The new 'obstetrical dilemma': stunting, obesity and the risk of obstructed labour

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Abstract

The 'obstetrical dilemma' refers to the tight fit between maternal pelvic dimensions and neonatal size at delivery. Most interest traditionally focused on its generic significance for humans, for example our neonatal altriciality and our complex and lengthy birth process. Across contemporary populations, however, the obstetrical dilemma manifests substantial variability, illustrated by differences in the incidence of cephalo-pelvic disproportion, obstructed labour and cesarean section. Beyond accounting for 12% of maternal mortality worldwide, obstructed labour also imposes a huge burden of maternal morbidity, in particular through debilitating birth injuries. This paper explores how the double burden of malnutrition and the global obesity epidemic may be reshaping the obstetrical dilemma. First, short maternal stature increases the risk of obstructed labour, while early age at marriage also risks pregnancy before pelvic growth is completed. Second, maternal obesity increases the risk of macrosomic offspring. In some populations, short maternal stature may also promote the risk of gestational diabetes, another risk factor for macrosomic offspring. These nutritional influences are furthermore sensitive to social values relating to issues such as maternal and child nutrition, gender inequality and age at marriage. Secular trends in maternal obesity are substantially greater than those in adult stature, especially in low- and middle-income countries. The association between the dual burden of malnutrition and the obstetrical dilemma is therefore expected to increase, because the obesity epidemic is emerging faster than stunting is being resolved. However, we currently lack objective population-specific data on the association between maternal obesity and birth injuries.

Keywords: Pelvis, birth weight, obesity, cesarean, obstructed labour, vesicovaginal fistula

Introduction

The challenges of giving birth in humans are familiar to all. For individual mothers, childbirth is routinely lengthy and painful, though to a variable degree. Several hundred thousand women worldwide die annually on account of pregnancy and childbirth, while tens of millions suffer from serious long-term complications following parturition (Hogan, Foreman et al. 2010). In the poorest populations, one in six women may die as a result of pregnancy or childbirth during their lifetime (Ronsmans, Graham et al. 2006). The provision of prompt access to comprehensive obstetrical emergency care, especially in low and middle-income countries, is therefore crucial to mitigate these burdens of morbidity and mortality.

Although there are multiple proximate factors accounting for variability in maternal mortality risk, at an ultimate level many of the challenges of childbirth have long been considered a legacy of our distant evolutionary heritage. Hominin evolution was characterized by the complex mosaic emergence of two 'quintessential' characteristics of modern humans – bipedal locomotion and large brain size (encephalization) – with the former preceding the latter. These two traits have mutual implications for each other, because in the absence of medical technology, the fetal head must pass through the dimensions of the maternal pelvis at birth (Rosenberg 1992, Rosenberg and Trevathan 2002). In the 1960s, it was suggested that antagonistic selective pressures, favouring both a constrained maternal pelvis for efficient locomotion and a large fetal head, maintained a relatively tight fit between these physical traits in Homo sapiens. Their problematic interaction became known as the 'obstetrical dilemma' (Washburn 1960), and was famously described by Krogman as a 'scar' of our evolutionary transition from quadripedal to bipedal stance (Krogman 1951).

One key consequence of the obstetrical dilemma, according to Washburn, was a shortened duration of pregnancy to allow the offspring to be delivered at an early stage of brain development, thus enabling passage through the maternal pelvis. This hypothesis has increasingly been questioned, and a competing hypothesis is that maternal metabolism reaches a threshold during pregnancy, beyond which fetal energy demands cannot be met. This results not in shorter gestation *per se*, rather in the offspring being delivered while still relatively 'immature' in comparison with the newborns of other ape species relative to maternal size (Ellison 2001, Ellison 2008, Dunsworth, Warrener et al. 2012).

A second key consequence of the obstetrical dilemma is an unusually complex birth process in our species, as highlighted by Trevathan and Rosenberg (Rosenberg and Trevathan 2002, Trevathan, Smith et al. 2008, Trevathan 2011). The duration of delivery is longer than in other apes, and the human fetus usually rotates as it passes through the pelvic canal, resulting in it facing away from the mother as it emerges. Human mothers therefore benefit from the assistance of others to minimize the risk of injury to the neonate, though solitary births have been recorded. The compressible fetal cranium facilitates delivery, and pelvic diameters can also expand slightly (Wells, DeSilva et al. 2012).

This broad view of the 'obstetrical dilemma' therefore focuses on issues fundamental to humans as a species. The anatomically complex birth process, the need for social assistance and the physical flexibility in the fetal cranium and maternal pelvis all emerge as components of a 'generic resolution' of this problem. Yet in many contemporary populations, birth complications are a major contributing factor to morbidity and mortality of both mothers and offspring (World Health Organisation 2005, Wall 2006, World Health

Organisation 2006). Obstructed labour, where delivery of the fetus causes harm to mother, offspring or both, may be considered to occur when the obstetrical dilemma is *not* resolved. It accounts for approximately 12% of maternal mortality worldwide (World Health Organisation 2005). Whilst many mothers deliver without complications, so that it is important not to unduly problematize a normal component of the human life cycle, an additional approach to the 'obstetrical dilemma' is required – focusing on its contribution to maternal and perinatal morbidity and mortality.

In other words, we need to understand the 'contemporary obstetrical dilemma', which need not necessarily replicate exactly that experienced by our distant ancestors (Wells, DeSilva et al. 2012). While metabolic constraints may account for our short gestation length relative to other ape species (Dunsworth, Warrener et al. 2012), this hypothesis cannot explain the frequency of obstructed labour, which occurs in the opposite context: the fetus is *larger* than the mother is capable of delivering without medical assistance. The contemporary obstetrical dilemma can thus be recast as one where each of mother and offspring is at risk of mortality arising from obstructed labour or other complications to the process of delivery.

Revisiting the obstetrical dilemma

Previously, I and colleagues explored the hypothesis that the obstetrical dilemma might have been exacerbated following the origins of agriculture (Wells, DeSilva et al. 2012). We argued that the widespread fall in stature that occurred around the time that agriculture emerged (Cohen and Armelagos 1984) is likely to have impacted adversely on the dimensions of the pelvis, as data from the Mediterranean indicate (Angel 1975). At the same time, a simultaneous shift towards agricultural diets high in carbohydrate, and an increased burden of infectious disease associated with sedentary living may each have favoured larger

92 neonatal adiposity and overall size at birth. On this basis, the obstetrical dilemma may have
93 been exacerbated prior to the historical period (Wells, DeSilva et al. 2012).

94 The implications for health are difficult to extract from the archaeological record, but
95 examples of birth injuries such as fistulae have been detected on ancient Egyptian mummies
96 from the second millennium BC, and the Ebers papyrus described several possible cures for
97 urinary incontinence, a common outcome of birth injury as discussed below (Mahfouz 1938).
98 In the 10th century AD, the Persian physician Ibn-Sīnā, known to Europeans as Avicenna, also
99 referred to urinary fistula as an incurable consequence of difficult childbirth (Mahfouz 1938).

100 Cesarean sections were recorded in antiquity, though their frequency is unknown and was
101 probably rare. The accounts typically refer to the fetus being extracted from a dead or dying
102 mother. The first reliable evidence for both mother and offspring surviving this process
103 comes from ancient Jewish writings in the 2nd century BCE to the 6th century CE (Lurie 2005).
104 In the late 19th century, a Scottish medical student observed a cesarean section performed
105 with skill and experience on a healthy young first-time mother in Uganda. The woman was
106 "liberally supplied with banana wine, and … in a state of semi-intoxication." The operator cut
107 rapidly through the abdominal and uterine walls, before removing the neonate. A red-hot
108 iron was used to staunch bleeding. The wound was subsequently closed with seven thin iron
109 spikes, a thick paste plastered on top, and a firm bandage applied. Within 11 days the
110 wound had fully healed, and both mother and child fared well (Felkin 1884, Allbrook 1962).
111 Thus, medical solutions to the obstetrical dilemma appear to have been possible in the past,
112 though there is minimal information on this issue in non-western societies.

113 The more recent archaeological record also provides numerous examples of fetuses
114 impacted in the maternal pelvis (Wells, DeSilva et al. 2012). Whilst the frequency of cephalo-

pelvic disproportion in the past remains unknown, it certainly occurred in various global regions. Yet surprisingly, susceptibility of the obstetrical dilemma to recent nutritional trends has received very little attention (Wells, DeSilva et al. 2012).

Globally, the incidence of obstructed labour varies substantially between geographical regions and between populations, suggesting that it is sensitive to ecological stresses. Furthermore, the incidence of obstructed labour has clearly changed over time within high-income populations, for example in association with declines in the prevalence of rickets (Dick 1922). On this basis, we can consider the 'obstetrical dilemma' as a labile health burden. If we can improve understanding of the basis of its variability, we may be more successful in preventing it, or minimizing its adverse consequences.

Global variability in the incidence of obstructed labour

Over recent decades, the global burden of maternal mortality has declined substantially, from an estimated 526,300 (95% CI 446,400 - 629,600) in 1980 to 342,900 (95% CI 302,100 - 394,300) in 2008 (Hogan, Foreman et al. 2010). Obstructed labour is a negligible component of maternal mortality in high-income countries, but it remains a major mortality burden in low-income countries (Khan, Wojdyla et al. 2006).

