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Evolutionary perspectives on human height variation

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ABSTRACT

Human height is a highly variable trait, both within and between populations, has a high heritability, and influences the manner in which people behave and are treated in society. Although we know much about human height, this information has rarely been brought together in a comprehensive, systematic fashion. Here, we present a synthetic review of the literature on human height from an explicit evolutionary perspective, addressing its phylogenetic history, development, and environmental and genetic influences on growth and stature. In addition to presenting evidence to suggest the past action of natural selection on human height, we also assess the evidence that natural and sexual selection continues to act on height in contemporary populations. Although there is clear evidence to suggest that selection acts on height, mainly through life-history processes but perhaps also directly, it is also apparent that methodological factors reduce the confidence with which such inferences can be drawn, and there remain surprising gaps in our knowledge. The inability to draw firm conclusions about the adaptiveness of such a highly visible and easily measured trait suggests we should show an appropriate degree of caution when dealing with other human traits in evolutionary perspective.

Key words: height, human, natural selection, sexual selection, evolution, ecology, development, genetics, environment, reproductive success.

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> 'Man alone has become a biped; and we can, I think, partly see how he has come to assume his erect attitude, which forms one of his most conspicuous characters'
>
> ~ Charles Darwin (1871)

I. INTRODUCTION

The shortest adult in recorded history is Chandra Bahadur Dangi, a 73-year old Nepalese man, who stands only 55 cm high. This makes him approximately the size of one of Robert Wadlow's feet, the tallest person ever to have lived. Wadlow reached a height of 2 m 72 cm, and was (allegedly) still growing at the time of his death, aged 22 (www.guinnessworldrecords.com). These extremes in height are clearly the result of pathological growth conditions, but natural variation in stature is almost as impressive: the Efe hunter-gatherers of the Congo, for example, barely reach 150 cm on average, whereas the Dutch are, on average, 35 cm taller, reaching an astounding 185 cm. Even within populations, substantial variation exists: the heights of the shortest 5% and tallest 5% of Dutch people also differ by approximately 30 cm (McEvoy & Visscher, 2009). Not surprisingly, height is one of the first things we notice about our fellow humans; indeed, as an upright walking mammal, height may be our most conspicuous feature.

Information on height within and between human populations is readily at our disposal because height is such an easy trait to study, being both quick and simple to measure accurately. More importantly, height is a trait of great biological significance: it is highly informative about growth rates, life-history trajectories, and health status at both the individual and population levels. Indeed, it is so reliable in this respect that it can be used as a proxy for social and economic conditions. It is perhaps no coincidence, then, that height has also been shown to have strong psychological influences on our behaviour, affecting perceptions of attractiveness, dominance and power, as well as influencing our choice of mates. For those interested in an evolutionary approach to human biology and behaviour, this wealth of information means that height is an excellent trait to study; one that lends itself to an integrative analysis, and where we can potentially measure natural selection in action.

In what follows, we attempt a comprehensive overview of studies of human height in evolutionary perspective: its variation across space and time, both historically and phylogenetically, aspects of its development, how environmental and genetic resources influence the patterns seen, how height relates to human life history and, finally, its influence on reproductive success. Our aim is not only to illustrate the value of an evolutionary perspective, but also to attempt an answer to the question: is height a trait with current adaptive value, or is height merely the incidental outcome of other processes more directly related to survival and reproduction? A greater understanding of the evolutionary significance of height is not only interesting in itself but, because of the interplay of economic, environmental and societal factors with biological factors (e.g. Hatton, 2013), it also helps to illustrate how culture is integral to any analysis of human behaviour from an evolutionary perspective.

II. VARIATION IN HUMAN HEIGHT

(1) Spatial variation in body size and height

Over the course of evolutionary history, humans have colonized almost every habitat, facing a variety of ecological stressors. The striking variation in body size and height across human populations has led researchers to ask whether this represents an adaptive response to environmental conditions, with thermal stress identified as a key influence (see Fig. 1 for the global distribution of female height). That is, humans are thought to conform to Bergmann's rule (body size increases with decreasing mean temperature, which produces a smaller surface area:volume ratio and so conserves heat) in much the same way as other homeotherms [Bergmann, 1847; but see Geist (1987), who argues that Bergmann's rule is invalid, and that body size differences actually reflect the manner in which the growing season influences energy availability during growth]. Body size can be interpreted in a number of ways, of course, and need not refer to stature alone. Indeed, studies of Bergmann's

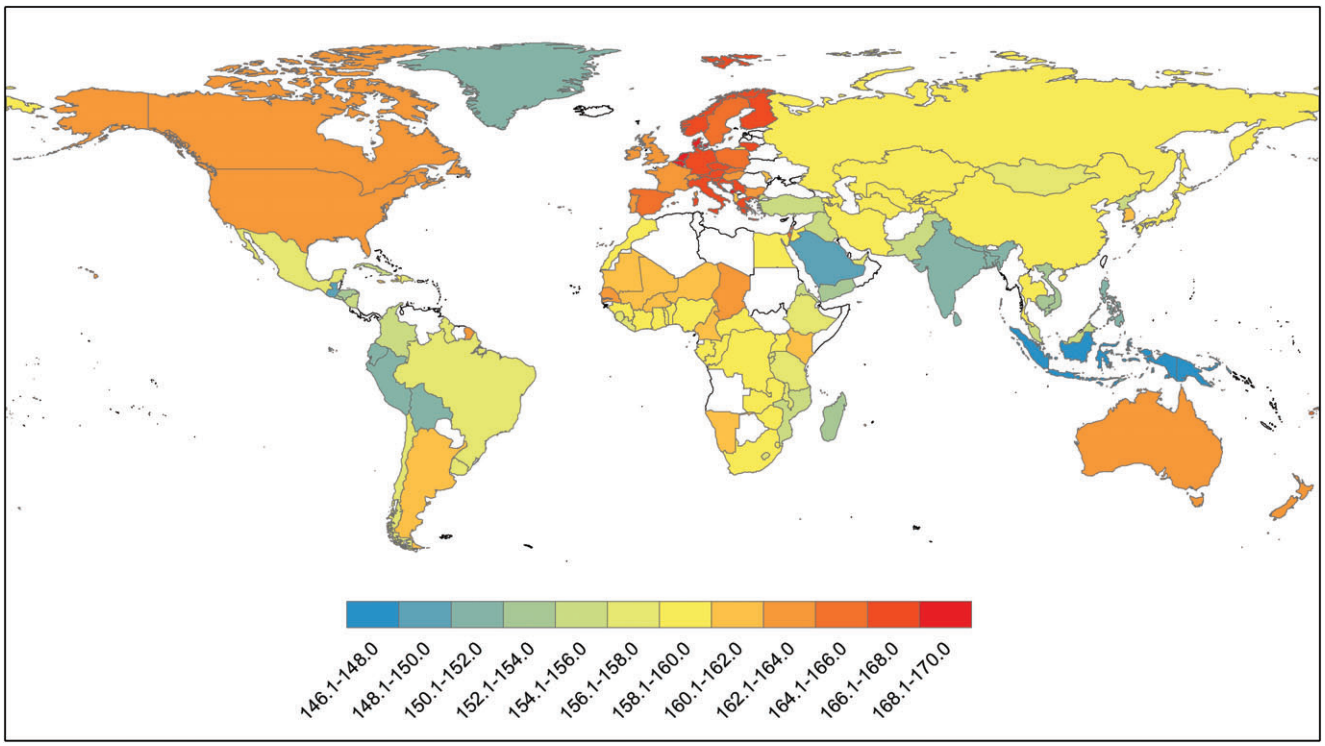

Fig. 1. Average female height across the world. Data vary with respect to the age at which height was measured and the sample year. Consequently, the map should be considered as merely illustrative of general patterns of height, rather than definitive. Colours indicate average height per 2-cm bin. Map drawn using Natural Earth (naturalearthdata.com) and ArcGIS 10.1. SP1, ESRL, Redlands, CA, USA. Data sources: The Demographic and Health Surveys (http://dhsprogram.com/; DHS data access: http://www.statcompiler.com/; average female height for women between ages 30 and 35 at the last sampling round); http://en.wikipedia.org/wiki/Template:Average_height_around_the_world (sources provided in link); Gustafsson & Lindenfors (2004).

rule have used a variety of measures, including body mass, surface area:volume ratio, body mass index (BMI), biiliac breadth (the distance between the protruberances of the hip bones on either side of the abdomen) and ponderal index (body mass divided by the cube of height). Studies also vary in whether temperature or latitude is used as the climatic variable of interest. Most studies show at least some support for Bergmann's rule, although results are mixed and effect sizes tend to be moderate at best (e.g. Schreider, 1950; Roberts, 1953, 1978; Ruff, 1991, 1993, 1994; Katzmarzyk & Leonard, 1998; Gustafsson & Lindenfors, 2009; Foster & Collard, 2013; Hadley & Hruschka, 2014). Furthermore, the most recent, and the most well-controlled, study demonstrated that Bergmann's rule holds only for humans in the Northern hemisphere, and not the south (Foster & Collard, 2013). Habitable regions of the Southern hemisphere arguably do not cover a sufficiently large temperature range to allow thermoregulation-related natural selection to exert a dominant influence on human body size.

