copenhagen, Denmark, and Tsinuel Girma and colleagues from the University of Jimma, Ethiopia. The authors are very grateful to Tim Cole of UCL Institute of Child Health for statistical advice, and to the editor and three reviewers for valuable constructive criticisms.

## LITERATURE CITED

- Abena Obama MT, Shasha VW, Fodjo J, Bonongkaho F, Mbede J, Kamdom Moyo J. 1995. Foetal macrosomia in Cameroon: prevalence, risk factors and complications. *West Afr J Med* 14:249–254.
- Abitbol MM. 1987. Obstetrics and posture in pelvic anatomy. *J Hum Evol* 16:243–255.
- Abitbol MM. 1995. Reconstruction of the STS 14 (*Australopithecus africanus*) pelvis. *Am J Phys Anthropol* 96:143–158.
- Abitbol MM. 1996. Birth and human evolution: anatomical and obstetrical mechanics in primates. Westport, CT: Bergin & Garvey.
- AbouZahr C. 1998. Prolonged and obstructed labour. In: Murray CJ, Lopez AD, editors. *Health dimensions of sex and reproduction Global burden of disease and injury series*, Vol.3. Cambridge, MA: Harvard University Press.
- Adadevoh SW, Hobbs C, Elkins TE. 1989. The relation of the true conjugate to maternal height and obstetric performance in Ghanaians. *Int J Gynaecol Obstet* 28:243–251.
- Aiello LC, Wells JC. 2002. Energetics and the evolution of the genus *Homo*. *Ann Rev Anthropol* 31:323–338.
- Akachi Y, Canning D. 2007. The height of women in Sub-Saharan Africa: the role of health, nutrition, and income in childhood. *Ann Hum Biol* 34:397–410.
- Aldoretta PW, Hay WW Jr. 1995. Metabolic substrates for fetal energy metabolism and growth. *Clin Perinatol* 22:15–36.
- Algovik M, Kivinen K, Peterson H, Westgren M, Kere J. 2010. Genetic evidence of multiple loci in dystocia–difficult labour. *BMC Med Genet* 11:105.
- Allbrook D. 1962. Some problems associated with pelvic form and size in the Ganda of East Africa. *J R Anthropol Inst* 92:102–114.
- Allen JA. 1877. The influence of physical conditions on the genesis of species. *Radic Rev* 1:108–140.

- Andersen GS, Girma T, Wells JC, Kaestel P, Michaelsen KF, Friis H. 2011. Fat and fat-free mass at birth: air displacement plethysmography measurements on 350 Ethiopian newborn. *Pediatr Res* 70:501–506.
- Andreasyan K, Ponsonby AL, Dwyer T, Morley R, Riley M, Dear K, Cochrane J. 2007. Higher maternal dietary protein intake in late pregnancy is associated with a lower infant ponderal index at birth. *Eur J Clin Nutr* 61:498–508.
- Angel JL. 1975. Paleocology, paleodemography and helath. In: Polgar S, editor. *Population, ecology, and social evolution*. The Hague: Moulton Publishers. p 167–190.
- Angel JL. 1978. Pelvic inlet form: a neglected index of nutritional status. *Am J Phys Anthropol* 48:378.
- Arriaza B, Allison M, Gerszten E. 1988. Maternal mortality in pre-Columbian Indians of Arica, Chile. *Am J Phys Anthropol* 77:35–41.
- Auerbach B. 2011. Reaching great heights: changes in indigenous stature, body size and body shape with agricultural intensification in North America. In: Pinhasi R, Stock JT, editors. *Human bioarchaeology of the transition to agriculture*. Chichester: Wiley Blackwell. p 203–233.
- Baird D. 1949. Social factors in obstetrics. *Lancet* 1:1079–1083.
- Barbosa GP, Giffin K, Angulo-Tuesta A, Gama Ade S, Chor D, D'Orsi E, dos Reis AC. 2003. Cesarean sections: who wants them and under what circumstances? *Cad Saude Publica* 19:1611–1620.
- Barton RA, Capellini I. 2011. Maternal investment, life histories, and the costs of brain growth in mammals. *Proc Natl Acad Sci USA* 108:6169–6174.
- Begun DR, Kordos L. 2004. Cranial evidence of the evolution of intelligence in fossil apes. In: Russon AE, Begun DR, editors. *The evolution of thought: evolutionary origins of great ape intelligence*. Cambridge: Cambridge University Press. p 260–279.
- Benjamin SJ, Daniel AB, Kamath A, Ramkumar V. 2012. Anthropometric measurements as predictors of cephalopelvic disproportion: can the diagnostic accuracy be improved? *Acta Obstet Gynecol Scand* 91:122–127.
- Berard J, Dufour P, Vinatier D, Subtil D, Vanderstichele S, Monnier JC, Puech F. 1998. Fetal macrosomia: risk factors and outcome. A study of the outcome concerning 100 cases >4500 g. *Eur J Obstet Gynecol Reprod Biol* 77:51–59.
- Berdnikovs S, Bernstein M, Metzler A, German RZ. 2007. Pelvic growth: ontogeny of size and shape sexual dimorphism in rat pelvis. *J Morphol* 268:12–22.
- Berg-Lekas ML, Hogberg U, Winkvist A. 1998. Familial occurrence of dystocia. *Am J Obstet Gynecol* 179:117–121.
- Berge C, Goularas D. 2010. A new reconstruction of Sts 14 pelvis (*Australopithecus africanus*) from computed tomography and three-dimensional modeling techniques. *J Hum Evol* 58:262–272.
- Berge C, Orban-Segebarth R, Schmid P. 1984. Obstetrical interpretation of the australopithecine pelvic cavity. *J Hum Evol* 13:573–587.
- Bergmann C. 1847. Über die Verhältnisse der wärmeökonomie der Thiere zu ihrer Grösse. *Göttinger Studien* 3:595–708.
- Bernard RM. 1950. Nutrition in relation to the shape and size of the human pelvis. *Br J Nutr* 4:i–ii.
- Betti M, Ceccatelli G, Belcari F, Moscuza F, Cuttano A, Vuerich M, Boldrini A, Ghirri P. 2011. Neonatal outcome in newborns from mothers with endocrinopathies. *Gynecol Endocrinol* 27:248–250.
- Björklund K, Lindgren PG, Bergström S, Ulmsten U. 1997. Sonographic assessment of symphyseal joint distention intra partum. *Acta Obstet Gynecol Scand* 76:227–232.
- Blinkov SM, Glezer II. 1968. *The human brain in figures and tables*. New York: Basic Books.
- Bocquet-Appel J-P. 2002a. Paleoanthropological traces of a Neolithic demographic transition. *Curr Anthropol* 43:637–650.
- Bocquet-Appel J-P. 2002b. Testing the hypothesis of a worldwide Neolithic demographic transition: corroborations from American cemeteries. *Curr Anthropol* 47:341–365.
- Bonmatí A, Gómez-Olivencia A, Arsuaga JL, Carretero JM, García A, Martínez I, Lorenzo C, Bermúdez de Castro JM, Carbonell E. 2010. Middle Pleistocene lower back and pelvis from an aged human individual from the Sima de los Huesos site, Spain. *Proc Natl Acad Sci USA* 107:18386–18391.
- Borja JB, Adair LS. 2003. Assessing the net effect of young maternal age on birthweight. *Am J Hum Biol* 15:733–740.
- Bowden D, Winter P, Ploog D. 1967. Pregnancy and delivery behavior in the squirrel monkey (*Saimiri sciureus*) and other primates. *Folia Primatol* 5:1–42.
- Brabin L, Brabin BJ. 1992. The cost of successful adolescent growth and development in girls in relation to iron and vitamin A status. *Am J Clin Nutr* 55:955–958.