An inability to resolve common birth complications accounts for much of the regional variability in this burden. For example, over half of all maternal deaths occurred in just six countries that are especially populous and poor - India, Pakistan, Afghanistan, Nigeria, Ethiopia and the Democratic Republic of Congo (Hogan, Foreman et al. 2010). As discussed in detail below, these countries also have high levels of gender inequality and under-age marriage. In the absence of medical facilities for performing a cesarean section, whether the

barrier be geographical, financial or social, obstructed labour may prove fatal for mother and offspring.

However, the fact that obstructed labour accounts for approximately one eighth of this maternal mortality burden severely underestimates its full impact. Obstructed labour is a major source of maternal morbidity, whilst also contributing substantially to perinatal morbidity and mortality (Lawn, Cousens et al. 2005). In the late 1990s, it was estimated that for every maternal death, an estimated 149, 259, 300 and 591 women suffered serious maternal injuries during delivery in Indonesia, Bangladesh, India and Egypt respectively (Fortney and Smith 1996).

As highlighted by Wall (2006), one of the most serious maternal birth injuries is vesicovaginal fistula: 'an abnormal opening between the bladder and vagina that results in continuous and unremitting urinary incontinence'. In obstructed labour:

> *… the soft tissues of the pregnant woman's vagina, bladder, and rectum are compressed between the fetal head and the maternal pelvic bones by the contractions of the uterus. As the fetal head is forced tighter and tighter into the pelvis, the blood supply to the mother's soft tissues is progressively constricted, and, ultimately is shut off completely. The result is a widespread ischaemic injury that produces massive tissue damage throughout the maternal pelvis as well as fetal death from asphyxiation. In a day or two the dead fetus becomes macerated, softens, and changes its conformation in the maternal pelvis sufficiently that it can be expelled through the vagina. A few days later a slough of necrotic tissue comes away, leaving a fistula between the bladder and the vagina (or sometimes between the rectum and the vagina) in its place.* (Wall 2006)

The consequences for the mother have been described as the 'obstructed labour injury complex' (Arrowsmith, Hamlin et al. 1996). Broadly, the mother may experience various forms of urological, gynaecological, gastrointestinal, musculoskeletal, neurological or dermatological injury, not to mention social isolation, divorce, poverty, malnutrition, depression and premature mortality. The fetal case-fatality rate is also around 95% (Wall 2006). Whilst much attention has been directed to the physical injuries, the social consequences are arguably even more severe.

> *Although husbands and family members may initially be supportive and compassionate …, when it becomes clear that the constant loss of urine or feces is a chronic condition (viewed as incurable in the context of the traditional local culture) these women are usually divorced or abandoned by their husbands and often cast out by their families*. (Wall 2006)

A variety of factors contribute to the high prevalence of maternal birth injuries in low- and middle-income countries. These include gender inequality, poverty and low socio-economic status, malnutrition, illiteracy and lack of formal education, limited social autonomy, early marriage which may result in childbearing before the pelvis has completed its growth, and lack of access to emergency of obstetric services (Wall 2006). Many of these factors ultimately relate to nutrition, growth and development - physical processes that concern both mother and offspring, and furthermore their status in society.

A biological anthropological perspective may improve understanding of the contemporary obstetrical dilemma, by examining the biological basis of cephalo-pelvic disproportion and its sensitivity to nutritional stresses. In particular, the aim of this article is to explore the significance of ongoing secular trends in human nutrition, affecting somatic traits such as

maternal height and obesity and newborn size, for the burden of obstructed labour. Many children continue to experience stunting during childhood, and grow up into short adults. In addition, the global obesity epidemic is now affecting every country, though to different degrees, yet most attention has focused on its links with chronic disease, and its broader implications for human health through the life-course remain poorly understood.

Cephalo-pelvic disproportion: the maternal contribution

Cephalo-pelvic disproportion arises from a mismatch between the size of the fetus at the time of delivery, and the dimensions of the maternal pelvis. This is the primary cause of obstructed labour, which occurs 'when the presenting part of the fetus cannot progress into the birth canal despite strong uterine contractions' (Dolea and AbouZahr 2000). Other potential causes, less common, include malpresentation or malposition of the fetus. The shoulders of the fetus can also hinder passage through the pelvic outlet, a problem termed shoulder dystocia.

Fundamentally, we therefore need to understand potential ecological factors shaping both fetal size and maternal pelvic dimensions. The review below will clarify that both components of the 'dual burden of malnutrition' – stunting of growth and obesity – can exacerbate the obstetrical dilemma, through antagonistic influences on maternal body proportions and the size of the neonate (Wells 2015).

Surprisingly, relatively little research has been conducted on the intra-population determinants of variability in pelvic shape. Thoms and Godfried (Thoms and Godfried 1939) reported a correlation of 0.64 between maternal height and size of the pelvic inlet in 98 women, indicating that ~41% of the variance in pelvic dimensions is explained by maternal

size (**Figure 1**). Another study from Aberdeen reported both smaller and flatter pelves in shorter compared to taller women (Bernard 1950), while a study in Northern Ireland found not only that taller females had larger pelvic proportions, but also that the secular increase in height across generations was accompanied by increasing pelvic width (Holland, Cran et al. 1982). Among Ghanaian women, the correlation between height and diameter of the anterior-posterior diameter of the pelvic inlet was 0.51 (Adadevoh, Hobbs et al. 1989). A few small studies have reported modest or negligible correlations between maternal height and pelvic dimensions (Pan 1929, Mahmood, Campbell et al. 1988), and there is a need for further data on this association.

Figure 1 near here

Nonetheless, the widespread persistence of stunting among children in low- and middle-income countries can be predicted to have major implications for the population profile of pelvic dimensions in adult life. Consistent with that hypothesis, many studies have reported reduced rates of cesarean delivery in taller mothers (Smith, White et al. 2005, Stulp, Verhulst et al. 2011, Benjamin, Daniel et al. 2012), with an exponentially increasing risk of obstructed labour as maternal height declines (**Figure 2**).

Figure 2 near here

Importantly, the association between height and obstructed labour risk does not appear to be uniform across populations (Moller and Lindmark 1997, Konje and Ladipo 2000). **Table 1** provides evidence for the elevated risks of cesarean delivery in women of short stature across a range of populations. Although the different odds ratios (OR) prevent direct comparison, the data broadly indicate that the threshold below which the likelihood of

cephalo-pelvic disproportion increases is lower in Asian and African populations compared to European populations. Moreover, either the height cut-offs are slightly lower in Asian than African populations, or for a similar cut-off, Asian populations show higher OR than African populations. This is consistent with the elevated risks of maternal and perinatal morbidity/mortality and obstructed labour in Asian populations described above and below, but it also indicates that the obstetrical dilemma has been partially resolved by smaller neonatal size in African and Asian populations. According to data tabulated by the World Health Organization, mean birth weight by geographical region is 3415 g in Europe, 3120 g in Central and South America, 3080 g in Africa and 3020 g in Asia (World Health Organization 1992). In India, mean birth weight is ~2800 g (World Health Organization 1992).

Table 1 near here

However, from an evolutionary perspective it is misleading to consider adult height itself as an adaptive trait, rather what is adaptive is the *pattern of growth* that leads to that height. We therefore need to consider growth patterns, to understand how factors that impact linear growth may thereby shape the dimensions of the adult pelvis. In other words, as recognized for many other components of adult phenotype relevant to health (Barker 1992), we need to consider the 'developmental origins' of variability in the dimensions of the maternal pelvis and their association with neonatal size.

Many ecological factors contribute to growth variability between populations, but I will emphasize just four here. First, it is well established that human body shape is influenced by thermal climatic conditions (Ruff 1994, Ruff 2010). The need to mitigate heat stress in hot conditions favours a slimmer body shape, which is considered to account for narrower pelves in African and South Asian populations exposed to relatively high heat loads (Ruff

1994, Ruff 2010, Wells, DeSilva et al. 2012). This means that there is not a constant association across populations between maternal height and the dimensions of the pelvis, nevertheless within any population we can still expect a positive correlation between these traits.

Second, infectious diseases are well established to constrain growth during early life (Scrimshaw, Taylor et al. 1959), hence the burden of infectious disease to which any given population is exposed is likely to have implications for the size of the pelvis in adult life. Some of the clearest evidence for the impact of diseases comes from efforts to reduce their health burden. For example, treatment for the common gastrointestinal infection *Helicobacter pylori* in Colombian children improved growth (Mera, Bravo et al. 2012), while malaria treatment in Vietnam reduced the prevalence of stunting (Hung, de Vries et al. 2005). In India, the Universal Immunization Program was associated with greater weight and height in childhood (Anekwe and Kumar 2012). Notably, not all such efforts have been successful. Some interventions may arrive too late in development, for studies in Gambia infants show that gastro-intestinal infections can cause long-term damage to the gut, reducing nutrient capture (Lunn 2000, Campbell, McPhail et al. 2004). Nevertheless, extrapolating from the inverse association of infant mortality rate with adult height in 19th century European cohorts, declines in infectious disease alone might have increased height by ~0.5 cm/decade (Crimmins and Finch 2006). This should translate directly into a secular increase in the dimensions of the maternal pelvis.