With respect to height specifically, the classic study by Roberts (1953) found a significant correlation between mean annual temperature and height, but also showed that this relationship disappeared when controlling for body mass. Similarly, Ruff (1994) found that the relationship between stature and latitude, as well as latitude and mass, became non-significant when body breadth was taken into account (indexed by biiliac breadth). By contrast, the relationship between biiliac breadth and latitude remained significant when stature and mass were controlled. Ruff (1994) concluded that body breadth, rather than height, was the variable under climatic selection (as breadth will always affect the surface area: volume ratio, whereas height would not: see also Ruff, 1993, 2002).

As Gustafsson & Lindenfors (2009) point out, neither Roberts (1953) nor Ruff (1994) controlled for potential collinearity between the morphological variables used in their studies, nor did they correct for shared ancestry among their samples. When correcting for these factors, and using a larger sample, Gustafsson & Lindenfors (2009) found a significant, albeit weak, relationship between latitude and height for both sexes, and they also tested for a curvilinear relationship, finding a peak in stature at around 40° latitude from the equator. This was suggested to reflect higher standards of living, and hence nutritional influences on height, in these regions. The relationship between height and latitude may also reflect the fact that humans tend to conform to Allen's rule (Allen, 1877), which states that body shape and proportions vary by minimizing exposed surface area in relation to decreasing mean temperature (to reduce surface area: volume ratio, and so conserve heat) and maximizing exposed

surface area with increasing mean temperature (increasing surface:volume and so improving heat dissipation). Roberts (1978), for example, showed that men from populations living in warmer regions tended to have longer limbs than those living in colder areas, while Katzmarzyk & Leonard (1998) found that relative sitting height was negatively correlated with temperature in both sexes, indicating that more tropical populations have a more linear body build.

Taken together, these studies provide some evidence to suggest that spatial variation in height across populations reflects past selection on morphology in relation to climatic factors, although as noted, results are mixed. This is not surprising given that the relationship is complicated by the fact that humans use unique forms of behavioural thermoregulation, like clothing and shelter, to deal with climatic stress (Gustafsson & Lindenfors, 2009), as well as the influence of other ecological and nutritional factors that affect body size and stature (Ruff, 1994, 2002; and see Section II.2*b*).

(2) Temporal variation in height

(*a*) *The ups and downs of human height evolution*

Changes in height and limb proportions also characterize the deep history of the human species, with a general consensus that early *Homo* species, in particular *H. erectus* (∼1.9 to 0.9Mya), were approximately one-third larger, with more human-like limb proportions, than the earlier, more ape-like australopithecine species (∼4 to 1.8 Mya) (Pontzer, 2012). These changes in body proportions and stature, along with an increase in brain size, are argued to reflect a shift in ecological niche in response to climatic change. Unlike the australopithecines, which subsisted primarily on plant foods, early *Homo* is considered to have been an active scavenger and possibly a persistence hunter of animal prey, i.e. activities that made more intense locomotor and cognitive demands, selecting for longer legs and a fully bipedal posture (Bramble & Lieberman, 2004). Ruff (1993) also suggests that a taller, more linear frame would be selectively advantageous with respect to thermoregulation, which fits with the evidence from contemporary populations described above.

Early estimates made from the almost complete skeleton of a young *H. erectus* male, known as 'the Nariokotome boy', suggested a very tall adult height of 1.85 m (Ruff & Walker, 1993). Recently, however, Graves *et al.* (2010) have argued that this value should be revised downwards to around 1.52–1.79 m, based on a reassessment of the life-history trajectory of *H. erectus*. This is in line with other findings of early *Homo* in Dmanisi, Georgia, where stature has been estimated at 1.56 m (Lordkipanidze *et al.,* 2007), and the recent analysis of a complete female *H. erectus* pelvis from Ethiopia, dated to between 1.4 and 0.9 Mya, which suggests a standing height of 1.2–1.4 m (Simpson *et al.,* 2008). On the one hand, these values suggest variation in height comparable to that seen within and between modern human populations. On the other, they may indicate methodological problems: the accuracy of the new estimates has been disputed, for example, and the species designation of the female pelvis has been questioned (Ruff, 2010). Nonetheless, even if the revised heights are confirmed, it remains the case that *H. erectus* stood taller than the australopithecines, with most authors agreeing that taller stature marks the appearance of early *Homo* in the fossil record (Graves *et al.,* 2010; Antón & Snodgrass, 2012).

Further large increases in body size, stature and brain size are seen among Middle Pleistocene *Homo* [780000 to 138000 years before present (BP)] in Africa and, as with the emergence of early *Homo,* are thought to reflect the impact of climatic factors on foraging strategies. Specifically, environmental changes increased the abundance of ungulate prey, which required a shift to more organized hunting and greater terrestrial mobility in order to capitalize on this food source. There is evidence to suggest that *Homo* species not only increased the size of their home ranges, both annually and seasonally, but also made their first movements beyond the African continent at this time (Gallagher, 2013). These ecological changes are linked to an increase in stature among the fossils of this era. *Homo antecessor*, from North-Central Spain, for example, is estimated to have stood around 1.73 m tall, close to mid-20th century European males (Gallagher, 2013). More generally, Gallagher (2013) gives values of between 1.50 and 1.90 m for early to late middle Pleistocene *Homo* species (*Homo heidelbergensis* and *Homo rhodesiensis*), and Graves *et al.* (2010) provide similar estimates. Values at the upper end of this range therefore exceed those of most contemporary human populations (Gallagher, 2013). Tall stature is also a feature of anatomically modern humans, *Homo sapiens*, from the Early Upper Palaeolithic (38000 to 22000 BP) in Europe. Heights are estimated to be 1.74 m for males, and 1.59 m for females (Gallagher, 2013). It is also worth noting that this trend of increasing stature did not occur gradually over the course of the previous 2 million years of human evolution but, as with brain size, was characterized by brief bursts during particular time periods and geographic locations; periods that coincided with marked shifts in both ecology and behaviour, as documented in the archaeological record (Gallagher, 2013).

This pattern is also true for the decline in stature that is seen during the Late Upper Palaeolithic (18000 to 9800 BP). Male height dropped by approximately 10 cm during this period, to an average of 1.64 m. Indeed, tall males (i.e. those with heights exceeding 1.80 m) more or less disappear from the fossil record at this time (Gallagher, 2013). This decline in height has been tied to the rise in agriculture (Larsen, 1995; Mummert *et al.,* 2011). Across 14 populations distributed worldwide, Mummert *et al.* (2011) found that a shift to agriculture resulted in a decline in height, regardless of the temporal period in which agriculture was adopted. The reasons why this should be are many and varied: agriculture results in greater variability in food availability across the year, with periods of poor nutrition—'hungry seasons'—experienced regularly while the next season's crop grows. Agricultural societies are also more vulnerable to events that result in periods of poor nutrition, such as crop failure, compared to other forms of subsistence. Finally, agricultural societies are associated with increased population

density and hence higher rates of infectious disease, which is known to have a negative influence on height (Mummert *et al.*, 2011).

From this, it is apparent that Pleistocene and early Palaeolithic *Homo* were not only taller than early *Homo* species, but were taller than both medieval and many contemporary human populations (Ruff, Trinkaus & Holliday, 1997; Mummert *et al.*, 2011; Gallagher, 2013). The secular trend of increasing height over the last century (see Section II.2*b*) must therefore be placed in the context of these broader evolutionary and historical patterns of human height variation. Although our immediate medieval ancestors were much shorter than many contemporary humans, this is not true of our more distant relatives, and the heights reached by some contemporary human populations are not, therefore, unprecedented in the history of our species. This suggests that some modern populations have yet to reach the upper limit of human height potential.

(*b*) *The modern secular trend in height*

Evidence from modern populations reveals quite clearly how humans have recovered from the 'agricultural low' in height, and are moving back toward those reached by Palaeolithic populations. The process by which entire populations undergo a change in mean size or shape across generations is known as a secular trend. The trend toward increasing height, which began in the middle of the last century, is found across the globe, and observed in both developed and developing countries, although the onset of the trend may vary (Hauspie, Vercauteren & Susanne, 1996, 1997; Cole, 2000, 2003; Hatton, 2013). Almost all of this increase is established in childhood, and some argue this is due to an increase in leg length. Tanner *et al.* (1982), for example, demonstrated that most of the secular increase in height observed in a Japanese sample could be attributed to increased leg length, and Malina *et al.* (2004) found that 60% of the secular height increase in both boys and girls could be attributed to longer legs in a population from rural Oaxaca, Mexico. Cole (2000, 2003) has argued, on the basis of the Japanese data, that secular changes in height have their genesis during the first 2 years of life: Japanese children aged 2 were 4 cm taller in 1990 than in 1950, and the same height difference was true for young adults, which suggests that the adult trend in height is already in place during the very earliest phase of childhood. As this is the period during which stunting manifests under poor environmental conditions, Cole (2000) argues that the secular height trend can, in effect, be viewed as a reduction in the degree of stunting. The secular change in height has also been accompanied by a secular increase in the tempo of growth, such that full adult height is reached at an earlier age: Hauspie *et al.* (1996) report that most European males now reach their full height at 18 years compared to 26 years in the early 1900s.