- Brabin L, Verhoeff F, Brabin BJ. 2002. Maternal height, birth-weight and cephalo pelvic disproportion in urban Nigeria and rural Malawi. *Acta Obstet Gynecol Scand* 81:502–507.
- Brandt EM, Mitchell G. 1971. Parturition in primates. In: Rosenblum LA, editor. *Primate behavior, developments in field and laboratory research*, Vol.2. New York: Academic Press. p 177–223.
- Brandt EM, Mitchell, G. 1973. Labor and delivery behavior in rhesus monkeys (*Macaca mulatta*). *Am J Phys Anthropol* 38:519–522.
- Brans YW, Shannon DL, Hunter MA. 1983. Maternal diabetes and neonatal macrosomia. II. Neonatal anthropometric measurements. *Early Hum Dev* 8:297–305.
- Brems S, Berg A. 1989. Eating down during pregnancy: nutrition, obstetric and cultural considerations in the third world. In: ACC/SCN. Washington, DC: World Bank.
- Brunvand L, Shah SS, Bergstrom S, Haug E. 1998. Vitamin D deficiency in pregnancy is not associated with obstructed labor. A study among Pakistani women in Karachi. *Acta Obstet Gynecol Scand* 77:303–306.
- Butte NF, Hopkinson JM, Mehta N, Moon JK, Smith EO. 1999. Adjustments in energy expenditure and substrate utilization during late pregnancy and lactation. *Am J Clin Nutr* 69:299–307.
- Caldwell WE, Moloy HC. 1933. Anatomical variations in the female pelvis and their effect in labor with a suggested classification. *Am J Obstet Gynecol* 26:479–505.
- Campbell DM, Hall MH, Barker DJ, Cross J, Shiell AW, Godfrey KM. 1996. Diet in pregnancy and the offspring's blood pressure 40 years later. *Br J Obstet Gynaecol* 103:273–280.
- Capellini I, Venditti C, Barton RA. 2011. Placentation and maternal investment in mammals. *Am Nat* 177:86–98.
- Catalano PM, Thomas A, Huston-Presley L, Amini SB. 2003. Increased fetal adiposity: a very sensitive marker of abnormal in utero development. *Am J Obstet Gynecol* 189:1698–1704.
- Chapman A, Hall LS, Bennett MB. 1994. Sexual dimorphism in the pelvic girdle of Australian flying foxes. *Austr J Zool* 42:261–265.
- Chen HY, Chen YP, Wei PY. 1956. A roentgenological study of the Chinese female pelvis. *Am J Obstet Gynecol* 72:1015–1020.
- Choudhry UK. 1997. Traditional practices of women from India: pregnancy, childbirth, and newborn care. *J Obstet Gynecol Neonatal Nurs* 26:533–539.
- Christian P, Srihari SB, Thorne-Lyman A, Khatri SK, LeClerq SC, Shrestha SR. 2006. Eating down in pregnancy: exploring food-related beliefs and practices of pregnancy in rural Nepal. *Ecol Food Nutr* 45:253–278.
- Cloete SW, Scholtz AJ, ten Hoop JM, Lombard PJ, Franken MC. 1998. Ease of birth relation to pelvic dimensions, litter weight and conformation of sheep. *Small Ruminant Res* 31:51–60.
- Cnattingius R, Cnattingius S, Notzon FC. 1998. Obstacles to reducing cesarean rates in a low-cesarean setting: the effect of maternal age, height, and weight. *Obstet Gynecol* 92:501–506.
- Cohen MN. 1989. *Health and the rise of civilisation*. New Haven: Yale University Press.
- Colagiuri S, Brand MJ. 2002. The 'carnivore connection'—evolutionary aspects of insulin resistance. *Eur J Clin Nutr* 56 (Suppl 1):S30–S35.
- Cole TJ. 2000a. Sympercents: symmetric percentage differences on the 100 log(e) scale simplify the presentation of log transformed data. *Stat Med* 19:3109–3125.
- Cole TJ. 2000b. Secular trends in growth. *Proc Nutr Soc* 59:317–324.

- Cordain L, Eaton SB, Sebastian A, Mann N, Lindeberg S, Watkins BA, O'Keefe JH, Brand-Miller J. 2005. Origins and evolution of the Western diet: health implications for the 21st century. *Am J Clin Nutr* 81:341–354.
- Cox M, Scott A. 1992. Evaluation of the obstetric significance of some pelvic characters in an 18th century British sample of known parity status. *Am J Phys Anthropol* 89:431–440.
- Crelin ES. 1969. Interpubic ligament: elasticity in pregnant free-tailed bat. *Science* 164:81–82.
- Crelin ES, Newton EV. 1969. The pelvis of the free-tailed bat: sexual dimorphism and pregnancy changes. *Anat Rec* 164:349–357.
- Crespi B. 2011. The evolutionary biology of child health. *Proc Biol Sci* 278:1441–1449.
- Crognier E. 1981. Climate and anthropometric variations in Europe and the Mediterranean area. *Ann Hum Biol* 8:99–107.
- Deacon T. 1997. The symbolic species: the co-evolution of language and the human brain. London: Allen Lane (Penguin Press).
- De Jonge A, Teunissen TA, Lagro-Janssen AL. 2004. Supine position compared to other positions during the second stage of labor: a meta-analytic review. *J Psychosom Obstet Gynaecol* 25:35–45.
- DeSilva JM. 2011. A shift toward birthing relatively large infants early in human evolution. *Proc Natl Acad Sci U S A* 108:1022–1027.
- DeSilva JM, Lesnik JJ. 2006. Chimpanzee neonatal brain size: Implications for brain growth in *Homo erectus*. *J Hum Evol* 51:207–212.
- DeSilva JM, Lesnik JJ. 2008. Brain size at birth throughout human evolution: a new method for estimating neonatal brain size in hominins. *J Hum Evol* 55:1064–1074.
- Diamond J. 1998. *Guns, germs and steel*. London: Vintage.
- Diamond-Smith N, Potts M. 2011. A woman cannot die from a pregnancy she does not have. *Int Perspect Sex Reprod Health* 37:155–158.
- Dick JL. 1922. *Rickets: a study of economic conditions and their effects on the health of the nation*. London: William Heinemann.
- Drews B, Harmann LM, Beehler LL, Bell B, Drews RF, Hildebrandt TB. 2011. Ultrasonographic monitoring of fetal development in unrestrained bonobos (*Pan paniscus*) at the Milwaukee County Zoo. *Zoo Biol* 30:241–253.
- Dundas L. 1987. The evolution of maternal birthing position. *Am J Publ Health* 77:636–641.
- Dunmire WW. 1955. Sex dimorphism in the pelvis of rodents. *J Mammal* 36:356–361.
- Elliot MG, Crespi BJ. 2008. Placental invasiveness and brain-body allometry in eutherian mammals. *J Evol Biol* 21:1763–1778.
- Elder JH, Yerkes RM. 1936. Chimpanzee births in captivity: a typical case history and report of sixteen births. *Proc R Soc Lond B Biol Sci* 120:409–421.
- Eaton SB, Eaton SB III. 2000. Paleolithic vs. modern diets—selected pathophysiological implications. *Eur J Nutr* 39:67–70.
- Elliot-Smith GB, Wood Jones F. 1910. *The archaeological survey of Nubia report for 1907-1908*, volume II: report on human remains. Cairo: National Printing Department.
- Ellison PT. 2001. *On fertile ground*. Cambridge: Harvard University Press.
- Ellison PT. 2008. Energetics, reproductive ecology, and human evolution. *PaleoAnthropol* 172–200.