Third, growth is clearly subject to intergenerational effects, demonstrated by correlations not merely between maternal size and birth weight, but also intergenerational correlations in birth weight itself (Emanuel, Filakti et al. 1992, Emanuel, Kimpo et al. 2004, Hypponen,

Power et al. 2004). Of particular interest here is the fact that the ovum that gives rise to a particular individual was already present in the form of an oocyte when the mother was herself *in utero* in the grandmother (Youngson and Whitelaw 2008). Given that plasticity in linear growth is greatest during fetal life and infancy (Smith, Truog et al. 1976), it is clear that inter-generational trends in stature are profoundly imprinted by maternal phenotype, thus damping the direct impact of external ecological conditions. In consequence, major secular trends in height tend to roll out across multiple generations, limiting the magnitude of change across any two generations. This also exposes the 'obstetrical dilemma' to maternal social status (Wells 2010) and may contribute to an inter-generational cycle of disadvantage regarding the risk of obstructed labour, as discussed below.

Fourth, beyond maternal height, micronutrient deficiencies may also reduce pelvic dimensions, since they also constrain early growth. Examples of micronutrients necessary for growth include zinc, iron and iodine (Branca and Ferrari 2002). Deficiency of vitamin D during development is specifically associated with constraint of the pelvis (Brabin, Verhoeff et al. 2002). However, rickets is uncommon in most contemporary populations, whereas variability in height clearly affects the risk of cephalo-pelvic disproportion and the need for cesarean delivery.

Beyond direct nutritional influences, maternal age is an additional factor of critical importance for the risk of cephalo-pelvic disproportion. Growth of the pelvis lags behind growth in stature (Moerman 1982) (**Figure 3**), and indeed there are some indications that pelvic growth continues into adult life, after final stature has been achieved (Tague 1994). The timing of menarche is also relevant, as early maturing girls have reduced pelvic dimensions during adolescence, whilst also being those in whom adolescent pregnancy is

most likely (Moerman 1982) (**Figure 4**). Pregnancy before pelvic growth has completed must inevitably increase the risk of obstructed labour, though it also provokes lower birth weight offspring. This connects nutrition and development directly with social values, customs and traditions.

Figures 3 & 4 near here

Of particular relevance, under-age marriage remains widely practiced in many low- and middle-income countries, in particular South Asia (**Figure 5**). In India, for example, despite the legal age for marriage being 18 years for girls (contrasting notably with an equivalent of 21 years for boys), over 200 million adult women aged 18+ years were married under-age (United Nations Children's Fund 2014), and under-age marriage remains very common in the 21st century. Even in 2005-6, 47% of Indian women aged 20-24 years had been married before 18 years. Key risk factors are poverty and lack of education, so that in many countries such as India, the wealthiest quintile marries around 4 years later than the poorest quintile. Yet in Bangladesh, even the wealthiest quintile has a median age of marriage of only 17 years (United Nations Children's Fund 2014).

Figure 5 near here

This makes low women's status a key contributing factor to the global variability in the obstetrical dilemma, so that the burden of the 'obstructed labour injury complex' is profoundly shaped by societal attitudes and values, not only through variable provision of medical care, but also in relation to susceptibility to birth difficulties. Indeed, gender inequality has significant implications for the inter-generational replication of short stature in women. A recent cross-country analysis linked societal gender inequality with increased

risk of low birth weight and childhood stunting, indicating that the burden of maternal morbidity associated with childbearing may be directly related to the status of women in society (Marphatia, Cole et al. 2016).

Collectively, therefore, we can see not only that a variety of ecological factors shape the dimensions of the maternal pelvis, with direct implications for the ease of delivery, but also that nutrition integrates both ecological and social stresses, each of which can profoundly influence patterns of growth and development. Looking beyond maternal phenotype, however, we must also take into account sources of variability in the size of the fetus.

Cephalo-pelvic disproportion: the offspring contribution

Traditionally, most attention has been directed to the association between neonatal head size and the risk of obstructed labour. For example, **Figure 6** illustrates how the association between maternal height and cesarean delivery in Guatemalan women is mediated by neonatal head girth (Merchant, Villar et al. 2001). However, head size is a highly canalized growth trait, demonstrated by a relatively low coefficient of variation in neonatal head girth between populations worldwide (Leary, Fall et al. 2006). The size of the neonatal body is likely to be of greater importance in understanding secular trends in the incidence of obstructed labour. For example, the coefficient of variation in neonatal skinfold is much greater than that in head girth (Leary, Fall et al. 2006). Central to the contemporary 'obstetrical dilemma' is therefore macrosomia, which refers to excessive fetal growth. Beyond large size *per se*, greater shoulder breadth (shoulder dystocia) can often impede delivery.

Figure 6 near here

Analysis of standardized data across 23 low- and middle-income countries shows clearly that macrosomia, like the association between maternal height and cephalo-pelvic disproportion, is not a uniform condition across populations (Ye, Torloni et al. 2015). Rather, definitions of macrosomia should be tailored to the risks experienced by both mothers and offspring. **Figure 7** illustrates the association between birth weight and the odds of maternal mortality and morbidity, perinatal mortality and morbidity, and intrapartum cesarean section stratified by the geographical regions Africa, Asia and Latin America. At any given birth weight, Asian populations demonstrate higher maternal and perinatal mortality/morbidity, and a higher risk of cesarean section. Of particular interest is the finding that the birth weight-associated increase in maternal morbidity/mortality is steeper than that for perinatal morbidity/mortality (Ye, Torloni et al. 2015), indicating that when women do have access to cesareans, the penalties of obstructed labour are on average greater for mothers than their offspring. In the absence of cesareans, however, the fetus has a much higher chance of dying.

Figure 7 near here

Once again, variability in offspring birth size reflects variation in fetal growth strategy. The fact that growth involves the deposition of new tissue indicates placental nutrition as the most obvious proximate determinant. Nevertheless, the regulation of fetal nutrition is complex, and the influence of maternal dietary intake during pregnancy appears relatively weak (Mathews, Yudkin et al. 1999, Mathews, Youngman et al. 2004), though maternal carbohydrate intake has been linked to birth weight (Moses, Luebcke et al. 2006). Broadly, maternal nutritional status (eg body mass index) at the time of conception and pregnancy weight gain appear stronger predictors of the offspring's size at birth than nutritional intake

363 during pregnancy (Neggers and Goldenberg 2003, Dietz, Callaghan et al. 2006, Frederick,
364 Williams et al. 2008). Nevertheless, this still leaves a substantial proportion of variability in
365 size at birth unexplained and curiously, the pelvis appears forgotten in this context.

366 At the start of the 20th century, Lane (Lane 1903) suggested that the size of the maternal
367 pelvis must inevitably constrain fetal growth: "*The child grows in utero in such a manner and*
368 *at such a rate that at full term his size is proportional to that of the mother's pelvis through*
369 *which he has to pass in order to be born*". Within four years, his study had been replicated
370 but his results and conclusion had been directly refuted (Leicester 1907). The potential value
371 for pelvic measurements to predict dystocia, obstructed labour and other birth
372 complications has been a controversial issue, with most researchers considering that at the
373 level of the individual, such measurements are of little value (Sporri, Hanggi et al. 1997, van
374 Dillen, Meguid et al. 2007, Maharaj 2010, Macones, Chang et al. 2013). Whilst various
375 components of maternal size (height, lean mass) are well-recognized predictors of birth
376 weight (Emanuel, Filakti et al. 1992, Emanuel, Kimpo et al. 2004, Emanuel, Kimpo et al. 2004,
377 Kulkarni, Shatrugna et al. 2006), the notion that the pelvis might mediate these associations
378 appears to have been wholly discarded.

379 Yet several indirect lines of evidence suggest that greater attention should be paid to the
380 notion that maternal pelvic dimensions may influence fetal growth. First, there is a
381 remarkable change in the level of heritability in body size during early life. During fetal life,
382 heritability of weight exceeds 50%, but declines to barely 20% around the time of birth,
383 before rising again to 60% by 15 months and 70% by 40 months (Gielen, Lindsey et al. 2008,
384 Mook-Kanamori, van Beijsterveldt et al. 2012). Although equivalent data are lacking for
385 length during mid-fetal life, a very similar pattern is evident from birth onwards. This

386 unusual pattern gives a strong indication that fetal growth becomes relatively plastic in the
387 period prior to birth, before becoming more strongly influenced by genetic factors in the
388 early postnatal period. Second, this dip in heritability is consistent with the notion that
389 natural selection has selected against 'birth weight genes' with large magnitude of effect,
390 precisely because secular shifts in maternal size could potentially result in high levels of
391 obstructed labour (Wells 2015).

392 If genetic factors are prevented from regulating late fetal growth, it is also clear that
393 mothers buffer their offspring from responding readily to a variety of environmental factors.
394 For example, maternal famine during pregnancy results in relatively modest declines in birth
395 weight of the offspring, while in the opposite direction, nutritional supplementation studies
396 during pregnancy produce relatively small increases in birth weight of the offspring, typically
397 <100 g (Wells 2003).

398 This indicates that the growing fetus tracks signals of maternal metabolism during
399 pregnancy, and maternal lean body mass may be a key mediating factor (Langhoff-Roos,
400 Lindmark et al. 1987, Kulkarni, Shatrugna et al. 2006, Elshibly and Schmalisch 2009). The
401 challenge is that fetal plasticity occurs one generation later than the equivalent period
402 during which growth of the maternal pelvis may have responded through plasticity to
403 ecological stresses (Wells 2015). Ecological stresses that shaped the maternal pelvis in the
404 past may be different from those affecting maternal metabolism during pregnancy.
405 Metabolism during pregnancy signals not only maternal body size to the fetus, but also the
406 mother's ability to maintain metabolic homeostasis. If the mother *cannot* maintain
407 homeostasis, the fetus – having evolved to track maternal metabolism – can only respond to
408 such metabolic perturbations (Wells 2007).