An increased standard of living as an explanation for these patterns is well supported by the strong correlation between increases in height and increases in gross national product (Floud *et al.*, 2011), child survival (Bozzoli, Deaton & Quintana-Domeque, 2009), and life expectancy (Steckel, 2002*a*,*b*) within a population. This in turn reflects several important changes in public health, including an understanding of the germ theory of disease, increased personal hygiene, better health care for children, including vaccinations, and improved diet (Steckel, 1995, 2009). Indeed, the secular trend in height is so reliable that increases in height are widely used by historians and economists as a proxy for a nation's development and welfare levels (Floud *et al.*, 2011). This approach should be used with caution, however, as it does not seem to apply to all African height trends (Deaton, 2007; Steckel, 2009).

Although economic prosperity and stability are undoubtedly important factors in cross-country comparisons, they cannot account for the major height differences that exist between populations of equivalent wealth. The people of the Netherlands provide a particularly noteworthy example in this respect. In 1860, the average height for Dutch (military) men was 165 cm, which was lower than many other Western populations. From the 18th to the early 20th century, for instance, men from the US ranked number one in terms of average height (Komlos & Baur, 2004), towering over the Dutch by around 5–8 cm. One hundred and fifty years later, Dutch men now rank as the tallest in the world, with an average height near 185 cm; an increase of 20 cm. This increase is echoed across the whole of Western Europe, albeit less dramatically; Hatton (2013) documents an increase of 11 cm in male height from the 1850s to 1980, although there is wide variation across different countries. This increase in stature has been continuous and regular, with the exception of the periods covered by the two World Wars (Liestøl & Rosenberg, 1995; Silventoinen, 2003). By contrast, the heights of US males have increased by only 6 cm during this same period (Komlos & Baur, 2004). Economic prosperity is not sufficient to explain the height discrepancy between the Dutch and the North Americans because the latter actually have a higher *per capita* income, invest more in health care, and have a similar caloric intake to the Dutch. Part of the difference in average height can, however, be attributed to differences in social equality: despite its higher *per capita* income, social inequality is much higher in the USA than in the Netherlands, and the health care system is not equally accessible to everyone. Greater equality in access to resources increases the average height of a population because the greater increases in height by the poor can counterbalance, or even outweigh, the stagnation in height of the wealthy (i.e. there are diminishing returns on height with increasing wealth) (Blum, 2013). Put differently, a much smaller fraction of the population is exposed to environmental influences that can result in stunting and reduced adult height when resources are more equitably distributed across socio-economic strata, thus pushing up the average height of the population as a whole.

Equivalent differences also exist for countries that lie in much closer proximity. Although in most Northern European countries, the secular trend in height has been slowed or stopped (Hauspie *et al.*, 1997; Garcia &

Quintana-Domeque, 2007), the Dutch, apparently not yet tall enough, keep on growing (or, at least, have yet to show any sign that the trend is slowing down: Fredriks *et al.*, 2000; Danubio & Sanna, 2008; but see Schönbeck *et al.* 2012). The reasons for Dutch superiority in height have been related to subtle variations in nutrition and health care. Social welfare, the medical system and universal health care are all of a very high standard in the Netherlands, and Dutch mothers are also known to make good use of the health care system (de Beer, 2004), all of which are likely to have substantial consequences for stature. It has also been suggested that the type and quality of food eaten by the Dutch may give them an anthropometric advantage: the Dutch consume a high proportion of dairy products, particularly milk (Fredriks *et al.*, 2000; de Beer, 2012). Similarly, the striking secular trend in height among the Japanese has been attributed to an increase in milk-drinking following the second World War (Takahashi, 1984). Thus, subtle variations in health care and nutrition between countries, which are not caught under the umbrella of general economic prosperity, probably account for much of the variation in height that exists between populations.

These shifts in average height over historical time in relation to resource availability also have implications for tests of evolutionary hypotheses concerning spatial variation in body size, such as Bergmann's rule described above. Katzmarzyk & Leonard (1998), for example, replicated the findings of Roberts (1953) study on body size and latitude, conducted 40 years previously, but found a much weaker relationship. As their study was also able to demonstrate a secular trend in body size among the Southern populations in their sample, Katzmarzyk & Leonard (1998) therefore suggested that improved nutrition, and hence increased average body size among these populations, could account for the weaker relationships found. This in turn suggests that at least some of the variation attributed to evolved differences in response to climate may actually reflect a developmental response to nutritional stress.

III. DEVELOPMENT OF HEIGHT AND SEXUAL DIMORPHISM

(1) Height across development

Having considered height variation in both time and space, it now becomes pertinent to consider growth patterns across development, given that shifts in growth rates and developmental timing are the mechanism by which evolutionary and historical changes in height are produced. An understanding of how height differences emerge during ontogeny also provides the background for exploring patterns of sexual dimorphism in height. In what follows, we draw mainly on Bogin's (1999*b*) comprehensive review of human growth, and interested readers can follow up in more detail by referring to this work, as well as Tanner (1962, 1990), or the more concise summaries found in Bogin (1999*a*, 2010).

One of the most prominent differences in the human growth curve compared to other mammals is that humans achieve their maximal rate of growth (in terms of both length and mass) during gestation, and growth rates then decelerate during infancy relative to other mammalian species. One reason why this should be is that humans generally carry only a single fetus, rather than a litter, which means faster rates of growth are possible during gestation (Bogin, 1999*a*, 2010). Once born, however, growth and maturation proceed much more slowly, with puberty and sexual maturation delayed by many years. This is true not only in relation to most other mammals, where sexual maturity is reached soon after weaning, but also in relation to other primate species. All primates show a significant juvenile growth period. In humans, however, this period is greatly prolonged (Bogin, 1999*a*, 2010; Leigh, 2001).

Puberty in humans is reached at the point when growth rates are at their lowest since birth. The adolescent growth spurt, considered to be a unique characteristic of human growth, is thus triggered by the hormonal changes that occur at puberty. This happens in both males and females, and is generally found in all human populations (Bogin & Smith, 1996; Bogin, 1999*a*; Gluckman & Hanson, 2006; but see below for some possible exceptions). Whereas other primates, like rhesus macaques (*Macaca mulatta*), show a brief increase in growth velocity at or around sexual maturity and sometimes continue to grow subsequently, humans experience a single, absolutely large and unambiguous growth spurt that follows the onset of puberty. In addition, the human adolescent growth spurt lasts for years, rather than months. Humans also differ by showing a more distinct sex difference in the expression of the adolescent growth spurt (Bogin, 1999*a*): women both start and stop their growth spurt at an earlier age than men, thus reaching their final height sooner. Indeed, many of the sex differences seen in adult body size and shape arise as a consequence of these differential growth patterns at adolescence (Tanner, 1962, 1990). Both the later occurrence of the male spurt, allowing an extra period of growth for boys, and the greater intensity of the spurt itself, results in higher levels of sexual size dimorphism in adulthood compared to earlier life-history stages (Tanner, 1962; Bogin, 1999*a*).

As the above description highlights, growth ontogeny is not a smooth progression through time, but is inherently dynamic, proceeding in a series of spurts (basically, changes in growth velocity). The timing and patterning of these spurts in humans – during gestation, middle childhood (around 6 years) and during adolescence – differs markedly from most other mammals, including other species of primate (Bogin & Smith, 1996; Bogin, 2010). The variable patterns of growth seen across species, in both the number and timing of growth spurts, has led to the suggestion that such spurts, by virtue of their modular nature, are highly evolvable features of ontogeny; the placement and timing of growth spurts across development may thus reflect the outcome of natural (and potentially sexual) selection (Bogin, 1999*a*).

It should also be apparent that factors that exert an influence on height, like periods of nutritional stress or the incidence of infectious disease, will do so to a greater or lesser extent depending on their timing in relation to

the human growth cycle. Adult stature in 18th and 19th century America and Europe, for example, was strongly associated with economic factors, like grain prices, with the lag observed between cycles suggesting that poor economic conditions during the early growth phase exerted the most significant influence on adult stature (Woitek, 2003). This effect was argued to be cumulative because of the way in which subsequent economic cycles mapped onto the human growth cycle: children born during a recession would experience subsequent economic downturns during both the middle childhood growth phase and again during the adolescent growth spurt, compounding the effects of poor growth during infancy (Woitek, 2003). It seems possible that patterns like these, which illustrate the sensitivity of growth to economic conditions, can also shed light on inter-population height differences in the present day: lower economic stability in general, and hence larger fluctuations in economic conditions over time, may explain both the finding that populations in developing nations tend to be shorter than those in the developed world, and that variation in height across social classes is known to be greater in poorer countries (Silventoinen, 2003; Deaton, 2007), but much reduced where standards of living are higher (Garcia & Quintana-Domeque, 2007). Such findings may also help explain the negative secular trends in height seen in some parts of Africa, India and South America (Malina, 2004).