- Engelmann GJ. 1882. *Labour among primitive peoples*. St Louis: JH Chambers.
- English J, Alcoair K. 1995. Normal pelvic dimensions for Saudi Arabian women in Tabuk obtained by computed tomography pelvimetry. *Ann Saudi Med* 15:236–239.
- Epstein HT. 1973. Possible metabolic constraints on human brain weight at birth. *Am J Phys Anthropol* 39:135–136.
- Evagelidou EN, Kiortsis DN, Bairaktari ET, Giapros VI, Cholevas VK, Tzallas CS, Andronikou SK. 2006. Lipid profile, glucose homeostasis, blood pressure, and obesity-anthropometric markers in macrosomic offspring of nondiabetic mothers. *Diabetes Care* 29:1197–1201.
- Ezegwui HU, Ikeako LC, Egbuji C. 2011. Fetal macrosomia: obstetric outcome of 311 cases in UNTH, Enugu, Nigeria. *Niger J Clin Pract* 14:322–326.
- Falhammar H, Davis B, Bond D, Sinha AK. 2010. Maternal and neonatal outcomes in the Torres Strait Islands with a sixfold increase in type 2 diabetes in pregnancy over six years. *Aust NZ J Obstet Gynaecol* 50:120–126.
- Falk D, Zollikofer CPE, Morimoto N, Ponce de León MS. 2012. Metopic suture of Taung (*Australopithecus africanus*) and its implications for hominin brain evolution. *Proc Natl Acad Sci USA* 109:8467–8470.
- Ferguson JE II, Newberry YG, DeAngelis GA, Finnerty JJ, Agarwal S, Turkheimer E. 1998. The fetal-pelvic index has minimal utility in predicting fetal-pelvic disproportion. *Am J Obstet Gynecol* 179:1186–1192.
- Forsum E, Sadurskis A, Wager J. 1989. Estimation of body fat in healthy Swedish women during pregnancy and lactation. *Am J Clin Nutr* 50:465–473.
- Franciscus RG. 2009. When did the modern human pattern of childbirth arise? New insights from an old Neandertal pelvis. *Proc Natl Acad Sci USA* 106:9125–9126.
- Galvin CA. 1992. Nutritional ecology of pastoralists in dry tropical Africa. *Am J Hum Biol* 4:209–221.
- Garenne M. 2011. Trends in nutritional status in adult women in sub-Saharan Africa. DHS Comparative Reports 27. Calverton, MD: ICF Macro.
- Geidam AD, Audu BM, Kawuwa BM, Obed JY. 2009. Rising trend and indications of caesarean section at the university of Maiduguri teaching hospital, Nigeria. *Ann Afr Med* 8:127–132.
- Gherman RB, Chauhan S, Ouzounian JG, Lerner H, Gonik B, Goodwin TM. 2006. Shoulder dystocia: the unpreventable obstetric emergency with empiric management guidelines. *Am J Obstet Gynecol* 195:657–672.
- Godfrey K, Robinson S, Barker DJ, Osmond C, Cox V. 1996. Maternal nutrition in early and late pregnancy in relation to placental and fetal growth. *BMJ* 312:410–414.
- Goldsmith LT, Weiss G. 2009. Relaxin in Human Pregnancy. *Ann NY Acad Sci* 1160:130–135.
- Goss CM, Popejoy LT II, Fusiler JL, Smith TM. 1968. Observations on the relationship between embryological development, time of conception, and gestation. In: Rosenblum LA, Cooper RW, editors. *The squirrel monkey*. New York: Academic Press. p 171–191.
- Greene MF, Allred EN, Leviton A. 1995. Maternal metabolic control and risk of microcephaly among infants of diabetic mothers. *Diabetes Care* 18:166–169.
- Greulich WW, Thoms H, Twaddle RC. 1939. A study of pelvic type: and its relationship to body build in white women. *JAMA* 112:485–493.
- Gudmundsson S, Henningsson AC, Lindqvist P. 2005. Correlation of birth injury with maternal height and birthweight. *BJOG* 112:764–767.
- Guegan JF, Teriokhin AT, Thomas F. 2000. Human fertility variation, size-related obstetrical performance and the evolution of sexual stature dimorphism. *Proc Biol Sci* 267:2529–2535.
- Guntupalli AM, Moradi A. 2008. What does gender dimorphism in stature tell us about discrimination in rural India, 1930–1975? In: Pal MB, Ghosh B, Vasulu TS, editors. *Gender and discrimination: health, nutritional status, and role of women in India*. New Delhi, India: Oxford University Press. p 258–277.
- Gupta M, Hockley C, Quigley MA, Yeh P, Impey L. 2010. Antenatal and intrapartum prediction of shoulder dystocia. *Eur J Obstet Gynecol Reprod Biol* 151:134–139.
- Haig D. 1993. Genetic conflicts in human pregnancy. *Q Rev Biol* 68:495–532.
- Hales CN, Barker DJ. 1992. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 35:595–601.
- Hampton JK, Hampton SH. 1965. Marmosets (*Hapiladae*): breeding seasons, twinning, and sex of offspring. *Science* 150:915–917.
- Hampton JK, Hampton SH, Landwehr BT. 1966. Observations on a successful breeding colony of the marmoset, *Oedipomidas oedipus*. *Folia Primatol* 4:265–287.
- Harrison KA, Fleming AF, Briggs ND, Rossiter CE. 1985a. Growth during pregnancy in Nigerian teenage primigravidae. *Br J Obstet Gynaecol* 5(Suppl):32–39.

- Harrison, KA, Rossiter CE, Chong H. 1985b. Relations between maternal height, fetal birth weight and cephalopelvic disproportion suggest that young Nigerian primigravidae grow during pregnancy. *Br J Obstet Gynaecol* 5(Suppl):40–48.
- Harrison KA, Briggs ND, John CT, Memberr MT, Lolomari DO. 1988. Growth during early teenage pregnancy. *Lancet* 1: 1226–1227.
- Harvey PH, Martin RD, Clutton-Brock TH. 1987. Life histories in comparative perspective. In: Smuts BB, Cheney DL, Seyfarth RM, Wrangham RW, Struhsaker TT, editors. *Primate societies*. Chicago: University of Chicago Press. p 181–196.
- Häusler M, Schmid P. 1995. Comparison of the pelvis of Sts 14 and AL288-1: implications for birth and sexual dimorphism in australopithecines. *J Hum Evol* 29:363–383.
- Hawkes SC, Wells C. 1975. An Anglo-Saxon obstetric calamity from Kingsworthy, Hampshire. *Med Biol Illus* 25:47–51.
- Hediger ML, Overpeck MD, Maurer KR, Kuczumarski RJ, McGlynn A, Davis WW. 1998. Growth of infants and young children born small or large for gestational age: findings from the Third National Health and Nutrition Examination Survey. *Arch Pediatr Adolesc Med* 152:1225–1231.
- Herculano-Houzel S. 2011. Scaling of brain metabolism with a fixed energy budget per neuron: implications for neuronal activity, plasticity and evolution. *PLoS One* 6:e17514.
- Heymsfield SB, Gallagher D, Mayer L, Beetsch J, Pietrobelli A. 2007. Scaling of human body composition to stature: new insights into body mass index. *Am J Clin Nutr* 86:82–91.
- Heyns OS. 1944. A study of the Bantu female pelvis. *J Anat* 78:151–166.
- Hiernaux J. 1968. *La diversité humaine en Afrique Sub-Saharienne*. Recherches biologiques. Bruxelles: Editions de l'Institut de Sociologie de l'Université Libre de Bruxelles.
- Hiernaux J, Froment A. 1976. The correlations between anthropological and climatic variables in sub-Saharan Africa: revised estimates. *Hum Biol* 48:757–767.