Maternal obesity and birth complications: the dual burden of malnutrition

Until recently, the primary nutritional challenges facing low- and middle-income countries were chronic protein-energy under-nutrition, micronutrient deficiencies and food insecurity. Accordingly, the primary risk factors for cephalo-pelvic disproportion were those associated with the constraint of pelvic dimensions, such as short stature, early marriage, poverty, infectious disease and malnutrition.

Within the last few decades, there has been a surge in the prevalence of obesity, now affecting urban populations in almost every country worldwide. Furthermore, the prevalence of obesity is typically greater in women compared to men in every country, particularly so in low-income countries and also those characterized by high levels of gender inequality (Wells, Marphatia et al. 2012). This epidemiological pattern means that obesity is increasing fastest in those populations already most prone to complications during childbirth. Since maternal obesity alters the physiological niche inhabited by the fetus, it has major implications for the risk of obstructed labour.

Even in the absence of gestational diabetes, maternal obesity influences the supply of fuel to the fetus. Already, therefore, one emerging consequence of the obesity epidemic is a systematic increase in the prevalence of macrosomia. As discussed above, since macrosomia refers to 'excessively large' neonates, and since the definition of 'excessively large' depends on maternal size, the epidemiology of macrosomia has been explored using a standardized approach, defining macrosomic neonates as those exceeding the 90th percentile of birth weight for any given population (Koyanagi, Zhang et al. 2013).

Table 2 provides evidence for associations between maternal overweight/obesity and the

odds of gestational diabetes in the mother, macrosomia in the offspring, and cesarean delivery in low- and middle-income countries. With very few exceptions, maternal overweight and obesity systematically increase all these risks, with dose-response associations evident between overweight and obese groups. These data are very similar to studies from high-income countries, which have linked maternal obesity with increased risks of the same outcomes (Gaudet, Ferraro et al. 2014).

Table 2 near here

In the cross-population analysis of Koyanagi and colleagues (2013), maternal diabetes and maternal obesity consistently doubled the risk of macrosomic offspring in Africa, Asia and Latin America. In this analysis, there was no evidence that macrosomic offspring increased the risk of maternal fistula, but since the data were collected from facilities able to conduct cesarean sections, this analysis is unlikely to offer substantial insight into the true association between maternal obesity and the risk of fistula.

A key question in low and middle-income countries is whether susceptibility to metabolic dysfunction in pregnancy, increasing the risk of cephalo-pelvic disproportion, may itself be exacerbated by under-nutrition in early life. It is not possible in most published studies to stratify obesity status in women according to whether or not they were stunted in early life (though see below), but it is possible to test for associations of stunting with later gestational diabetes. **Table 3** describes differences in height between mothers with gestational diabetes and those with normal glucose control, from a variety of countries. In most cases, diabetic mothers are significantly shorter and this association can be linked back to early maternal development, for low maternal birth weight also predicts gestational diabetes (Innes, Byers et al. 2002).

However, there are some exceptions, including Pune in India (Kale, Kulkarni et al. 2005), Bangladesh, Jamaica (Richardson and Trotman 2014) as well as some studies from West Africa (Lawoyin 1993, Abena Obama, Shasha et al. 1995, Onyiriuka 2006), where tall mothers are at greater risk of gestational diabetes. In these populations, it is likely that nutritional intake was high throughout development, so that obesity in adulthood is correlated with above-average linear growth during childhood. **Figure 8** shows how the association between growth rate in early life and obesity and gestational diabetes in adult life appears to be U-shaped, with contrasting underlying metabolic mechanisms. More commonly, however, diabetic mothers appear to have shorter stature, consistent with studies linking growth retardation during fetal life and infancy with adverse effects on growth of the pancreas, reducing the capacity for insulin secretion. This reduced beta-cell function is then exposed under the stress of adult obesity.

Table 3 and Figure 8 near here

Looking from another perspective, **Table 4** describes the odds ratio for gestational diabetes according to maternal short stature across countries, taking into account varying thresholds for defining short stature. In all these studies, shorter women have an elevated risk of gestational diabetes, though the studies are too few to identify any more specific pattern. **Figure 9** illustrates the association between maternal height and the risk of gestational diabetes in Brazilian women, mediated by adiposity (Branchtein, Schmidt et al. 2000).

Table 4 and Figure 9 near here.

Taken together, this evidence suggests that short stature is a widespread risk factor for diabetes, though with some exceptions, indicative of contrasting pathways linking maternal

476 growth patterns with poor glycemic control during pregnancy. Hypothetically, gestational
477 diabetes could mediate the interaction between maternal short stature and obesity, as
478 highlighted in **Figure 10**. As yet, however, it remains unclear the extent to which short
479 stature might contribute to macrosomic offspring through perturbation of maternal
480 metabolism. In the study of Koyanagi and colleagues (Koyanagi, Zhang et al. 2013), based on
481 hospital samples, tall maternal stature was a clear risk factor for macrosomia across Africa,
482 Asia and Latin America, and short maternal stature was protective.

483 *Figure 10 near here*

484 As yet, the strongest evidence is that both maternal short stature and maternal obesity and
485 diabetes contribute to the risk of cephalo-pelvic disproportion, the first constraining the
486 maternal pelvis, and the second promoting excessive offspring size. An interaction between
487 maternal short stature and fetal macrosomia in predicting birth injury is explicit in several
488 studies from high-income countries (Cnattingius, Cnattingius et al. 1998, Gudmundsson,
489 Henningsson et al. 2005, Dyachenko, Ciampi et al. 2006). In low- and middle-income
490 countries, however, this interaction appears to have attracted little attention, because
491 studies have focused on discrete populations of short or obese mothers, and have not yet
492 explored their combined effects.

493 Since the two maternal traits identified here – short stature and obesity – are both
494 replicated across generations, through both genetic and non-genetic mechanisms, the risk of
495 obstructed labour may likewise have a heritable component. This inter-generational
496 association may be both promoted and exacerbated by gender inequality. For example, the
497 social isolation experienced by mothers suffering debilitating birth injury may increase the
498 risk of malnutrition and stunting in their offspring, making the next generation susceptible to

the same burden of ill-health (**Figure 11**). Other factors such as poverty and early marriage may also persist across generations.

Figure 11 near here

Conclusion

Going beyond the generic nature of the complex birth process common to all humans, the contemporary 'obstetrical dilemma' merits consideration as the product of antagonistic nutritional stresses that may constrain maternal pelvic dimensions or promote fetal growth. The 'dual burden of malnutrition' may paradoxically be generating a 'new obstetrical dilemma', where the human birth process may be more risky for many women than it typically was during broader hominin evolution. In other words, the high levels of maternal mortality (often provoking offspring mortality) occurring in many low and middle income countries in the present and recent past may not provide an accurate indication of the burden of mortality in our ancestors in the more distant past.

As yet, we can detect two clear clusters of risk for obstructed labour. On the one hand is short maternal stature, associated with poverty, malnutrition and societal gender inequality. A key mediating factor is young maternal age at childbearing, which may constrain the development of the pelvis, or result in offspring being produced before pelvic growth has been completed. Women's subordinate status may likewise deny them access to appropriate medical care. On the other hand, maternal obesity may provoke fetal macrosomia, resulting in oversized offspring that are difficult to deliver. Gestational diabetes exacerbates this effect. Thus, short women and obese women are each independently expected to be at the risk of obstructed labour, for contrasting reasons.

These two clusters of risk are predicted increasingly to overlap, to the extent that women experience both stunting in early life and excess weight gain subsequently. When first reported, the dual burden of malnutrition appeared to affect very different populations, but it is increasingly clear that the obesity epidemic is impacting populations in whom widespread stunting has yet to be resolved (Wells 2013). An increasing proportion of women may be characterized by both stunting and obesity (Ramirez-Zea, Kroker-Lobos et al. 2014), but as yet, we have very little data on the potentially elevated risks of obstructed labour that might afflict such women. Gestational diabetes could potentially amplify this composite risk, both provoked by short stature and also exacerbating the negative impact of obesity.

Similarly, there are negligible data on the potential link between maternal obesity and the risk of fistula. The pioneering analysis of Koyanagi and colleagues (2013), was conducted in hospital samples, where the complications of maternal obesity could be resolved by medical interventions. As increasing proportions of rural populations in low and middle-income countries are exposed to the obesogenic niche, the combined impact of stunting and obesity in mothers may begin to be apparent. There are thus several key issues for further research (**Box 1**).