If, as Cole (2000) suggests, we can view secular height trends as a reduction in stunting, it should follow that understanding the causes of stunting will also shed light on patterns of increased growth across time. This is aided by the fact that, as Walker *et al.* (2006) point out, most bioanthropological studies of human growth have focused on the impact of constraints on growth, with an emphasis on the effects of nutrition. Indeed, lack of nutrition, particularly protein deficiency, is recognized as one of the main contributors to reduced infant growth in developing countries, and supplementary food programs have been shown to produce clear improvements in growth rates and reduced stunting (Edozien, Khan & Waslien, 1976; Beaton & Ghassemi, 1982; Silventoinen, 2003). Due to the strong association between growth and nutrition, both UNICEF and the World Health Organization collect growth data as a means to assess the success of its supplementary food programs and to establish indicators of malnutrition (De Onis *et al.*, 1993; Steckel, 1995). The positive effects of supplemental feeding are obviously not restricted to developing nations, however; experiments conducted as long ago as 1928 showed that the provision of extra milk increased the growth of schoolchildren in the UK (Orr, 1928; Leighton & Clark, 1929). Indeed, even in contemporary populations, drinking milk can substantially increase height (de Beer, 2012).

In addition to, and intertwined with, the effects of malnutrition, childhood disease is known to adversely affect growth: mounting an immune response to fight infection increases metabolic requirements and can thus affect net nutrition, and hence productivity. Disease also prevents food intake, impairs nutrient absorption, and causes nutrient loss (Silventoinen, 2003). Accordingly, recurrent infection is associated with a lower height-for-age (Dowd, Zajacova & Aiello, 2009). Adverse economic and social conditions in childhood, such as psychosocial stress, housing conditions and physically strenuous work, also lead to short stature in adulthood (Mascie-Taylor, 1991; Peck & Lundberg, 1995; Cavelaars *et al.*, 2000). Family size can also be a risk factor (Rona, Swan & Altman, 1978; Lawson & Mace, 2008): the presence of (many) siblings, for instance, significantly reduces an individual's height (presumably because finite resources have to be distributed across all children, which leads to both reduced nutrient intake and increased psychosocial stress) (see also Hatton & Martin, 2010). Even in a wealthy, well-nourished population like the UK, and adjusting for family-level socioeconomic factors, those raised with four siblings are, on average, 3 cm shorter than those born without siblings (Lawson & Mace, 2008). Perhaps the best illustration of the dramatic plasticity in growth, however, comes from studies comparing the offspring born to those who emigrated to more affluent populations with those remaining in their less-affluent native population (Kim, 1982; Bogin *et al.*, 2002). Maya children born in the USA, for instance, are over 10 cm taller than Maya children born in Guatemala (Bogin *et al.*, 2002). Many of the above patterns, including the secular trend in height and height differences between individuals and populations, have also been attributed to differences in microbial transmission (Beard & Blaser, 2002). Interactions with microbes influence not only levels of infection, but also affect the colonization of the gastrointestinal tract by microbes that assist with digestion (the microbiota), both of which may act to reduce adult height.

(2) Why grow up, rather than out?

Growing taller over the course of childhood is something we take entirely for granted; we simply accept that adults are taller than children. One can, however, pose the question as Ellison (2009) does: why do we grow up, rather than out? That is, why is metabolic effort expended on skeletal development, rather than increasing body mass, whether by storing fat or building muscle? Given the costs of reproduction, for females in particular, larger body mass would seem more advantageous than taller stature. Yet, skeletal growth, which occurs mainly in the long bones of the limbs, followed secondarily by the spine, pelvis and skull, is more tightly canalized than increases in body mass, and is highly buffered against short to medium term variation in energetic conditions (Bogin, 1999*b*). In addition, skeletal growth is much less plastic than growth in body mass: although bone can be resorbed, and the skeleton provides an important reservoir of minerals like calcium, this process does not cause the long bones to shrink as a consequence (Ellison, 2009). Overall then, as Ellison (2009) notes, height is given much greater metabolic priority than mass: our physiology seems designed to sacrifice growth in mass to preserve growth in height (at least in the short to medium term).

One plausible explanation for why this occurs is linked to the allied process of reproductive maturation: menarche in

girls usually occurs shortly after the peak of the adolescent growth spurt has been passed, which is not too surprising given that both are influenced by steroid hormones (Tanner, 1981; Ellison, 1990). Increased production of oestrogen slows the process of growth, and eventually causes it to cease, by stimulating mineral deposition in the growth plates at the ends of the long bones, thus terminating cell proliferation, and resulting in the fusion of the growth plates to the shaft of the bone. Increases in oestrogen also lead to the onset of menstrual bleeding. Thus, the change in hormonal profile that results in the slowing and eventual cessation of growth during the period from late teens to early adulthood is the same as that which results in the onset of sexual maturity. This suggests that skeletal maturity is, in some way, key to females' ability to reproduce successfully.

Ellison (2009) argues that it is not height *per se* that is crucial, but the fact that height indexes another aspect of skeletal size: namely, pelvic width. The size of the pelvic canal imposes an absolute mechanical constraint on the ability to give birth successfully among all primates, but this constraint is particularly pressing in humans due to the antagonistic selection pressures that give rise to the so-called 'obstetric dilemma': a widely accepted hypothesis is that selection for increased brain size, which served to increase the size of the neonate's head at birth, was countered by earlier selection for efficient bipedal locomotion. This re-shaped the pelvis and so narrowed the birth-canal, resulting in a situation where, relative to other primate species, a much larger-brained infant was now required to exit through a much narrower opening (e.g. Wittman & Wall, 2007). Although there has been selection on pelvic width and shape independently of height in females (particularly so in some small-statured populations: Kurki, 2007, 2011, 2013) this has been limited by the costs in terms of effective locomotion, with the result that height constrains reproductive functioning: females have to achieve a certain level of skeletal height because, to a large extent, they rely on the correlated growth of the pelvis to reach a width that is sufficient for the passage of an infant, without adversely affecting locomotion. A biiliac breadth of around 24–25 cm seems to be a good approximation of this 'pelvic size threshold.' The age at which this threshold is reached correlates strongly with age at menarche (Ellison, 1982, 1990, 2009). This, then, may help explain why skeletal growth is prioritized over adding muscle mass or storing fat: until the pelvis reaches a size at which birth is possible, there is no point to storing energy for future reproduction (Ellison, 1990, 2009). Gluckman & Hanson (2006) further suggest that there may have been positive selection on female height in the hominin line: larger brained offspring would require a wider pelvic canal, which would lead to selection for prolonged skeletal growth to attain the pelvic size threshold required, and hence to taller stature.

In line with this, it is well established that, across contemporary human populations, taller women experience fewer problems during labour and birth, and have a lower mortality risk (Wells, DeSilva & Stock, 2012), because of a lower risk of a mismatch between fetal head size and the size of the birth canal; the 'obstetric dilemma' referred to above. It is also important to realize, however, as Wells *et al.* (2012) demonstrate, that the dangers posed by the obstetric dilemma are not fixed, as is often supposed, but tend to shift over both time and space, reflecting phenotypic plasticity in maternal pelvic dimensions and, to a lesser degree, infant growth patterns. Changes in stature are therefore critical in this regard, as height is a trait that is sensitive to environmental conditions and, as we've noted, changes in stature and body proportions affect the dimensions of the pelvis. The pelvic size threshold should therefore be seen as the absolute minimum size at which birth becomes possible, and not as an optimum. Accordingly, factors like variation in thermal stress have been linked to the obstetric dilemma (Ruff, 1994). Under high thermal stress, where taller, narrower bodies are favoured, the obstetric dilemma may be exacerbated relative to less-stressed populations, especially as fetal mass contributes directly to maternal endogenous heat production, further increasing the pressure to retain a body size and shape that can effectively dissipate heat (Ruff, 1994; Wells *et al.*, 2012).

Reductions in nutritional status and the impact of infectious disease, both of which lead to smaller body size, can also be expected to increase the obstetric dilemma by reducing pelvic dimensions, and in the case of certain diseases of malnutrition, like rickets, by altering pelvic shape in ways that can impede birth (Wells *et al.*, 2012). Given that such reductions in stature characterize the shift to agriculture discussed above, it is thus possible that some of the increased mortality associated with this period reflects an intensification of the obstetric dilemma (Wells *et al.*, 2012). By the same token, recent secular trends in height across the developed world may have acted to alleviate it to some degree. This observation is, of course, complicated by technological interventions that can also reduce the impact of birth complications. We revisit the impact of stature on birth complications below, when we consider the extent and nature of current selection pressures on height.