- Hill JC, Krishnaveni GV, Annamma I, Leary SD, Fall CH. 2005. Glucose tolerance in pregnancy in South India: relationships to neonatal anthropometry. *Acta Obstet Gynecol Scand* 84:159–165.
- Hirata S, Fuwa K, Sugama K, Kusunoki K, Takeshita H. 2011. Mechanism of birth in chimpanzees: humans are not unique among primates. *Biol Lett* 7:686–688.
- Hofman MA. 1983. Evolution of brain size in neonatal and adult placental mammals: a theoretical approach. *J Theor Biol* 105:317–332.
- Hogan MC, Foreman KJ, Naghavi M, Ahn SY, Wang M, Makela SM, Lopez AD, Lozano R, Murray CJ. 2010. Maternal mortality for 181 countries, 1980–2008: a systematic analysis of progress towards Millennium Development Goal 5. *Lancet* 375:1609–1623.
- Holland EL, Cran GW, Elwood JH, Pinkerton JH, Thompson W. 1982. Associations between pelvic anatomy, height and year of birth of men and women in Belfast. *Ann Hum Biol* 9:113–120.
- Holliday MA. 1978. Body composition and energy needs during growth. In: *Human growth; a comprehensive treatise*. Falkner F, Tanner JM, editors. New York: Plenum. p 101–117.
- Homko CJ, Sivan E, Reece EA, Boden G. 1999. Fuel metabolism during pregnancy. *Semin Reprod Endocrinol* 17:119–125.
- Hoppe C, Molgaard C, Thomsen BL, Juul A, Michaelsen KF. 2004a. Protein intake at 9 mo of age is associated with body size but not with body fat in 10-y-old Danish children. *Am J Clin Nutr* 79:494–501.
- Hoppe C, Udam TR, Lauritzen L, Molgaard C, Juul A, Michaelsen KF. 2004b. Animal protein intake, serum insulin-like growth factor I, and growth in healthy 2.5-y-old Danish children. *Am J Clin Nutr* 80:447–452.
- Howell N. 1972. *Demography of the Dobe !Kung*. New York: Aldine de Gruyter.
- Illsley R. 1966. Preventive medicine in the perinatal period. Early prediction of perinatal risk. *Proc R Soc Med* 59:181–184.
- Ince JH, Young M. 1938. The bony pelvis and its influence on labour: a radiological and clinical study of 500 women. *J Obstet Gynaecol* 47:130–190.
- Indorf C, Sherry B, Mancl L. 2001. Comparisons of Yupik infant growth measurements with NCHS/CDC reference data. *Alaska Med* 43:6–12,23.
- Isler K, van Schaik CP. 2006. Metabolic costs of brain size evolution. *Biol Lett* 2:557–560.
- Jarcho J. 1934. *Posture and practices during labour among primitive peoples*. New York: Paul B Hoeber.
- Johnson MR, Abbas AA, Allman AC, Nicolaidis KH, Lightman SL. 1994. The regulation of plasma relaxin levels during human pregnancy. *J Endocrinol* 142:261–265.
- Jönsson T, Granfeldt Y, Ahrén B, Branell UC, Pålsson G, Hansson A, Söderström M, Lindeberg S. 2009. Beneficial effects of a Paleolithic diet on cardiovascular risk factors in type 2 diabetes: a randomized cross-over pilot study. *Cardiovasc Diabetol* 8:35.
- Ju H, Chadha Y, Donovan T, O'Rourke P. 2009. Fetal macrosomia and pregnancy outcomes. *Aust NZ J Obstet Gynaecol* 49:504–509.
- Kamanu CI, Onwere S, Chigbu B, Aluka C, Okoro O, Obasi M. 2009. Fetal macrosomia in African women: a study of 249 cases. *Arch Gynecol Obstet* 279:857–861.
- Katzmarzyk PT, Leonard WR. 1998. Climatic influences on human body size and proportions: ecological adaptations and secular trends. *Am J Phys Anthropol* 106:483–503.
- Kelley JO, Angel JL. 1987. Life stresses of slavery. *Am J Phys Anthropol* 74:199–211.
- Kelly RL. 1995. *The foraging spectrum*. Washington: Smithsonian Institution Press.
- Khunpradit S, Patumanond J, Tawichasri C. 2005. Risk indicators for cesarean section due to cephalopelvic disproportion in Lamphun hospital. *J Med Assoc Thai* 88 (Suppl 2):S63–S68.
- Kibii JM, Churchill SE, Schmid P, Carlson KJ, Reed ND, de Ruiter DJ, Berger LR. 2011. A partial pelvis of *Australopithecus sediba*. *Science* 333:1407–1411.
- Kleiber M. 1961. *The fire of life: an introduction to animal energetics*. Huntingdon, NY: Krieger.
- Klein CJ. 2002. Nutrient requirements for preterm infant formulas. *J Nutr* 132 (6 Suppl 1):1395S–1577S.
- Kolawole TM, Adamu SP, Evans KT. 1978. Comparative pelvic measurements in Nigerian and Welsh women. *Clin Radiol* 29:85–90.
- Kramer MS. 1987. Determinants of low birth weight: methodological assessment and meta-analysis. *Bull World Health Organ* 65:663–737.
- Kramer MS, Kakuma R. 2003. Energy and protein intake in pregnancy. *Cochrane Database Syst Rev* (4):CD000032.
- Krogman WM. 1951. The scars of human evolution. *Sci Am* 184:54–57.
- Kruger DJ, Nesse RM. 2006. An evolutionary life-history framework for understanding sex differences in human mortality rates. *Hum Nat* 17:74–97.
- Kurki HK. 2007. Protection of obstetric dimensions in a small-bodied human sample. *Am J Phys Anthropol* 133:1152–1165.
- Kurki HK. 2011. Pelvic dimorphism in relation to body size and body size dimorphism in humans. *J Hum Evol* 61:631–643.
- Kurki HK, Ginter JK, Stock JT, Pfeiffer S. 2008. Adult proportionality in small-bodied foragers: a test of ecogeographic expectations. *Am J Phys Anthropol* 136:28–38.
- Kuzawa CW. 1998. Adipose tissue in human infancy and childhood: an evolutionary perspective. *Yearb Phys Anthropol* 27:177–209.
- Kwawukume EY, Ghosh TS, Wilson JB. 1993. Maternal height as a predictor of vaginal delivery. *Int J Gynaecol Obstet* 41:27–30.
- Larsen CS. 1995. Biological changes in human populations with agriculture. *Ann Rev Anthropol* 24:185–213.
- Larsen LB, Vinther BM, Briffa KR, Melvin TM, Clausen HB, Jones PD, Siggaard-Andersen M-L, Hammer CU, Eronen M, Grudd H, Gunnarson BE, Hantemirov RM, Naurzbaev MM, Nicolussi K. 2008. New ice core evidence for a volcanic cause of the A.D. 536 dust veil. *Geophys Res Lett* 35:L04708.
- Larsson J, Svanberg L. 1983. Teenage deliveries in a Swedish population in the 1970's. *Acta Obstet Gynecol Scand* 62:467–472.
- LaVelle M. 1995. Natural selection and developmental sexual variation in the human pelvis. *Am J Phys Anthropol* 98:59–72.

- Lawrence M, Coward WA, Lawrence F, Cole TJ, Whitehead RG. 1987. Fat gain during pregnancy in rural African women: the effect of season and dietary status. *Am J Clin Nutr* 45:1442–1450.
- Leary S, Fall C, Osmond C, Lovel H, Campbell D, Eriksson J, Forrester T, Godfrey K, Hill J, Jie M, et al. 2006a. Geographical variation in neonatal phenotype. *Acta Obstet Gynecol Scand* 85:1080–1089.