Intuitively, one might expect that increases in the supply of food might resolve the obstetrical dilemma, by promoting secular trends in height rather than body fat. The outstanding height of the contemporary Dutch population emerged for example through a secular trend in height drawn out over 150 years (Drukker and Tassenaar 1997, Fredriks, van Buuren et al. 1998). Yet the available data from low- and middle-income countries shows

545 that secular trends in height are much more modest than those evident in Europe (Deaton
546 2007, Deaton 2008, Subramanian, Özaltin et al. 2011), whereas increases in obesity are rapid
547 (Ng, Jones-Smith et al. 2010, Finucane, Stevens et al. 2011, Ng, Fleming et al. 2014). For
548 complex reasons, an increase in energy supply is not being converted into linear growth, and
549 is instead driving an epidemic of excess body weight. One potential explanation is that low
550 and middle income countries remain subject to higher burdens of infectious disease, which
551 may constrain increases in height during early life (Wells 2016). Exposure to higher levels of
552 energy intake from childhood onwards maybe unable to impact height, and may hence drive
553 excess weight gain instead.
554
555 In any case, the nutrition transition is not simply an increase in food availability, but also a
556 shift towards energy dense diets that are simultaneously deficient in micronutrients. Related
557 trends are a reduction in physical activity, further perturbing metabolism (Hallal, Andersen
558 et al. 2012). Poor diets are therefore central to both the persistence of stunting and the
559 rapid emergence of obesity. Women's disempowerment plays a key role in this scenario, for
560 gender-unequal societies simultaneously have higher prevalences of stunted children
561 (Marphatia, Cole et al. 2016) and an 'excess exposure' of women to the obesogenic niche
562 (Wells, Marphatia et al. 2012). Healthy nutrition is thus critical for reducing the
563 contemporary obstetrical dilemma, and gender equality must be promoted to achieve this
564 aim.
565
566 **Acknowledgements**
567 I am very grateful to Akanksha Marphatia of the University of Cambridge for valuable
568 comments on the manuscript.

Box 1. Key issues for further research

Research – epidemiology

What is the prevalence of stunted-obese mothers in low- and middle-income countries?

What are the independent and combined contributions of maternal short stature and maternal obesity to the risk of obstructed labour?

What are the independent and combined contributions of maternal short stature and maternal obesity to the risk of maternal birth injuries?

Are the associations of maternal obesity with obstructed labour and birth injuries different in adolescent mothers versus adult mothers?

Research – mechanisms

How does gestational diabetes relate to early-life stunting and adult obesity, and the timing of their emergence through the life-course?

How does the pelvis grow through adolescence and into early adult life?

Policy issues

How do we provide universal access to comprehensive emergency obstetric care for all

588 pregnant women, especially in low- and middle-income countries?

589 What impact do policies preventing under-age marriage have on the prevalence of
590 obstructed labour?

591 What impact do policies promoting gender equality have on the prevalence of obstructed
592 labour and social responses to birth injuries?

593 What progress has been made in the provision of medical treatment for birth injuries?

594

595

596 **Legends for illustrations**

597

598 **Figure 1.** Correlation table for maternal height and the size of the pelvic inlet in 98 women.
599 Reproduced with permission from Thoms and Godfried, 1939.

600 **Figure 2**. Associations between incidence of intrapartum cesarean per 1000 deliveries and
601 maternal height, in women from Ouagadougou, Burkina Faso. Based on data of (Sokal,
602 Sawadogo et al. 1991).

603 **Figure 3**. Growth remaining in stature and four pelvic dimensions (inlet diameter,
604 interspinous diameter, maximum breadth of the sacral alae, and inferior breadth of the
605 ischial tuberosities. in the period post-menarche, showing the relative delayed growth of the
606 pelvis relative to stature. All data indexed to size at 18 years. Based on data of Moerman
607 (1982).

608 **Figure 4**. Growth in stature and diameter of pelvic inlet following menarche, stratified by
609 early versus late menarche girls, showing the relatively smaller dimensions in girls
610 experiencing early menarche. Based on data of Moerman (1982).

611 **Figure 5**. Regional distribution as percentage of global total of 700 million of women aged
612 18+ years who were married or in union before the age of 18 years. Based on data of UNICEF
613 2014.

614 **Figure 6**. Associations between incidence of intrapartum cesarean delivery and maternal
615 height, stratified by neonatal head girth. Data from Merchant et al., 2001.

616 **Figure 7**. Associations between neonatal birth weight categories and the risk of (a) maternal

617 mortality and morbidity, (b) perinatal mortality and morbidity and (c) intrapartum cesarean
618 section in low- and middle-income countries, stratified by global region. Based on data of Ye,
619 Torloni et al. 2015.

620 **Figure 8.** Schematic diagram of the U-shaped association between growth in early life and
621 risk of obesity in adult life. Populations therefore differ in whether tall or short stature is a
622 risk factor for cephalo-pelvic disproportion.

623 **Figure 9.** Association between the odds of gestational diabetes and maternal height,
624 mediated by sum of skinfolds (high/low, categorized as above/below below average) in
625 women from 6 state capitals in Brazil. Based on data of Branchtein et al., 2000.

626 **Figure 10**. Schematic diagram illustrating the associations of maternal short stature and
627 obesity with the risk of obstructed labour, mediated by associations of maternal stature and
628 obesity with gestational diabetes.

629 **Figure 11**. Schematic diagram illustrating potential inter-generation associations in relation
630 to obstructed labour, whereby the phenotype of offspring exposed to this experience in
631 early life may increase the risk of obstructed labour recurring when they themselves deliver
632 offspring.

633

Table references (this inserts them in bibliography, this list can be deleted in the production process)

Table 1 refs

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Table 2 refs

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Table 3

(Anastasiou, Alevizaki et al. 1998, Jang, Min et al. 1998, Kousta, Lawrence et al. 2000, Yang, Hsu-Hage et al. 2002, Moses and Mackay 2004, Kale, Kulkarni et al. 2005, Keshavarz, Cheung et al. 2005, Iqbal, Rafique et al. 2007, Ogonowski and Miazgowski 2010)

Table 4

(Jang, Min et al. 1998, Branchtein, Schmidt et al. 2000, Di Cianni, Volpe et al. 2003, Zargar, Sheikh et al. 2004, Rudra, Sorensen et al. 2007, Dode and Santos Ida 2009)

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Table 1. Maternal height risk-thresholds for caesarean delivery by geographical region

Country	Population	N	Height cut-off	OR Cesarean delivery	Reference
Europe/Australia					
UK	Inverness	563	< 159 cm	2.8 (1.7, 4.6)	Mahmood et al., 1988
Australia	Western Australia	3,641	< 164 cm to 160 cm	2.4 (1.7, 3.5)	Read et al., 1994
			< 160 cm	5.3 (3.7, 7.6)	
Sweden	National sample	92,623	<164 to 155 cm	2.0 (1.8, 2.2)	Cnattingius et al., 1988
			< 154 cm	4.5 (3.9, 5.2)	
US	Baltimore	4,346	< 157 cm	1.6 (1.3, 1.8)	Witter et al., 1995
Latin America					
Guatemala	Guatemala City	3,377	< 146 cm	2.5 (2.1, 2.9)	Merchant et al., 2001
Africa					
DRC	Kinshasa	605	< 150 cm	2.2 (0.9, 5.4)	Liselele et al., 2000
Burkina Faso	Ouagadougou	1,733	< 155 cm	4.9 (2.3, 10.5)	Sokal et al., 1991
Zimbabwe		502	< 160 cm	2.0 (1.3, 3.0)	Tsu et al., 1992
Malawi	Rural non-adolescents	1,523	< 150 cm	1.5 (0.5, 4.5)	Brabin et al., 2002
	Rural adolescents		< 150 cm	5.6 (0.8, 41.1)	
Asia					
Thailand	Chiang Mai	9,198	< 145 cm	2.4 (1.8, 3.0)	Toh-Adam et al., 2012
Thailand	Lamphun	200	< 152 cm	3.6 (1.6, 8.2)	Khunpradit et al., 2005
India	Vellore	987	< 150 cm	4.1 (2.2, 7.3)	Seshadri and Mukherjee 2005
India	Baroda	1202	< 145 cm	8.1 (5.0, 13.1)	Desai et al., 1989
Oceania					
Papua New Guinea	Port Moresby	438	< 150 cm	2.9 (1.2, 7.4)	Amoa et al., 1997

OR – odds ratio and 95% confidence intervals

Table 2. Associations of maternal overweight and obesity with the risk of maternal gestational diabetes, offspring macrosomia, and cesarean delivery