Obviously, the pelvic size hypothesis does not apply to males but, equally obviously, boys also continue to grow for many years before reaching sexual maturity. In the case of males, skeletal growth is linked to increases in muscle mass and shoulder breadth at puberty, and Ellison (2009) speculates this may relate to male reproductive strategies in some way. Gluckman & Hanson (2006), for example, suggest that taller males would be stronger and hence more dominant, and may have been more strongly favoured as mates. If this kind of process did operate, then it may have pulled female height along in its wake (as the same genes operate in both sexes), thus amplifying the direct influence of natural selection on female height.

(3) Sexual dimorphism in height

Sexual dimorphism in body size is often taken as evidence for sexual selection. Among most mammals, larger male size often indicates competition by males for access to females (e.g. Fairbairn, Blanckenhorn & Székely, 2007). Larger males experience greater success in competing

against other males, and hence achieve higher fitness, which then leads to pronounced sexual dimorphism in size. The moderate degree of sexual dimorphism seen in humans (males are approximately 15% heavier and 9% taller than females on average) has been attributed to a history of mild polygyny and inter-male competition, as well as a variety of other factors (all of which remain under debate, with no consensus reached), including high levels of egalitarianism, phylogenetic inertia, and sex-biased parental investment.

Examining sexual dimorphism in deep evolutionary time, it is notable that changes in height among *Homo* species are accompanied by a decrease in sexual dimorphism (Frayer & Wolpoff, 1985). Gray (2013) notes that *Australopithecus afarensis* (4 to 3 Mya) males are believed to be around 44% bigger than females, whereas this figure is 26% for *Homo habilis* (2.4 to 1.6 Mya), 13% for *Homo erectus* (1.7 to 0.7 Mya), and a very moderate 9% for modern humans. European Upper Palaeolithic populations also show more body dimorphism than later Holocene populations, but they are less dimorphic than Middle Pleistocene populations (Frayer & Wolpoff, 1985). Frayer (1980, 1981) has suggested that the 'gracilization' of males reflects changes in prey assemblages, which became both smaller and less aggressive over time, and hence required reduced physical size and robustness to hunt and kill; the kinds of weapons used to kill prey changed also, with a shift from spears to bow and arrow, which requires less in terms of upper body strength (but see Wolfe & Gray, 1982; Holden & Mace, 1999). Gray (2013) adds to this by arguing that the egalitarian ways of band hunter-gatherers may have been a force in the reduction of sexual dimorphism.

A decrease in dimorphism has also been argued to reflect the shift to agriculture, where it indicates changes in the division of labour, with each sex engaging in more similar kinds of economic chores compared to hunting and gathering (Frayer, 1980). Ruff (1987), for example, documents a decline in the sexual dimorphism of the femur and tibia from the Middle Palaeolithic toward the present day, which is suggested to reflect a change to more similar mobility levels between males and females. A meta-analysis of five North American populations by Vick (2005), however, found no evidence of a significant decline in dimorphism during the agricultural transition. The idea that sexual dimorphism arises as a result of selection acting differentially on sex-specific ecological strategies among contemporary humans is also raised by several other authors (Brace & Ryan, 1980; Wolfe & Patrick Gray, 1982; Shine, 1991; Holden & Mace, 1999; but see Harvey & Bennett, 1985). Thus, changes in human body size and stature over evolutionary time, both in absolute terms, and relatively between the sexes, may reflect changes in ecological characteristics, including the increasing interplay of biological and cultural influences, and not only aspects of sexual selection.

With this caveat in place, we can consider the evidence for sexual selection as the cause of sexual dimorphism. One of the earliest studies applying sexual selection theory to human populations is by Alexander *et al.* (1979). After establishing that sexual size dimorphism (SSD) was positively correlated to the degree of polygyny in pinnipeds, ungulates, and primates, the authors presented evidence to suggest that, among humans, populations with polygynous mating systems displayed higher levels of SSD than those with monogamous systems (but only where such monogamy was not socially imposed). These results are, however, widely disputed. Gray & Wolfe (1980), in particular, highlighted severe methodological problems with the Alexander *et al.* (1979) study (including issues relating to data selection, marriage system attribution, and height measurements), and their reanalysis suggested that polygyny was not related to SSD, but that dietary factors exerted a strong influence on sex differences in stature across populations. Nutritional deficiencies have a stronger effect on male than on female growth, which could account for the observation that well-nourished populations are more dimorphic than malnourished ones (Hiernaux, 1968; Brauer, 1982; Hamilton, 1982; but see Guegan, Teriokhin & Thomas, 2000; Gustafsson *et al.*, 2007). Gaulin & Boster (1985), in turn, were also skeptical of earlier research on SSD, arguing that polygyny was not related to cultural differences in dimorphism, and that cross-cultural variation was minimal when excluding populations for which estimates of heights are based on very few individuals. Ironically, these same authors went on themselves to study variation in SSD in relation to social stratification and polygyny (Gaulin & Boster, 1992), which more or less replicated the findings of Alexander *et al.* (1979).

Most early work on SSD is, however, severely hampered by a lack of phylogenetic methods (which, of course, were not readily available at the time). At least two studies have demonstrated that both sex differences in size and the explanatory factors used (e.g. mating system, division of labour, subsistence strategy) show a strong phylogenetic signal, which must therefore be taken into account in statistical analyses (Holden & Mace, 1999; Gustafsson & Lindenfors, 2004). Holden & Mace (1999) present just this kind of phylogenetically controlled analysis (also controlling for geographical proximity), finding that sexual dimorphism was associated only with the contribution of women to food production, and not with subsistence strategy nor marriage system (although they do note that, because polygyny is a highly 'clustered' trait, its effects are not easily detected, which means an influence of polygyny cannot be ruled out). When the sexual division of labour is less distinct, and women contribute more to the production of food, sexual dimorphism tends to be lower. Holden & Mace (1999) argue that, in populations in which women perform a large proportion of subsistence labour, parents may bias their investment towards daughters, and hence lower dimorphism in stature will be expected. A more recent hypothesis concerns the selection pressure of maternal death in childbirth: in high-fertility countries with high maternal mortality, selection should strongly favour increased female height (as height is positively associated with ease of the delivery of a child) and hence reduce SSD (Guegan *et al.*, 2000).

Some authors have also studied whether Rench's rule applies to humans. This rule states that, in taxa where males are larger than females, there is also a positive correlation

between body size and SSD (Fairbairn, 1997). This is generally well established in animals, although exceptions exist (Abouheif & Fairbairn, 1997). Thus, one would expect that, in tall human populations, dimorphism in stature between men and women should also be greater compared to 'shorter' populations. This prediction does not, however, seem to be met in humans (Gustafsson & Lindenfors, 2004, 2009; Gustafsson *et al.*, 2007).

In contrast to the studies described above, Rogers & Mukherjee (1992) have argued that we should not, in fact, expect sexual dimorphism to represent an adaptation to the environment because the response to selection is likely to be very slow due to the genetic correlation in stature between the sexes (i.e. the same genes for height operate in both sexes). It is doubtful whether there has been sufficient time, and if cultural practices have been sufficiently stable, for sexual dimorphism to be attuned to specific population conditions. As should be apparent from these conflicting theoretical perspectives, empirical findings, and methodological criticisms, support for any of the hypotheses explaining sexual dimorphism is not particularly strong, and all require further testing. The causes of (human) size dimorphism are clearly complex and our understanding of these at present is poor, but it seems likely that several mechanisms operate both within and between populations (Plavcan, 2011, 2012).

IV. GENETIC INFLUENCES ON HEIGHT

Although environmental variation is an important factor influencing adult height, such influences are considered insufficient to account fully for observed population differences. Some African populations are considerably taller than others, for example, despite experiencing poorer nutrition and elevated levels of pathogen exposure (Deaton, 2007), suggesting that such differences may have a genetic basis. To date, very few studies have addressed this issue. Notable exceptions are studies investigating the difference in height observed between the Baka pygmies of Cameroon and taller neighbouring non-Pygmy populations (Becker *et al.*, 2011; Jarvis *et al.*, 2012). Both of these studies showed that Pygmy individuals who were genetically more similar to non-Pygmy individuals (i.e. higher levels of genetic admixture) were taller. Most recently, Perry *et al.* (2014) have shown that the pygmy phenotype likely arose several times independently due to positive natural selection for short stature. Additional evidence for genetic factors underlying population differences in height come from a Korean population (Cho *et al.*, 2009). Several loci previously identified in European populations were also detected in this sample. Most pertinently, one of the genes with the largest effects on stature observed in Europeans (HMGA2) was also associated with height in the Korean population, although its effect was much smaller and the frequency of the 'height-raising' allele in the population was lower. These findings suggest that genetic differences may, in part, explain why Koreans are, on average, shorter than Europeans (Cho *et al.*, 2009).