- Leary S, Fall C, Osmond C, Lovel H, Campbell D, Eriksson J, Forrester T, Godfrey K, Hill J, Jie M, et al. 2006b. Geographical variation in relationships between parental body size and offspring phenotype at birth. *Acta Obstet Gynecol Scand* 85:1066–1079.
- Lee AC, Darmstadt GL, Khatri SK, LeClerq SC, Shrestha SR, Christian P. 2009. Maternal-fetal disproportion and birth asphyxia in rural Sarlahi, Nepal. *Arch Pediatr Adolesc Med* 163:616–623.
- Leedam E. 1985. Traditional birth attendants. *Int J Gynecol Obstet* 23:249–274.
- Lefebvre L, Carli G. 1985. Parturition in non-human primates: Pain and auditory concealment. *Pain* 21:315–327.
- Lefebvre Y, Voorhoever H. 1997. Practices and beliefs of traditional birth attendants: lessons for obstetrics in the north? *Trop Med Int Health* 2:1175–1179.
- Lehner, NDM, Bullock, BC, Feldner MA, Clarkson TB. 1967. Observations on reproduction of laboratory-maintained squirrel monkeys. *Lab Primate Newsl* 6:1–3.
- Leutenegger W. 1973. Maternal-weight relationships in primates. *Folia Primatol* 20:280–293.
- Leutenegger W. 1974. Functional aspects of pelvic morphology in simian primates. *J Hum Evol* 3:207–222.
- Leutenegger W. 1982. Encephalization and obstetrics in primates with particular reference to human evolution. In: Armstrong E, Falk D, editors. *Primate brain evolution: methods and concepts*. New York: Plenum Press. p 85–95.
- Lewton KL. 2012. Complexity in biological anthropology in 2011: species, reproduction, and sociality. *Am Anthropol* 114:196–202.
- Li XF, Fortney JA, Kotelchuck M, Glover LH. 1996. The postpartum period: the key to maternal mortality. *Int J Gynaecol Obstet* 54:1–10.
- Liston MA, Papadopoulos JK. 2004. The “rich Athenian lady” was pregnant: the anthropology of a geometric tomb reconsidered. *Hesperia* 73:7–38.
- Liston WA. 2003. Rising caesarian section rates: can evolution and ecology explain some of the differences of modern childbirth? *J R Soc Med* 96:559–561.
- Lovejoy CO. 2005. The natural history of human gait and posture Part 1. Spine and pelvis. *Gait Posture* 21:95–112.
- MacDonell WR. 1913. On the expectation of life in ancient Rome, and in the provinces of Hispania and Lusitania, and Africa. *Biometrika* 9:366–380.
- Mahmood TA, Campbell DM, Wilson AW. 1988. Maternal height, shoe size, and outcome of labour in white primigravidas: a prospective anthropometric study. *BMJ* 297:515–517.
- Majzoub JA, McGregor JA, Lockwood CJ, Smith R, Taggart MS, Schulkin J. 1999. A central theory of preterm and term labor: putative role for corticotropin-releasing hormone. *Am J Obstet Gynecol* 180:S232–S241.
- Malina RM. 1990. Research on secular trends in auxology. *Anthropol Anzeig* 48:209–227.
- Manahan CP, Marquez C, Mangay AS. 1954. A roentgenologic study of the Filipino female pelvis. *Am J Obstet Gynecol* 68:228–235.
- Mandal D, Manda S, Rakshi A, Dey RP, Biswas SC, Banerjee A. 2011. Maternal obesity and pregnancy outcome: a prospective analysis. *J Assoc Phys India* 59:486–489.
- Marino L. 2006. Absolute brain size: did we throw the baby out with the bathwater? *Proc Natl Acad Sci USA* 103:13563–13564.
- Marland H, Rafferty AM. 1997. Midwives, society, and childbirth: debates and controversies in the modern period. London: Routledge.
- Martin R. 1928. *Lehrbuch der anthropologie*. Jena, Vienna: Gustav Fischer.
- Martin RD. 1983. Human brain evolution in an ecological context. 52nd James Arthur lecture on the evolution of the human brain. New York: American Museum of Natural History.
- Mathews F, Yudkin P, Neil A. 1999. Influence of maternal nutrition on outcome of pregnancy: prospective cohort study. *BMJ* 319:339–343.
- McCallum C. 2005. Explaining caesarean section in Salvador da Bahia, Brazil. *Sociol Health Illn* 27:215–242.
- McFarland MB, Trylovich CG, Langer O. 1998. Anthropometric differences in macrosomic infants of diabetic and nondiabetic mothers. *J Matern Fetal Med* 7:292–295.
- McLean M, Smith R. 2001. Corticotrophin-releasing hormone and human parturition. *Reproduction* 121:493–501.
- McMichael AJ. 2001. Diabetes, ancestral diets and dairy foods. In: Ethnicity and health. Macbeth H, Shetty P, editors. London: Taylor and Francis. p 133–146.
- Meire HB, Farrant P. 1981. Ultrasound demonstration of an unusual fetal growth pattern in Indians. *Br J Obstet Gynaecol* 88:260–263.
- Mendez MA, Plana E, Guxens M, Foradada Morillo CM, Albarreda RM, Garcia-Esteban R, Goni F, Kogevinas M, Sunyer J. 2010. Seafood consumption in pregnancy and infant size at birth: results from a prospective Spanish cohort. *J Epidemiol Commun Health* 64:216–222.
- Merbs CF. 2004. Sagittal cleaving of the body and other vertebral developmental errors in Canadian Inuit skeletons. *Am J Phys Anthropol* 123:236–249.
- Merewood A, Mehta SD, Chen TC, Bauchner H, Holick MF. 2009. Association between vitamin D deficiency and primary cesarean section. *J Clin Endocrinol Metab* 94:940–945.
- Michel SC, Rake A, Treiber K, Seifert B, Chaoui R, Huch R, Marincek B, Kubik-Huch RA. 2002. MR obstetric pelvimetry: effect of birthing position on pelvic bony dimensions. *Am J Roentgenol* 179:1063–1067.
- Mieklejohn C, Babb J. 2011. Long bone length, stature and time in the European Late Pleistocene and Early Holocene. In: Pithasi R, Stock JT, editors. *Human bioarchaeology of the transition to agriculture*. Chichester: Wiley Blackwell. p 153–175.
- Milton K. 2000. Hunter-gatherer diets—a different perspective. *Am J Clin Nutr* 71:665–667.
- Moerman ML. 1982. Growth of the birth canal in adolescent girls. *Am J Obstet Gynecol* 143:528–532.
- Moller B, Lindmark G. 1997. Short stature: an obstetric risk factor? A comparison of two villages in Tanzania. *Acta Obstet Gynecol Scand* 76:394–397.
- Moller-Christensen V. 1958. *Bogen om Aebelholt kloster*. Copenhagen: Dansk Videnskabs Forlag.
- Monasta L. 2011. Maternal height should be considered in the evaluation of macrosomia related risk of infant injuries at birth. *Acta Obstet Gynecol Scand* 90:198.
- Montagu A. 1961. Neonatal and infant immaturity in man. *JAMA* 178:56–57.
- Moore VM, Davies MJ, Willson KJ, Worsley A, Robinson JS. 2004. Dietary composition of pregnant women is related to size of the baby at birth. *J Nutr* 134:1820–1826.
- Morris FH, Boyd RDH. 1988. Placental transport. In: Knobil E, Neill JD, editors. *The physiology of reproduction*. New York: Raven Press. p 2043–2085.