Country	Population	N	Exposure *	Reference *	OR GDM	OR Macrosomia	OR Cesarean	Author
Africa								
Djibouti	Djibouti City	497	25-29.9	18.5-24.9	1.6 (0.9, 2.8)	1.4 (0.5, 3.9)	1.5 (0.8, 2.6)	Minsart et al., 2014
			30-34.9	18.5-24.9	1.9 (1.0, 3.6)	2.8 (1.0, 7.8)	2.3 (1.2, 4.4)	
			>35	18.5-24.9	2.3 (1.1, 5.1)	2.5 (0.7, 9.0)	3.2 (1.4, 7.0)	
Morocco	Benslimane	1,408	25-29.9	20-24.9		1.3 (0.9, 1.8)	1.2 (0.8, 1.7)	Mochhoury et al., 2013
			>30	20-24.9		4.2 (2.7, 6.8)	3.0 (1.8, 5.0)	
Nigeria	SE region	648	>30	18.5-24.9	4.3 (1.6, 11.7)	4.1 (1.1, 8.4)	4.3 (1.2, 5.4)	Iyoke et al., 2013
Ghana	Accra	1,000	25-29.9	18.5-24.9		2.5 (1.2, 5.2)	1.4 (0.9, 2.4)	Van der Linden et al., 2016
			>30	18.5-24.9		2.1 (0.9, 5.1)	2.1 (1.2, 3.6)	
South Africa	Johannesburg	767	25-29.9	18.5-24.9	0.3 (0.1, 2.0)		0.9 (0.6, 1.4)	Basu et al., 2010
			30-39.9	18.5-24.9	1.1 (0.3, 4.5)		1.0 (0.7, 1.5)	
			>40	18.5-24.9	2.6 (0.4, 16.0)	1.5 (0.7, 3.1)		
Asia								
China	Beijing	12,396	24-27.9	18-23.9	1.9 (1.7, 2.1)	1.5 (1.3, 1.8)	1.7 (1.5, 1.8)	Wei et al., 2015
			>28	18-23.9	2.5 (2.2, 3.0)	1.7 (1.4, 2.2)	2.6 (2.2, 3.1)	
China	Shenyang	5,047	24-28	18.5-24	2.5 (1.8, 3.4)	1.4 (1.1, 1.9)	1.5 (1.3, 1.7)	Liu et al., 2011
			>28	18.5-24	4.3 (3.0, 6.3)	1.9 (1.3, 2.7)	2.5 (2.0, 3.2)	
Thailand	Bangkok	3,715	25-29.9	18.5-24.9	4.0 (2.7, 6.1)	1.7 (0.9, 3.3)	1.4 (1.1, 1.7)	Saereeporncharenkul 2011
			>30	18.5-24.9	6.0 (3.5, 10.3)	5.4 (2.7, 10.5)	2.1 (1.5, 2.9)	
India	New Delhi	400	25-29.9	20-24.9	3.1 (1.7, 3.6)		2.6 (1.7, 3.9)	Kumari et al., 2014
			>30	20-24.9	7.4 (3.1, 17.4)		7.2 (3.0, 17.6)	
Pakistan	Karachi	4,735	25-29.9	18.5-24.9		1.5 (1.1, 2.1)	1.0 (0.8, 1.1)	Munim and Maheen 2012
			>30	18.5-24.9		2.3 (1.6, 3.1)	1.2 (1.0, 1.5)	
Latin Am								
Brazil	6 state capitals	5564	25-30	18.5-24.9	2.0 (1.6. 2.5)	1.6 (1.3, 2.0)		Nucci et al., 2001
			>30	18.5-24.9	2.4 (1.6, 3.4)	1.5 (1.1, 2.2)		

* Exposure and reference refer to BMI values in kg/m^2

OR – odds ratio and 95% confidence intervals

Table 3. Differences in height between women with gestational diabetes and control women

Country	Population	Height - GDM	Height - controls	Difference	Reference
High-income					
Greece	Athens	158.7 ± 0.2	161.0 ± 0.1	- 2.5 (-3.1, -2.0)	Anastasiou et al., 1988
UK	London - Europeans	162.9 ± 0.5	165.3 + 0.4	-2.4 (-3.6, -1.2)	Kousta et al., 2000
UK	London – South Asians	155.2 ± 0.5	158.2 ± 0.6	-3.0 (-4.7, -1.3)	Kousta et al., 2000
Australia	Wollongong	162.1 ± 0.9	164.9 ± 0.5	-2.8 (-4.8, -0.8)	Moses and Mackay, 2004
Poland	Szczecin	163.8 ± 0.4	165.7 ± 0.2	-1.9 (-2.7, -1.0)	Ogonowski and Miazgowski, 2010
Middle-income					
China	Tianjin	160.8 ± 0.4	161.7 ± 0.1	- 0.9 (-1.7, -0.1)	Yang et al., 2002
South Korea	Seoul	158.1 ± 0.4	160.0 ± 0.1	-1.9 (-2.6, -1.1)	Jang et al., 1998
Iran	Shahrood	153.9 ± 0.7	156.1 ± 0.1	- 2.2 (-3.6, -0.8)	Keshavarz et al., 2005
India	Pune	154.4 ± 0.4	153.0 ± 0.4	1.4 (1.1, 2.6)	Kale et al., 2005
Pakistan	Karachi	158.2 ± 0.8	159.1 ± 0.2	-0.9 (-2.5, 0.7)	Iqbal et al., 2007

Table 4. Odds ratios for gestational diabetes in women of short stature

Country	Population	Height groups	OR Short stature	Author
High-income				
USA	Washington State	≤ 160 cm vs > 170 cm	4.1 (1.7, 9.4)	Rudra et al., 2007
Italy	Pisa	≤ 155 cm vs > 170 cm	2.4 (1.4, 3.9)	Di Cianni et al., 2003
Middle-income				
Brazil	Six state capitals	≤ 151 cm vs > 160 cm	1.6 (1.1, 2.2)	Branchtein et al., 2000
Brazil	Pelotas	< 154 cm vs > 163 cm	1.8 (1.0, 3.0)	de Oliveira Dode and dos Santos, 2009
South Korea	Seoul	≤ 157 cm vs ≥ 163 cm	2.0 (1.4, 3.0)	Jang et al., 1998
India	Srinigar	≤ 157 cm vs >157 cm	6.5 (4.0, 10.5)	Zargar et al., 2004

OR – odds ratio and 95% confidence intervals

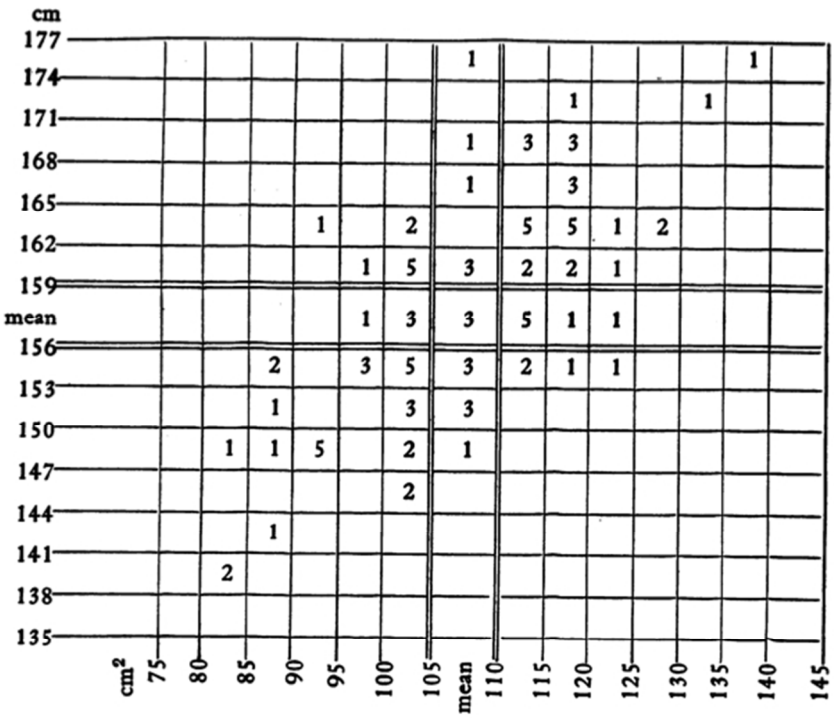

Figure 1. Correlation table for maternal height and the size of the pelvic inlet in 98 women. Reproduced with permission from Thoms and Godfried, 1939.

254x190mm (72 x 72 DPI)

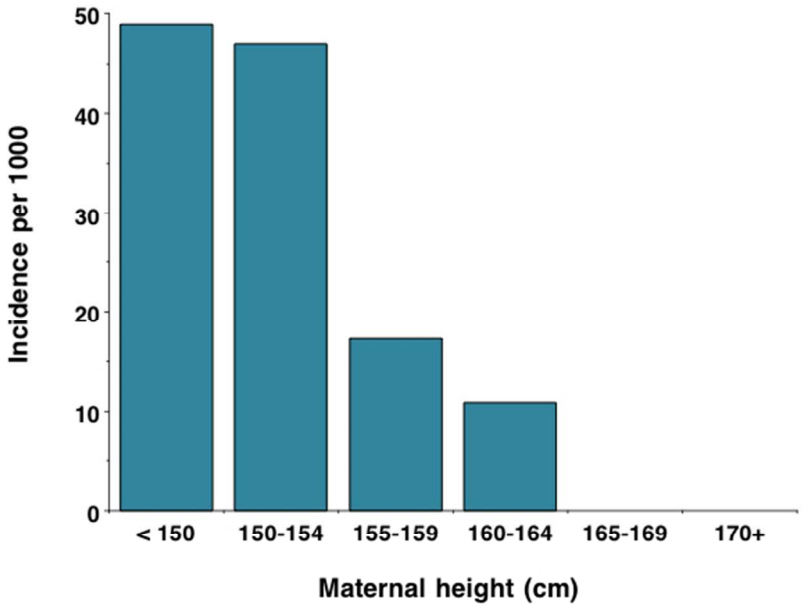

Figure 2. Associations between incidence of intrapartum cesarean per 1000 deliveries and maternal height, in women from Ouagadougou, Burkina Faso. Based on data of (Sokal, Sawadogo et al. 1991).