Although studies investigating genetic differences in height across populations are still rare, the genetics of height itself is, of course, a very well-studied phenomenon. Human height is, perhaps, the classic example of a quantitative trait under genetic influence; indeed, the study of stature was essential to the emergence of fundamental concepts in genetics, with Francis Galton first demonstrating the statistical relationship between the heights of parents and their children (Galton, 1886), and establishing its high heritability. These studies have stood the test of time with 'twin studies' consistently demonstrating that the heritability of height is around 0.80 (Silventoinen *et al.*, 2003*b*; Perola *et al.*, 2007; see also Visscher *et al.*, 2006, 2007, which used the genetic similarity between non-twin siblings to arrive at a similar value). The heritability estimate for height therefore seems to be exceptionally reliable: it has been observed repeatedly across different populations and using different methods, although its high value is restricted to more affluent populations. Heritabilities in developing countries are slightly lower with, for instance, a value of 0.62 in a Nigerian sample (Luke *et al.*, 2001), 0.65 in a Chinese sample (Li *et al.*, 2004), and 0.74 in a Jamaican sample (Luke *et al.*, 2001). Similarly, with an increased standard of living, heritability also increases over time within a population (Silventoinen, Kaprio & Lahelma, 2000). A potential explanation for these findings is that differences in factors like food abundance exert a greater influence on differences in adult height than an individual's genetic make-up in poor environments. By contrast, in richer environments, differences in food abundance are less severe and, as a consequence, their effects on growth will be diminished compared to genetic effects (Charmantier & Garant, 2005).

The high heritability of height does not, however, mean that environmental influence cannot exert large effects, but merely that, in populations where environmental conditions are very similar for most individuals, most phenotypic variation will be due to genetic differences. Nonetheless, variation in environmental factors may affect height substantially, milk consumption being a very good example of an environmental influence that exerts large effects on height in environmentally homogenous populations (de Beer, 2012).

The high heritability of stature should also not be taken to imply that height is a result of a few genes that exert large effects. As early as 1918, Sir Ronald Fisher proposed that a 'polygenic' model could explain variation in height, with many genes, each with a small effect, influencing stature (Fisher, 1918). Since the completion of the Human Genome Project in 2003, scientists have been able to identify the genetic variants associated with certain traits and diseases by comparing individual genomes. To date, human genome-wide association studies (GWAS) have identified 180 loci, which account for about 10–20% of the observed variation in height (Allen *et al.*, 2010), and this same genetic information can be accurately used to identify those of tall stature within a population (Liu *et al.*, 2013). Moreover, using sophisticated statistical modelling, Yang *et al.* (2010) found that common genetic markers could explain fully 45% of the variance in height when all genetic variants were

considered simultaneously. Height is thus a truly polygenic trait, with almost 1% of all human genes contributing to height variation in some way (Allen *et al.*, 2010).

One of the great advantages of modern genetic techniques is that they offer insight into the functional significance of the genes that regulate height. The height-associated loci identified by GWAS have been shown to cluster together in biologically relevant pathways, including those related to bone formation, chronodocyte proliferation and differentiation (the cells that produce and maintain cartilage), and the growth hormone signalling pathway (Lettre, 2011). In addition, 21 loci have been found to be situated near a skeletal/growth gene known to be associated with pathological growth syndromes when mutated, with 13 of these located within the gene itself (Allen *et al.*, 2010; Lettre, 2011).

There has also been interest in identifying whether any adult-height-associated loci are implicated in the growth process, especially as twin studies have suggested a strong genetic component to growth variation (Silventoinen *et al.*, 2003*b*, 2008). The few studies conducted to date suggest that most adult-height-linked loci are not, however, associated with children's height, which may reflect the highly heterogenous growth patterns seen among children. Instead, it has been suggested that height velocity (i.e. growth rates) may represent a more useful phenotypic trait for studies of this nature (Lettre, 2011). Again, data testing these ideas are very limited at present, and so far have not revealed any genetic associations that reached genome-wide significance (Lettre, 2011). Sovio *et al.* (2009) have shown, though, that loci associated with adult height are at least nominally associated with height velocity during infancy and puberty. In addition, Widén *et al.* (2010) found an association between variants near the LIN28B gene (a gene known to control developmental progression in *Caenorhabditis elegans*: Moss, Lee & Ambros, 1997) and growth increases between the age of 14 years and adulthood. This same locus has also been associated with adult height (Allen *et al.*, 2010) and age at menarche (Elks *et al.*, 2010), suggesting it has an influence on pubertal timing. This makes sense given the idea that skeletal maturity, and in the case of females, pelvic geometry (Ellison, 1990), are hypothesized to be key to future successful reproduction. Experiments on mice have shown that those with a Lin28a transgene show increased body size and delayed puberty (Zhu *et al.*, 2010), which supports the pubertal timing interpretation.

Understanding more about genetic influences on height is not only beginning to reveal how and where genes exert their effects on the growth process, but also helps to reinforce the fact (perhaps ironically) that the secular trend in height must primarily reflect environmental influences, and is unlikely to be the consequence of significant genetic change; height would not be expected to increase so rapidly over such a short period if this were the case (Deaton, 2007; McEvoy & Visscher, 2009). To be more precise, if the Dutch increase in height of 20 cm in the last 150 years were solely genetic, this would require that, in every generation, fully 30% of the shortest individuals in the population must fail to reproduce at all (assuming a generation time of 25 years and a heritability of 0.8); a selection pressure high enough to be considered implausible (Kingsolver *et al.*, 2001). It is worth noting again, however, that while genes are unlikely to have a major role in the secular trend in height seen around the world, their effects cannot and should not be ruled out entirely. For example, height-increasing alleles are found to occur at higher frequencies in northern than in southern Europeans (Turchin *et al.*, 2012), and this difference is suggested to be a consequence of selection on gene variants associated with height, rather than with genetic drift (i.e. changes in gene frequencies that occur by chance). Thus, adult height differences across populations of European descent are not driven solely by environmental differences, but apparently retain a signature of past differential selection.

V. LIFE HISTORY AND HEIGHT

Having now surveyed patterns of height variation and their links to environmental and genetic factors, it is apparent that variation in height is systematically related to variation in environmental conditions, and can largely be attributed to developmental plasticity in growth velocity during childhood, although there is also some evidence for genetic differences between populations. There is also good anthropometric evidence for a link between skeletal maturity and sexual maturity, with recent genetic evidence in line with such a link. The energy devoted to growth in childhood and the timing of the switch from growth to reproduction thus combine to determine adult height in a population. This raises the further question of how and why resources are allocated to growth during development, and the scope and limits of developmental plasticity in relation to height. These are issues that can be most productively explored from a life-historical perspective.

(1) Principles of life-history evolution

Body size at maturity, and the process of growth toward this size, is an evolutionary dilemma for all species. At its the heart lies a classic trade-off between growth, maintenance and reproduction (Stearns, 1992), which in turn reflects a basic 'principle of allocation': energy and resources can only be invested once, such that investment in one direction, e.g. towards maintenance, diverts investment away from the other two components (Stearns, 2000). The costs of maintenance (e.g. the metabolic costs of being alive, the materials and energy needed for tissue maintenance and repair, healing from injury and general wear and tear, and defence against disease) can be subtracted from energy intake to determine the net resources available for growth and subsequent reproduction once growth is completed, with the latter known as 'productivity' (Charnov, 1991, 1993; Stearns, 1992). It should be obvious that higher productivity means greater energy available for growth and/or reproduction. Life-history theory therefore predicts that, all else being equal, greater capital investment in body size, i.e. growing larger and maturing

later, will increase adult reproductive capacity; the same may be true for individuals of increased height.

Allocation of resources, however, also depends on mortality rates, as these dictate the extent to which the greater reproductive capacity acquired during the pre-reproductive growth phase can be translated into offspring. High levels of adult mortality (especially from extrinsic causes, e.g. violence or disease) should lead animals to forego extra growth and mature earlier so that reproduction can occur at an earlier age. Mortality during the juvenile phase is also expected to exert a strong influence on life-history trajectories, as this determines the likelihood that an individual will actually survive to reach reproductive age at all; again, higher rates of extrinsic mortality acting on juveniles should favour earlier sexual maturity. The incorporation of mortality rates into life-history theory thus builds in the opportunity costs of a delay in reproduction, and is thus crucial for setting the balance between growth, reproduction and hence adult body size across species (Harvey & Clutton-Brock, 1985; Harvey & Zammuto, 1985; Promislow & Harvey, 1990; Harvey & Purvis, 1999).

(2) Life-history trade-offs and norms of reaction

Similar trade-offs between growth and reproduction may also occur within species, including humans, in response to environmental variability and mortality risk, generating plasticity in growth trajectories, body size and the onset of maturity. Systematic variation at this scale may therefore reflect the operation of evolved reaction norms for optimal growth (i.e. the set of phenotypes that result from a single genotype across different environments: Roff, 1992; Stearns, 1992; Walker *et al.*, 2006). For example, in a detailed study of small-scale societies, Walker *et al.* (2006) found that poor environmental conditions generally lead to a trade-off between maintenance and growth (favouring energy invested in maintenance), resulting in small adult stature, slow growth rates, and later ages of menarche compared to higher quality habitats (although when juvenile mortality risks are high, faster growth rates are favoured: see Section V.5). This may represent an adaptive response because it reduces bodily maintenance costs, lowering both intrinsic mortality risk and increasing the relative allocation of resources available for reproduction (Arendt, 1997; Ellison, 2001; Worthman, 2003; Walker & Hamilton, 2008). Poor growth conditions can thus serve both as a constraint on growth and as a valid indicator of the kind of future nutritional regimes an individual is likely to face, ensuring that individuals attain the body size best suited to a nutritionally poor environment. Slow growth spreads nutritional demands over a longer period of development, and short adult stature lowers the cost of maintenance across the lifespan, both of which can represent an adaptive trade-off between growth and maintenance (Walker *et al.*, 2006) as we discuss further below (Section V.5). That is, phenotypic variation across environments may represent the expression of an evolved reaction norm, which ensures an appropriate trade-off between life-history components.