- Moses RG, Luebecke M, Davis WS, Coleman KJ, Tapsell LC, Petocz P, Brand-Miller JC. 2006. Effect of a low-glycemic-index diet during pregnancy on obstetric outcomes. *Am J Clin Nutr* 84:807–812.
- Mossman HW. 1987. Vertebrate fetal membranes: comparative ontogeny and morphology, evolution, phylogenetic significance, basic functions, research opportunities. New Brunswick, NJ: Rutgers University Press.
- Murphy NJ, Butler SW, Petersen KM, Heart V, Murphy CM. 1996. Tobacco erases 30 years of progress: preliminary analysis of the effect of tobacco smoking on Alaska Native birth weight. *Alaska Med* 38:31–33.
- Naroll F, Naroll R, Howard FH. 1961. Position of women in childbirth. *Am J Obstet Gynecol* 82:943–954.
- Nasrat H, Abalkhail B, Fageeh W, Shabat A, el Zahranly F. 1997. Anthropometric measurement of newborns of gesta-

- tional diabetic mothers: does it indicate disproportionate fetal growth? *J Matern Fetal Med* 6:291–295.
- Nkwabong E, Fomulu JN. 2009. Adolescent pregnancies and deliveries: problems encountered. *Trop Doct* 39:9–11.
- Norwitz ER, Robinson JN, Repke JT. 2003. Labor and delivery. In: Gabbe SG, Niebyl JR, Simpson JL, editors. *Obstetrics: Normal and problem pregnancies*, 3rd ed. New York: Churchill Livingstone.
- Nystrom MJ, Caughey AB, Lyell DJ, Druzin ML, El-Sayed YY. 2008. Perinatal outcomes among Asian-white interracial couples. *Am J Obstet Gynecol* 199:e381–385.
- Ohlsén H. 1980. Radiologic low-dose pelvimetry. Indications and pelvimetry data. *Acta Radiol Diagn (Stockh)* 21:747–758.
- Olhager E, Forsum E. 2003. Total energy expenditure, body composition and weight gain in moderately preterm and full-term infants at term postconceptional age. *Acta Paediatr* 92:1327–1334.
- Olsen SF, Halldorsson TI, Willett WC, Knudsen VK, Gillman MW, Mikkelsen TB, Olsen J. 2007. Milk consumption during pregnancy is associated with increased infant size at birth: prospective cohort study. *Am J Clin Nutr* 86:1104–1110.
- Owens DW, Brattmiller B. 1983. Mortality of pregnant females in Arikara villages: osteological evidence. *Am J Phys Anthropol* 61:331–336.
- Pagel MD, Harvey PH. 1988. How mammals produce large-brained offspring. *Evolution* 42:948–957.
- Pagel MD, Harvey PH. 1990. Diversity in the brain sizes of newborn mammals. *Bioscience* 40:116–122.
- Pan N. 1929. Measurements of the Pelvis in Hindu Females. *J Anat* 63:263–266.
- Patel RR, Steer P, Doyle P, Little MP, Elliott P. 2004. Does gestation vary by ethnic group? A London-based study of over 122,000 pregnancies with spontaneous onset of labour. *Int J Epidemiol* 33:107–113.
- Paterson JD. 1996. Coming to America: acclimation in macaque body structures and Bergmann's Rule. *Int J Primatol* 17:585–612.
- Pfeiffer S. 2011. Pelvic stress injuries in a small-bodied forager. *Int J Osteoarchaeol* 21:694–703.
- Pfeiffer S, Harrington L. 2012. Child growth among mid-Holocene foragers of southern Africa. In: Moffat T, Prowse T, editors. *Human diet and nutrition*. Oxford: Berghahn Press.
- Pfeiffer S, Sealy J. 2006. Body size among Holocene foragers of the Cape Ecozone, southern Africa. *Am J Phys Anthropol* 129:1–11.
- Phillips IR. 1976. The reproductive potential of the common cotto-eared marmoset (*Callithrix jacchus*) in captivity. *J Med Primatol* 5:49–55.
- Pinhasi R, Stefanovic S, Papathanasiou A, Stock J. 2011. Variability in long bone growth patterns and limb proportions within and amongst Mesolithic and Neolithic populations from Southeast Europe. In: Pinhasi R, Stock JT, editors. *Human bioarchaeology of the transition to agriculture*. Chichester: Wiley Blackwell. p 177–202.
- Pomeroy E, Stock JT. 2012. Estimation of stature and body mass from the skeleton among coastal and mid-altitude Andean populations. *Am J Phys Anthropol* 147:264–279.
- Ponce de Leon MS, Golovanova L, Doronichev V, Romanova G, Akazawa T, Kondo O, Ishida H, Zollikofer CP. 2008. Neanderthal brain size at birth provides insights into the evolution of human life history. *Proc Natl Acad Sci USA* 105:13764–13768.
- Popkin BM, Adair LS, Ng SW. 2012. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev* 70:3–21.
- Poppitt SD, Prentice AM, Goldberg GR, Whitehead RG. 1994. Energy-sparing strategies to protect human fetal growth. *Am J Obstet Gynecol* 171:118–125.
- Portmann A. 1941. Die Tragzeiten der Primaten und die Dauer der Schwangerschaft beim Menschen: ein Problem der vergleichenden Biologie. *Rev Suisse Zool* 48:511–518.
- Potter JE, Berquó E, Perpétuo IH, Leal OF, Hopkins K, Souza MR, Formiga MC. 2001. Unwanted caesarean sections among public and private patients in Brazil: prospective study. *BMJ* 323:1155–1158.
- Potts R. 1996. *Humanity's descent: the consequences of ecological instability*. New York: William Morrow & Co.
- Price KC, Hyde JS, Coe CL. 1999. Matrilineal transmission of birth weight in the rhesus monkey (*Macaca mulatta*) across several generations. *Obstet Gynecol* 94:128–134.
- Pritchard J, MacDonald PC. 1985. *Williams obstetrics*, 16th ed. New York: Appleton-Century-Crofts.
- Pu F, Xu L, Li D, Li S, Sun L, Wang L, Fan Y. 2011. Effect of different labor forces on fetal skull molding. *Med Eng Phys* 33:620–625.
- Read AW, Prendiville WJ, Dawes VP, Stanley FJ. 1994. Caesarean section and operative vaginal delivery in low-risk primiparous women, Western Australia. *Am J Public Health* 84:37–42.
- Reynolds E, Hooton EA. 1936. Relation of the pelvis to erect posture: an exploratory study. *Am J Phys Anthropol* 21:253–278.
- Rhodes ET, Pawlak DB, Takoudes TC, Ebbeling CB, Feldman HA, Lovesky MM, Cooke EA, Leidig MM, Ludwig DS. 2010. Effects of a low-glycemic load diet in overweight and obese pregnant women: a pilot randomized controlled trial. *Am J Clin Nutr* 92:1306–1315.
- Ridley M. 1995. Brief communication: pelvic sexual dimorphism and relative neonatal brain size really are related. *Am J Phys Anthropol* 97:197–200.
- Roberts C, Cox M. 2003. *Health and disease in Britain: from prehistory to the present day*. Stroud: Sutton Publishing.
- Roberts DF. 1953. Body weight, race and climate. *Am J Phys Anthropol* 11:533–558.
- Roberts DF. 1973. *Climate and human variability*. An Addison-Wesley module in anthropology, No. 34. Reading, MA: Addison-Wesley Publishing Co, Inc.
- Rolland-Cachera MF, Deheeger M, Akrouf M, Bellisle F. 1995. Influence of macronutrients on adiposity development: a follow up study of nutrition and growth from 10 months to 8 years of age. *Int J Obes* 19:573–578.
- Rosenberg K. 1992. The evolution of modern childbirth. *Yearb Phys Anthropol* 35:89–124.