254x190mm (72 x 72 DPI)

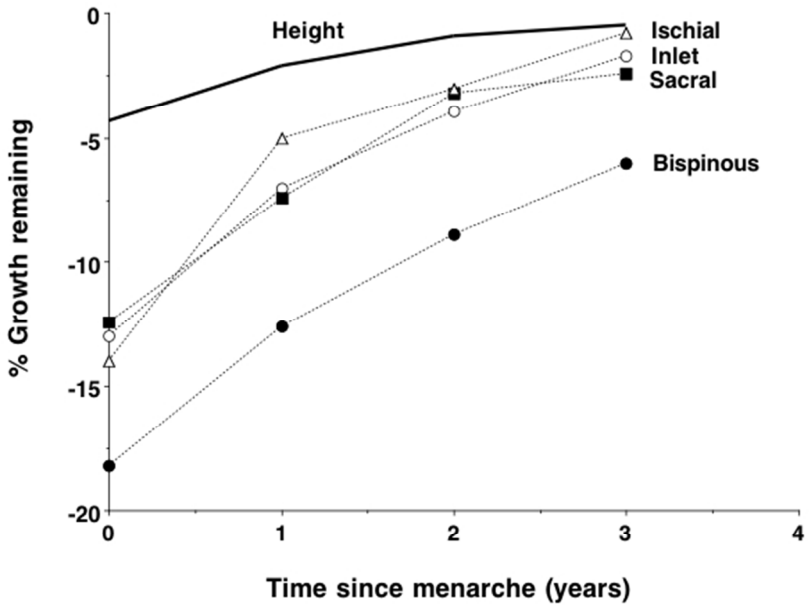

Figure 3. Growth remaining in stature and four pelvic dimensions (inlet diameter, interspinous diameter, maximum breadth of the sacral alae, and inferior breadth of the ischial tuberosities. in the period post-menarche, showing the relative delayed growth of the pelvis relative to stature. All data indexed to size at 18 years. Based on data of Moerman (1982).

254x190mm (72 x 72 DPI)

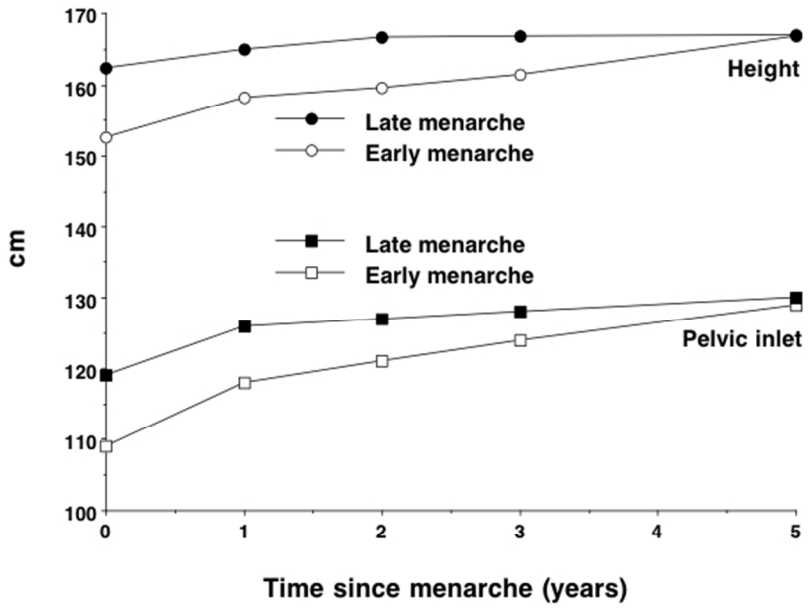

Figure 4. Growth in stature and diameter of pelvic inlet following menarche, stratified by early versus late menarche girls, showing the relatively smaller dimensions in girls experiencing early menarche. Based on data of Moerman (1982).

254x190mm (72 x 72 DPI)

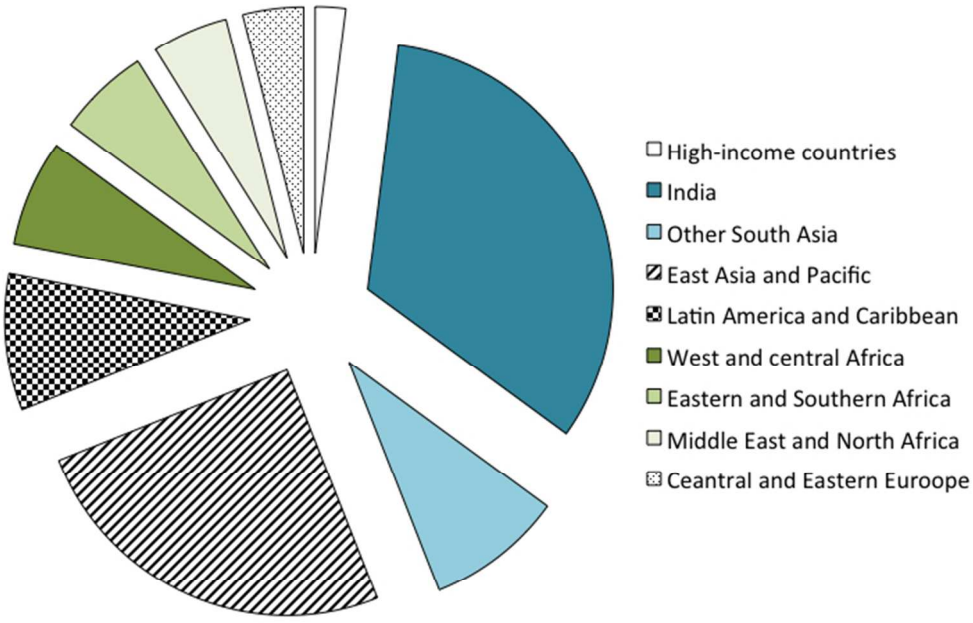

Figure 5. Regional distribution as percentage of global total of 700 million of women aged 18+ years who were married or in union before the age of 18 years. Based on data of UNICEF 2014.

254x190mm (72 x 72 DPI)

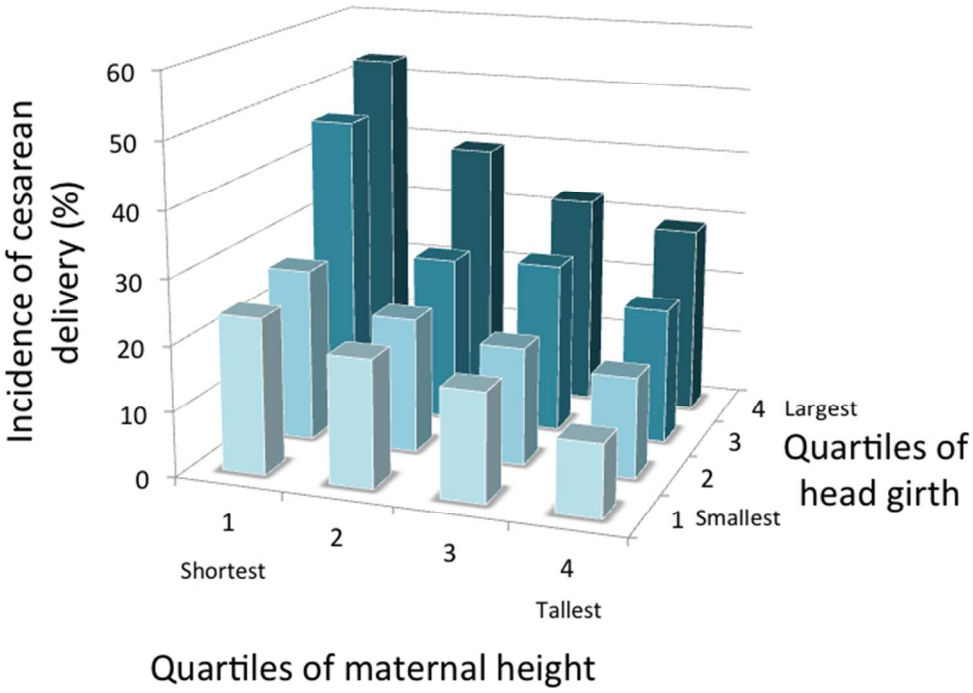

Figure 6. Associations between incidence of intrapartum cesarean delivery and maternal height, stratified by neonatal head girth. Data from Merchant et al., 2001.

254x190mm (72 x 72 DPI)

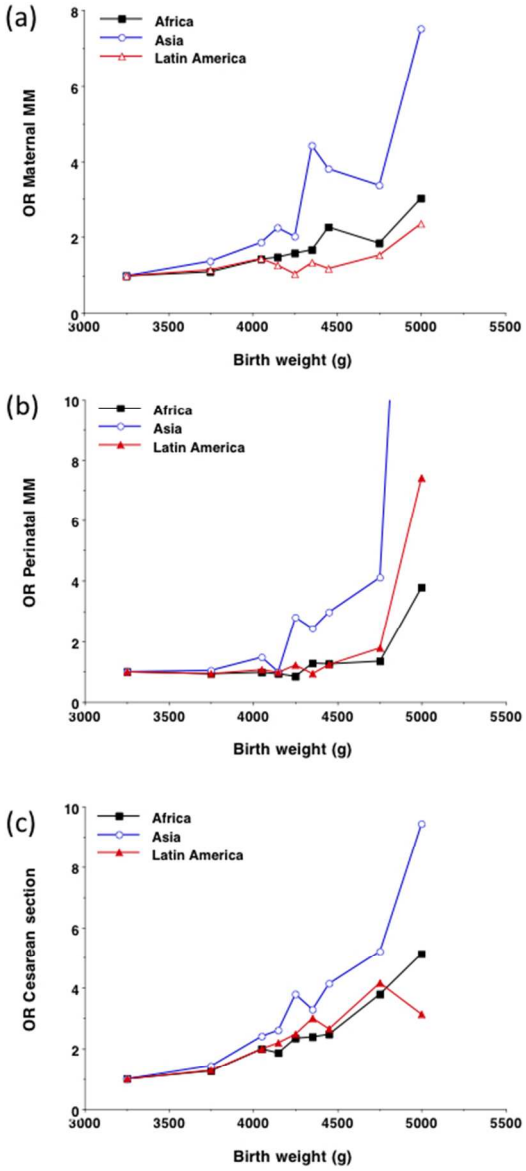

Figure 7. Associations between neonatal birth weight categories and the risk of (a) maternal mortality and morbidity, (b) perinatal mortality and morbidity and (c) intrapartum cesarean section in low- and middle-income countries, stratified by global region. Based on data of Ye, Torloni et al. 2015.