(3) Height and sexual maturity in contemporary industrial populations

Among industrialized nations, females are not only absolutely taller but they also display earlier menarche compared to small-scale societies (e.g. Cole, 2000) (although it should be noted that, under good conditions, women in small-scale societies can also reach menarche early and at a larger body size compared to those in poor conditions, that is, similar patterns can be seen, even if absolute values differ). A good-quality environment helps to reduce bodily maintenance costs (e.g. because fewer immune responses need to be mounted), leading to increased growth rates and hence earlier reproductive function. These changes in height and age at sexual maturity can be seen as the consequence of sustained good nutrition and low environmental stress, as discussed above in relation to the secular trend in height (Section II.2*b*); hence, the secular trend can be viewed as the manifestation of an evolved reaction norm that regulates life-history transitions in ways appropriate to the environment. A recent meta-analysis by Mcintyre & Kacerosky (2011), however, demonstrates that the relationship between height and age at menarche may have reversed in industrialized nations: taller heights are now associated with later maturity (although even 'late' menarche still occurs much earlier than in small-scale societies, at approximately 14 years compared to around 17–18 years) (see also Cole, 2000).

Mcintyre & Kacerosky (2011) suggest that this shift in life-history strategy from small-scale to industrial societies fits with Day & Rowe's (2002) developmental overhead threshold model. This model is based on the assumption that individuals cannot make the transition from the growth phase to the reproductive phase until they have reached a threshold body size (which they term an 'overhead threshold', because a certain fixed proportion of body tissue is required to support reproductive functioning). As we have seen, human females are required to reach a certain level of skeletal maturity before becoming fertile in order to ensure a pelvis of sufficient width to permit birth (Ellison, 1990). One can therefore interpret this early investment in skeletal growth as a form of overhead threshold. Day & Rowe (2002) show that, in poor conditions, growth prior to reaching the threshold is prolonged (because growth rates are slow) and individuals switch to the reproductive phase as soon as possible after the threshold is reached, because they cannot afford to delay reproduction any longer. By contrast, under very good conditions, the time needed to reach the body size threshold is shorter (due to faster growth rates), and individuals continue to grow for longer after reaching the threshold in order to increase later fecundity. Mcintyre & Kacerosky (2011) therefore suggest that this kind of effect gives rise to a negative relationship between age at maturity and size under poor conditions (as seen in small-scale human societies and early industrial society), but a positive relationship under good conditions (as seen in contemporary industrial societies).

Mcintyre & Kacerosky (2011) caution that their interpretation is tentative, and note that the positive relationship seen

across industrialized nations could be a statistical artifact, reflecting changes in both the sources and degree of trait variation. They suggest, for example, that under poor conditions, environmentally caused variation in growth rates will largely determine age at puberty (faster growth leading to earlier maturation) but that, as conditions improve, environmentally induced variation in growth rates and age at sexual maturity will decline, and genetic differences between individuals will become more prominent. As Mcintyre & Kacerosky (2011) note (echoing Bogin, Silva & Rios, 2007), we still know surprisingly little about population-level patterns and individual-level biological processes in relation to age, growth rates and size at maturity, all of which makes testing adaptive *versus* non-adaptive hypotheses an ongoing challenge.

(4) Variation in growth: adaptive, constrained or pathological?

Indeed, the question of what counts as the optimal growth pattern, and whether to characterize the effects of poor nutrition on height as an 'adaptive' norm of reaction or as 'maladaptive' is an ongoing source of debate (see e.g. Bogin *et al.*, 2007; Ellison & Jasienska, 2007; Kuzawa, 2007). Whereas 'adaptive' in this particular context is taken to mean the phenotypically plastic responses that produce the optimal environment-specific body size, 'maladaptive' responses can be divided into those that reflect 'constraints' (defined as physiology directed at the same goal as the unconstrained condition but unable to achieve it fully) and 'pathology' (defined as physiology that has been thrown off its normal optimal trajectory and arrives at maladaptive outcomes) (Ellison & Jasienska, 2007). This usage deviates from the definitions more commonly used in evolutionary biology, which are themselves often disputed (see e.g. Fox & Westneat, 2010). Instead, it is linked more strongly to evolutionary theorizing regarding 'predictive adaptive responses' and developmental programming (Gluckman, Hanson & Spencer, 2005; Langley-Evans, 2006). Broadly speaking, these theories rest on the idea that there is a mismatch between the conditions experienced by the fetus and those experienced during the post-natal period. This in turn is suggested to reflect a broader mismatch between our evolved propensities and the environmental conditions of the modern world, particularly those of industrialized nations.

The costs experienced in later adult life, in terms of extremely poor health outcomes, under conditions when deprivation during infancy or childhood is severe, are regarded as clear indicators of pathology in just this sense (e.g. Barker *et al.*, 1989; Barker & Clark, 1997). Some of these costs seem to be related to 'catch-up' growth (the increase in growth velocity needed to put individuals back on the optimal trajectory for their environment once deprivation ends, e.g. Metcalfe & Monaghan, 2001; Monaghan, 2008), which suggests there are trade-offs between excessive growth in early life and later health outcomes (something which may explain why secular trends continue over several generations, rather showing a single rapid increase in a single generation once good growth conditions are achieved: Cole, 2000). Even when deprivation is less severe, constraints on achieving optimal growth can produce negative downstream effects (e.g. being stunted is unlikely to lead to optimal health in later life: Bogin *et al.*, 2007), such that the line between these two 'maladaptive' responses is often unclear, and indeed may not be especially useful when considering human growth patterns from a purely evolutionary life-historical perspective. This is because adaptations do not need to exert wholly positive effects (and indeed, rarely do so) and we should expect to see costs and benefits traded off against each other. Sickle cell anaemia, as is well known, is a pathological consequence of an adaptation that prevents malarial infection, so the mere existence of costs (even when severe) does not necessarily rule out an adaptive response.

Drawing a contrast between adaptive *versus* constrained/pathological outcomes is also complicated by the fact that cross-population comparisons of growth trajectories and adult height are carried out by calibrating against a reference group. If different populations face different life-history trade-offs, and their heights reflect the operation of an evolved reaction norm, then such comparisons will never be straightforward, given that these depend on the reference group used to define the optimal (average, or 'normal') growth trajectory. Stunting, for example, is identified by comparing measurements of children's heights to the USA National Center for Health Statistics growth reference population, but environmental conditions in, say, rural Venezuela may actively favour shorter adult stature with respect to life-history trade-offs, and hence result in a locally attuned growth trajectory that is neither 'constrained' nor 'pathological' when viewed in an evolutionary perspective. A more productive approach perhaps would be to construct and compare individual growth trajectories under a wide range of environmental conditions, rather than assume that there is an optimum growth trajectory that applies across all environments. In other words, 'adaptive' can be considered as an entirely relative term that compares the success of individuals facing similar conditions: extremely poor environmental conditions will obviously constrain growth and reproductive patterns in some way but, given those conditions, the patterns on display may represent a 'best of a bad job' strategy and cannot simply be dismissed as 'maladaptive'. This bears resemblance to Scrimshaw & Young's (1989) notion of 'accommodation', to describe those responses that favour survival while simultaneously resulting in losses to other important functions, and so impair optimal adjustment to the environment.

A more radical suggestion is that we abandon the idea that there is some 'target' height from which deviation is possible (e.g. Tanner, 1963), thus leading to 'constrained' or 'pathological' outcomes. In this respect, approaching the issue from a 'developmental systems theory' (DST) perspective may prove valuable (Griffiths & Gray, 1994, 2001). As Oyama, Griffiths & Gray (2001) argue, the standard interactionist view, which sees traits as a product of genes plus environmental influences, naturally leads us to think of context-sensitivity and contingency as factors that

lead the genetically derived developmental 'program' to deviate from an evolved 'norm' in some or other way. It also points to the possibility of neatly disentangling genetic from environmental influences. DST instead views development as inherently context-sensitive and contingent, with no absolute priority given to any particular processes, and no conception of a 'target' or 'standard' from which it is possible to deviate. This would therefore mean abandoning aspects of Tanner's (1963) 'target-seeking' model of growth, which suggests that 'children, no less than rockets, have their trajectories, governed by the control systems of their genetic constitution and powered by energy absorbed from the natural environment' (p. 818). Instead, individuals would be viewed as turning out more or less the same because they are constructed using the same suite of developmental resources, with no underlying genetic program governing this process nor defining what the organism is 'meant to be'.