- Rosenberg K, Trevathan W. 1996. Bipedalism and human birth: the obstetrical dilemma. *Evol Anthropol* 4:161–168.
- Rosenberg K, Trevathan W. 2002. Birth, obstetrics and human evolution. *BJOG* 109:1199–1206.
- Rosenberg KR, Zune L, Ruff CB. 2006. Body size, body proportions, and encephalization in a Middle Pleistocene archaic human from northern China. *Proc Natl Acad Sci USA* 103:3552–3556.
- Rozenholc AT, Ako SN, Leke RJ, Boulvain M. 2007. The diagnostic accuracy of external pelvimetry and maternal height to predict dystocia in nulliparous women: a study in Cameroon. *BJOG* 114:630–635.
- Ruff C. 2002. Variation in human body size and shape. *Ann Rev Anthropol* 31:211–232.
- Ruff CB. 1994. Morphological adaptation to climate in modern and fossil humans. *Yearb Phys Anthropol* 37:65–107.
- Ruff CB. 1995. Biomechanics of the hip and birth in early Homo. *Am J Phys Anthropol* 98:527–574.
- Ruff C. 2010. Body size and body shape in early hominins—implications of the Gona pelvis. *J Hum Evol* 58:166–178.
- Rushton E, McGrew WC. 1980. Breech birth of a chimpanzee (*Pan troglodytes*). A case report and literature review. *J Med Primatol* 9:189–193.
- Russell JG. 1969. Moulding of the pelvic outlet. *Br J Obstet Gynaecol* 76:817–820.
- Russell JG. 1982. The rational of primitive delivery position. *Br J Obstet Gynaecol* 89:712–715.
- Sargent C. 1982. Solitary confinement: Birth practices among the Bariba of the People's Republic of Benin. In: Kay MA, editor. *Anthropology of human birth*. Philadelphia: F A Davis Company. p 193–210.
- Sargent, CF. 1989. *Maternity, medicine, and power: Reproductive decisions in urban Benin*. Berkeley: University of California Press.
- Saxena P, Tyagi S, Prakash A, Nigam A, Trivedi SS. 2011. Pregnancy outcome of women with gestational diabetes in a tertiary level hospital of north India. *Indian J Commun Med* 36:120–123.

- Scholl TO, Hediger ML, Ances IG. 1990. Maternal growth during pregnancy and decreased infant birth weight. *Am J Clin Nutr* 51:790–793.
- Scholl TO, Chen X, Khoo CS, Lenders C. 2004. The dietary glycaemic index during pregnancy: influence on infant birth weight, fetal growth, and biomarkers of carbohydrate metabolism. *Am J Epidemiol* 159:467–474.
- Schultz AH. 1949. Sex differences in the pelvis of primates. *Am J Phys Anthropol* 7:401–424.
- Schultz AH. 1969. *The life of primates*. New York: Universe Books.
- Schutz H, Donovan ER, Hayes JP. 2009a. Effects of parity on pelvic size and shape dimorphism in *Mus*. *J Morphol* 270:834–842.
- Schutz H, Polly PD, Krieger JD, Guralnick RP. 2009b. Differential sexual dimorphism: size and shape in the cranium and pelvis of grey foxes (*Urocyon*). *Biol J Linn Soc* 96:339–353.
- Sewell JE. 1993. *Cesarean section—a brief history*. Washington: American College of Obstetricians and Gynecologists.
- Sharma K. 2002. Genetic basis of human female pelvic morphology: a twin study. *Am J Phys Anthropol* 117:327–333.
- Shostak M. 1981. *Nisa: the life and words of a !Kung woman*. Boston: Harvard University Press.
- Shy K, Kimpo C, Emanuel I, Leisenring W, Williams MA. 2000. Maternal birth weight and cesarean delivery in four race-ethnic groups. *Am J Obstet Gynecol* 182:1363–1370.
- Sibley LM, Armelagos GJ, Van Gerven DP. 1992. Obstetric dimensions of the true pelvis in a medieval population from Sudanese Nubia. *Am J Phys Anthropol* 89:421–430.
- Simpson SW, Quade J, Levin NE, Butler R, Dupont-Nivet G, Everett M, Semaw S. 2008. A female *Homo erectus* pelvis from Gona, Ethiopia. *Science* 322:1089–1092.
- Sivan E, Homko CJ, Chen X, Reece EA, Boden G. 1999. Effect of insulin on fat metabolism during and after normal pregnancy. *Diabetes* 48:834–838.
- Skippen MW. 2009. *Obstetric practice and cephalopelvic disproportion in Glasgow between 1840 and 1900*. Glasgow: University of Glasgow.
- Slijper EJ. 1942. Biologic-anatomical investigations on the bipedal gait and upright posture in mammals, with special reference to a little goat, born without forelegs. II. *Proc Koninklijke Nederlandse Akademie Wetenschappen* 45:407–415.
- Sloan NL, Lederman SA, Leighton J, Himes JH, Rush D. 2001. The effect of prenatal dietary protein intake on birth weight. *Nutr Res* 21:129–139.
- Smith GC, White IR, Pell JP, Dobbie R. 2005. Predicting cesarean section and uterine rupture among women attempting vaginal birth after prior cesarean section. *PLoS Med* 2:e252.
- Smith RJ. 2009. Use and misuse of the reduced major axis for line-fitting. *Am J Phys Anthropol* 140:476–486.
- Smith RJ, Jungers WL. 1997. Body mass in comparative primatology. *J Hum Evol* 32:523–559.
- Smith RJ, Leigh SR. 1998. Sexual dimorphism in primate neonatal body mass. *J Hum Evol* 34:173–201.
- Sokal D, Sawadogo L, Adjibade A. 1991. Short stature and cephalopelvic disproportion in Burkina Faso, West Africa. Operations Research Team. *Int J Gynaecol Obstet* 35:347–350.
- Sorbe B, Dahlgren S. 1983. Some important factors in the molding of the fetal head during vaginal delivery—a photographic study. *Int J Gynaecol Obstet* 21:205–212.
- Specker B, Binkley T. 2005. High parity is associated with increased bone size and strength. *Osteoporos Int* 16:1969–1974.
- Stalberg K, Bodstedt A, Lyrenas S, Axelsson O. 2006. A narrow pelvic outlet increases the risk for emergency cesarean section. *Acta Obstet Gynecol Scand* 85:821–824.
- Steckel RH, Rose JC. 2002. *The backbone of history*. Cambridge: Cambridge University Press.
- Stein AD, Barnhart HX, Hickey M, Ramakrishnan U, Schroeder DG, Martorell R. 2003. Prospective study of protein-energy supplementation early in life and of growth in the subsequent generation in Guatemala. *Am J Clin Nutr* 78:162–167.
- Studel K. 1981. Sexual dimorphism and allometry in primate ossa coxae. *Am J Phys Anthropol* 55:209–215.
- Stock JT, Migliano AB. 2009. Stature, mortality, and selection for small body size among Indigenous populations of the Andaman Islands from 187101986. *Curr Anthropol* 50:713–725.
- Stock JT, Neill MCO, Ruff CB, Zabecki M, Shackelford L, Rose JC. 2011. Body size, skeletal biomechanics, mobility and habitual activity from the Late Palaeolithic to the Mid-Dynastic Nile Valley. In: Pinhasi R, Stock JT, editors. *Human bioarchaeology of the transition to agriculture*. Chichester: Wiley Blackwell. p 347–367.
- Storey R. 1986. Perinatal mortality at pre-Columbian Teotihuacan. *Am J Phys Anthropol* 69:541–548.