254x338mm (72 x 72 DPI)

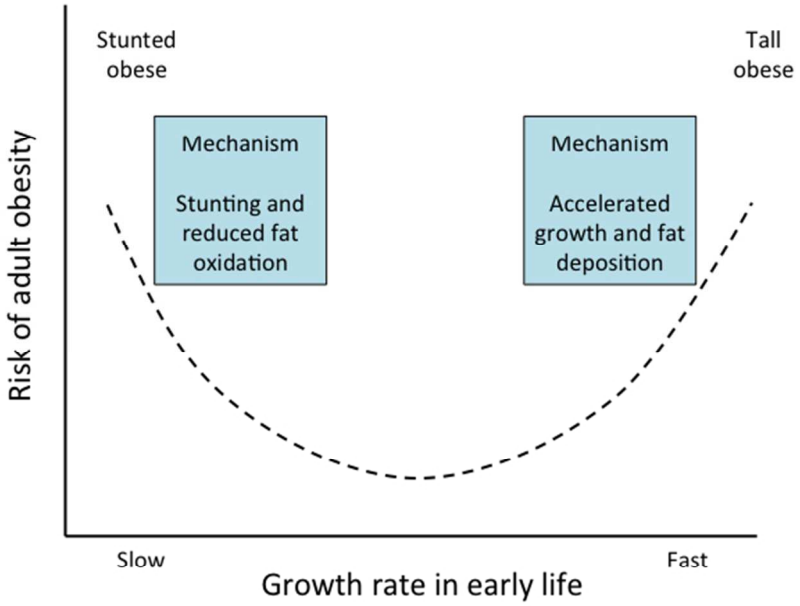

Figure 8. Schematic diagram of the U-shaped association between growth in early life and risk of obesity in adult life. Populations therefore differ in whether tall or short stature is a risk factor for cephalo-pelvic disproportion.

254x190mm (72 x 72 DPI)

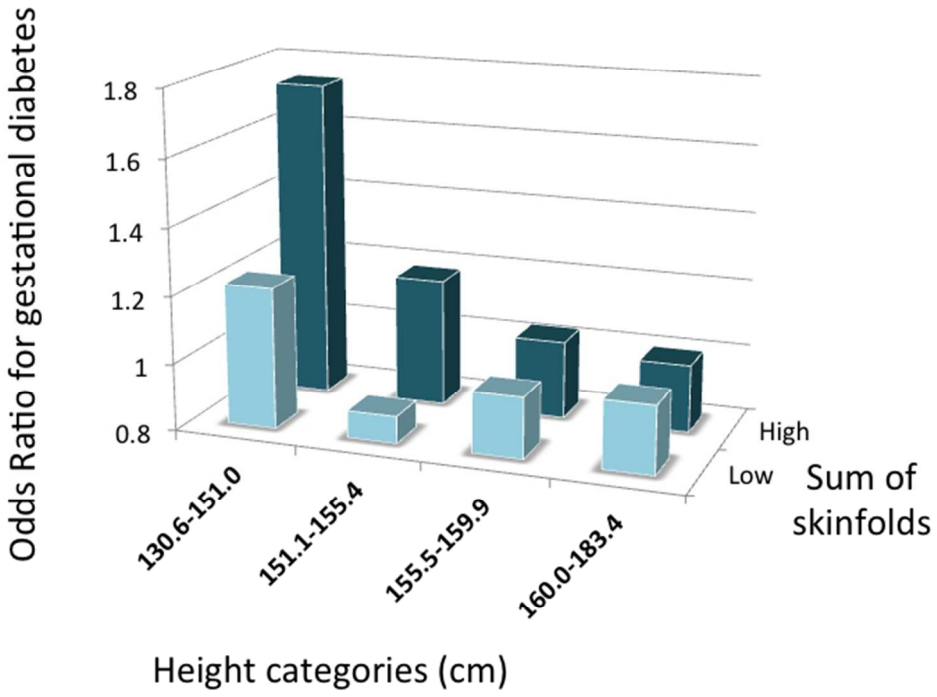

Figure 9. Association between the odds of gestational diabetes and maternal height, mediated by sum of skinfolds (high/low, categorized as above/below below average) in women from 6 state capitals in Brazil. Based on data of Branchtein et al., 2000.

254x190mm (72 x 72 DPI)

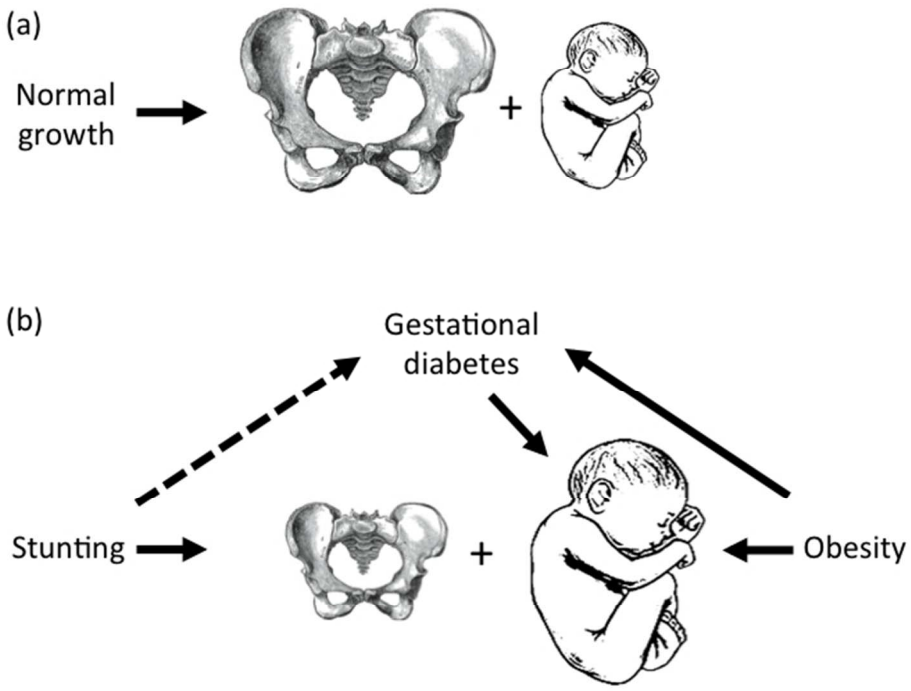

Figure 10. Schematic diagram illustrating the associations of maternal short stature and obesity with the risk of obstructed labour, mediated by associations of maternal stature and obesity with gestational diabetes.

254x190mm (72 x 72 DPI)

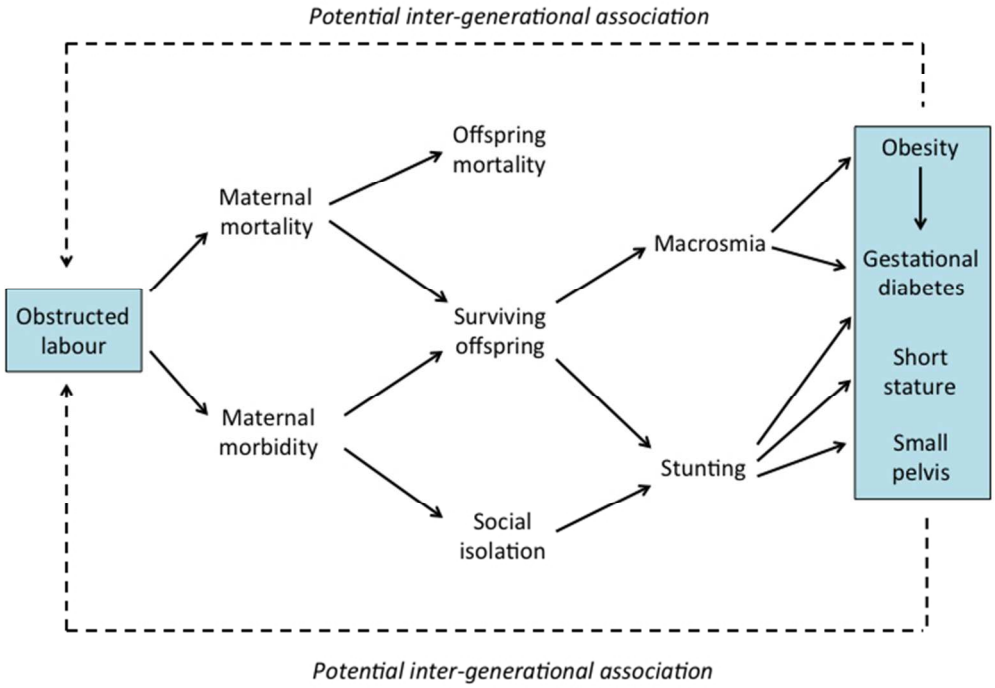

Figure 11. Schematic diagram illustrating potential inter-generation associations in relation to obstructed labour, whereby the phenotype of offspring exposed to this experience in early life may increase the risk of obstructed labour recurring when they themselves deliver offspring.

254x190mm (72 x 72 DPI)