- Strickland SS. 1993. Human nutrition in Mongolia: maternal mortality and rickets. *Nomadic Peoples* 3:231–239.
- Stulp G, Verhulst S, Pollet TV, Nettle D, Buunk AP. 2011. Parental height differences predict the need for an emergency caesarean section. *PLoS One* 6:e20497.
- Subramanian SV, Ackerson LK, Smith GD. 2010. Parental BMI and childhood undernutrition in India: an assessment of intrauterine influence. *Pediatrics* 126:e663–e671.
- Tague RG. 1992. Sexual dimorphism in the human bony pelvis, with a consideration of the Neandertal pelvis from Kebara Cave, Israel. *Am J Phys Anthropol* 88:1–21.
- Tague RG. 2000. Do big females have big pelvises? *Am J Phys Anthropol* 112:377–393.
- Tague RG. 2005. Big-bodied males help us recognize that females have big pelvises. *Am J Phys Anthropol* 127:392–405.
- Tague RG. 1986. *Obstetric adaptations of the human bony pelvis*. Ph.D. Thesis. Ohio: Kent State University.
- Tague RG, Lovejoy CO. 1986. The Obstetric Pelvis of A.L. 288-1 (Lucy). *J Hum Evol* 15:237–255.
- Tague RG, Lovejoy CO. 1998. AL 288-1—Lucy or Lucifer: gender confusion in the Pliocene. *J Hum Evol* 35:75–94.
- Thoms H. 1937. Pelvic variations in 300 primiparous white women; a clinical study and a proposed classification. *Surg Gynecol Obstet* 64:700–704.
- Tobias PV. 1985. The negative secular trend. *J Hum Evol* 14:347–356.
- Tocheri MW, Dupras TL, Sheldrick P, Molto JE. 2005. Roman period fetal skeletons from the East Cemetery (Kellis 2) of Kellis, Egypt. *Int J Osteoarchaeol* 15:326–341.
- Tollanes MC, Rasmussen S, Irgens LM. 2008. Cesarean section among relatives. *Int J Epidemiol* 37:1341–1348.
- Trevathan W. 1988. Fetal emergence patterns in evolutionary perspective. *Am Anthropol* 90:674–681.
- Trevathan W, Rosenberg KR. 2000. The shoulders follow the head: postcranial constraints on human childbirth. *J Hum Evol* 39:583–585.
- Trevathan WR. 2011. *Human birth: an evolutionary perspective*. New Brunswick: AldineTransaction.
- Trivers RL. 1974. Parent-offspring conflict. *Am Zool* 14:249–264.
- van Steijn L, Karamali NS, Kanhai HH, Ariens GA, Fall CH, Yajnik CS, Middelkoop BJ, Tamsma JT. 2009. Neonatal anthropometry: thin-fat phenotype in fourth to fifth generation South Asian neonates in Surinam. *Int J Obes* 33:1326–1329.
- Varner MW, Fraser AM, Hunter CY, Corneli PS, Ward RH. 1996. The intergenerational predisposition to operative delivery. *Obstet Gynecol* 87:905–911.
- Vidarsdottir H, Geirsson RT, Hardardottir H, Valdimarsdottir U, Dagbjartsson A. 2011. Obstetric and neonatal risks among extremely macrosomic babies and their mothers. *Am J Obstet Gynecol* 204:e421–e426.
- Walker MJ, Ortega J, Parmová K, López MV, Trinkaus E. 2011. Morphology, body proportions, and postcranial hypertrophy of a female Neandertal from the Sima de las Palomas, southeastern Spain. *Proc Natl Acad Sci USA* 108:10087–10091.
- Walrath D. 2003. Rethinking pelvic typologies and the human birth mechanism. *Curr Anthropol* 44:5–32.
- Walsh JA. 2008. Evolution and the cesarean section rate. *Am Biol Teach* 70:401–404.
- Washburn SL. 1960. Tools and human evolution. *Sci Am* 203:63–75.
- Weaver TD. 2003. The shape of the Neandertal femur is primarily the consequence of a hyperpolar body form. *Proc Natl Acad Sci USA* 100:6926–6929.

- Weaver TD, and Hublin JJ. 2009. Neandertal birth canal shape and the evolution of human childbirth. *Proc Natl Acad Sci USA* 106:8151–8156.
- Weisbecker V, Goswami A. 2010. Brain size, life history, and metabolism at the marsupial/placental dichotomy. *Proc Natl Acad Sci USA* 107:16216–16221.
- Wells C. 1975. Ancient obstetric hazards and female mortality. *Bull NY Acad Med* 51:1235–1249.
- Wells JC. 2002. Thermal environment and human birth weight. *J Theor Biol* 214:413–425.
- Wells JC. 2003. Parent-offspring conflict theory, signaling of need, and weight gain in early life. *Q Rev Biol* 78:169–202.
- Wells JC. 2009. What was human birth weight in the past? Simulations based on data on stature from the palaeolithic to the present. *J Life Sci* 1:115–120.
- Wells JC. 2012a. Ecogeographical associations between climate and human body composition: analyses based on anthropometry and skinfolds. *Am J Phys Anthropol* 147:169–186.
- Wells JC. 2012b. Obesity as malnutrition: the role of capitalism in the obesity global epidemic. *Am J Hum Biol* 24:261–276.
- Wells JC, Cole TJ. 2002. Birth weight and environmental heat load: a between-population analysis. *Am J Phys Anthropol* 119:276–282.
- Wells JC, Murphy AJ, Buntain HM, Greer RM, Cleghorn GJ, Davies PS. 2004. Adjusting body cell mass for size in women of differing nutritional status. *Am J Clin Nutr* 80:333–336.
- Wells JC, Stock JT. 2011. Re-examining heritability: genetics, life history and plasticity. *Trends Endocrinol Metab* 22:421–428.
- Wells JC, Marphatia AA, Cole TJ, McCoy D. 2012. Associations of economic and gender inequality with global obesity prevalence: understanding the female excess. *Soc Sci Med* 75:482–490.
- Wertz RW, Wertz DC. 1977. *Lying in: a history of childbirth in America*. New York: Macmillan.
- Wheeler PE. 1992. The thermoregulatory advantages of large body size for hominids foraging in savanna environments. *J Hum Evol* 23:351–362.
- Wheeler PE. 1993. The influence of stature and body form on hominid energy and water budgets: a comparison of Australopithecus and early Homo. *J Hum Evol* 24:13–28.
- Willis A, Oxenham MF. 2011. A case of maternal and perinatal death in Neolithic Southern Vietnam, c. 2100–1050 BCE. *Int J Osteoarchaeol*, doi 10.1002/oa1296.
- Winder NR, Krishnaveni GV, Veena SR, Hill JC, Karat CL, Thornburg KL, Fall CH, Barker DJ. 2011. Mother's lifetime nutrition and the size, shape and efficiency of the placenta. *Placenta* 32:806–810.
- Wittman AB, Wall LL. 2007. The evolutionary origins of obstructed labor: bipedalism, encephalization, and the human obstetric dilemma. *Obstet Gynecol Surv* 62:739–748.
- Wooding P, Burton G. 2008. *Comparative placentation: structures, functions and evolution*. Berlin: Springer.
- World Health Organisation. 2005. *World Health Report: make every woman and child count*. Geneva: World Health Organisation.
- World Health Organisation. 2006. *Neonatal and perinatal mortality: country, regional and global estimates*. Geneva: World Health Organisation.
- Yajnik CS, Fall CH, Coyaji KJ, Hirve SS, Rao S, Barker DJ, Joglekar C, Kellingray S. 2003. Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. *Int J Obes* 27:173–180.
- Young M, Ince JG. 1940 A radiographic comparison of the male and female pelvis. *J Anat* 74:374–85.