These suggestions for reconfiguring how we view adaptation *versus* constraint/pathology are rendered problematic, however, by the fact that, in anthropology, the term 'adaptation' is used, not only in an evolutionary sense, but also to refer to immediate adjustment to the current environment (i.e. a person's ability to function unimpaired in everyday life), which brings in aspects of well-being and life satisfaction. That is, when we demonstrate that short stature under poor conditions enhances fitness and is therefore evolutionarily adaptive, nothing inherently good or bad is implied by the term, even if this comes with certain costs. We may very well consider such costs unacceptable from a humanitarian perspective, however, because they indicate a low level of adjustment to current conditions, and hence a reduced quality of life (see e.g. Pelto & Pelto, 1989). 'Adaptation' in this sense is thus a much more loaded concept, and its usage has clear political implications. For example, Seckler's (1982) controversial 'small but healthy' hypothesis suggested that the short stature resulting from mild to moderate malnutrition was adaptive, because children who faced such deprivation showed no impairment as adults other than slowed growth and shorter adult height. In life-history terms, we can see this argument as rather straightforward: being small under conditions of marginal food availability is beneficial because small bodies are easier to maintain (even if this entails costs in other domains).

This hypothesis has come in for major criticism, however, not only because the idea of a 'no-cost' adaptation is disputed, and indeed there is considerable evidence against it (e.g. Pelto & Pelto, 1989; Henneberg, Harrison & Brush, 1998; Bogin *et al.*, 2007), but also because it holds implications for the provision of food aid and other humanitarian efforts (i.e. if people are adapted to low food intakes, it implies there is no need to supplement their diet, even if they fall short of Western standards of nutrition). In other words, deciding on what counts as 'adaptive' or 'impaired' (pathological) will never be a simple matter because these terms can never be divorced from their cultural and political context. This issue obviously requires a more thorough and careful discussion than we can provide here, but we feel it is important to mention this, not least because it highlights that height cannot be viewed simply as a straightforward 'biological' trait (see also Butt, 1999; Spencer & Logan, 2002).

(5) Mortality, growth and height

In addition to nutritional influences on development and life-history processes, any factors that acutely increase the risk of mortality for particular age-classes can also have an effect on growth rates. As juvenile mortality increases, and the chance of reaching adulthood decreases, postponement in the onset of reproduction becomes more costly and growth rates should thus increase, giving rise to an earlier onset of sexual maturity (see e.g. Moorad & Promislow, 2010; Walker *et al.*, 2006). In their detailed study of small-scale subsistence societies, Walker *et al.* (2006) report evidence for just such a trade-off among three pygmy populations, the central African Baka, the Hiwi of Venezuela and the Negritos of the Phillipines, who all show an accelerated developmental pattern. These groups show both faster and more linear growth across development than expected for their body size, and do so despite poor environmental conditions. In addition, the adolescent growth spurt is either diminished or absent (although as only cross-sectional data were used to determine these effects, this latter point should be viewed with some caution). Walker *et al.* (2006) also show that, for the Hiwi, Negritos and Baka, juvenile mortality is positively associated with accelerated growth rates relative to age.

Baka pygmy girls show a childhood growth rate of 7.1 cm/year, while Hiwi girls grow at an almost identical rate of 7 cm/year (Walker *et al.*, 2006). These values approach or exceed those of USA children (average childhood growth rates of 6.5 cm/year). As a consequence, girls in these groups display much higher levels of skeletal maturity at much younger ages relative to other groups. While a Baka or Hiwi girl has reached about 70% of her adult size by age 10, in other groups, such as the Maya, girls have reached only 40–45% of their adult size by this age (Walker *et al.*, 2006). As we have seen, menarche is cued to skeletal maturity, and occurs only after girls have completed ~95% of their growth (Ellison, 1990; Kramer & Greaves, 2010). This suggests that fast growth is geared toward permitting earlier reproduction and occurs at the expense of larger body size (because growth ceases at a younger age, and accelerated growth rates cannot fully compensate for this reduction in the pre-pubertal growth period).

Migliano, Vinicius & Lahr (2007) also found support for this idea in their detailed analysis of pygmy *versus* non-pygmy populations [but see Becker *et al.* (2010) for criticism of this study, and Migliano, Vinicius & Lahr (2010) for a response]. Assuming that life-history trade-offs do, in fact, explain short stature and accelerated growth among these pygmy populations, it raises the question of why other groups that experience high juvenile mortality do not exhibit this same pattern. Walker *et al.* (2006) suggest this may reflect habitat differences: in the humid forest habitats occupied by pygmies, mortality risks from disease are particularly acute, and larger juvenile body size may confer a direct benefit

via a more robust immune system, as well as allowing for earlier onset of reproduction. In the drier, higher elevation habitats occupied by many of the other groups in their sample, disease risk lessens and malnutrition and starvation become more important sources of mortality, which favours slower childhood growth.

The developmental patterns shown by pygmy populations are underpinned by distinctive hormonal profiles that have a genetic basis (Dávila *et al.,* 2002), and stature in these populations accordingly shows relatively low levels of plasticity compared to other groups (Walker *et al.,* 2006; Becker *et al.,* 2011). The Baka, Hiwi and Negritos growth response may therefore reflect selection for a specific life-history pattern, with growth rates more strongly fixed by natural selection, rather than reflecting the expression of a flexible norm of reaction. In this respect, data from Pumé girls and women in Venezuela are especially valuable: early reproduction is common within this high-mortality population, with a mean age at first birth of 15.5 years. This does not occur in the context of either very short stature or distinctive hormonal profiles compared to other northern South American indigenous groups, however, suggesting that this specific growth pattern has not come under selection (Kramer & Greaves, 2010). Thus, any increase in growth rates seen among these girls can more confidently be attributed to the expression of a reaction norm.

Similar to the Baka and Hiwi, Pumé girls have reached 87% of their adult height by age 10. As might be expected, the timing of their peak growth velocity occurs considerably earlier compared to other groups, and persists for longer, and hence they do not show a distinct adolescent growth spurt (Kramer & Greaves, 2010) (this, incidentally, suggests that, contrary to current consensus, an adolescent growth spurt, while unique to humans, is not a unique feature of all human groups: Walker *et al.,* 2006). Pumé girls illustrate clearly that faster growth rates can occur as a plastic developmental response under poor environmental conditions and high mortality risk, giving rise to early and advantageous reproduction, and can be achieved in the absence of stunting.

It is clear that life-history trade-offs account for many of the differences in height seen within and between populations, and that the timing of menarche in contemporary societies is constrained by the rates at which women, in particular, achieve the level of skeletal maturity needed to support reproduction. From an evolutionary perspective, this suggests that height itself is less important as a trait than the underlying components of growth rates and the timing of reproductive maturity that give rise to it. This raises the question of whether height does, in fact, carry any selective advantage independent of its links to life history. Aspects of the obstetric dilemma suggest this might be the case, at least for women, and stature has also been linked to various ecological selection pressures, suggesting this is a question worth pursuing. In Section VI, then, we assess the evidence that natural selection acts on height in contemporary human populations.

VI. NATURAL SELECTION ON HEIGHT

(1) Mortality risks and height

(a) *Height, health and mortality in adults*

It seems reasonable to suppose that natural selection will act on height to the extent that this trait is related to health and survival (Batty *et al.,* 2009). Generally, height is positively related to measures of health (Silventoinen, Lahelma & Rahkonen, 1999; Stulp *et al.,* 2014), although curvilinear effects for women have also been observed (Silventoinen *et al.,* 1999). Moreover, the relationship between height and health is much stronger (and positive) when assessing short to average height men than when comparing average height to tall men (Stulp *et al.,* 2014). It seems likely that there are diminishing returns to increasing height, and very tall men may have much poorer health outcomes.

As one might expect given these patterns in relation to health, there is also a relationship between height and all-cause mortality. A review by Sear (2010) indicated that, in Western populations, taller men have lower mortality than shorter men. Taller women also experienced lower mortality than shorter women in most studies, although some reported a curvilinear effect, with average-height women experiencing the highest survival (Sear, 2010; but see Samaras, 2009). A recent meta-analysis, with data on over 1 million individuals generally supports Sear's (2010) analysis: taller men and women had a lower risk for all-cause mortality, although there were opposing effects of height on mortality for specific causes of deaths. In general, shorter people were more at risk of dying from cardiovascular and non-cancerous disease, whereas taller people were more at risk of death from cancer (Wormser *et al.,* 2012). The overall increased risk of mortality for shorter people may be due partly to associated differences in socioeconomic background. Although some indicators of economic background were controlled for in this meta-analysis, it is also the case that, as we have seen, poor nutrition during childhood may lead to both reduced height and poorer health as adults; controlling for current measures of economic status does not necessarily eliminate or control for these childhood effects. With respect to increased cancer risk of taller individuals, one suggestion is that they simply have a greater number of cells in their body, and hence an increased risk of defects in cellular repair mechanisms. This fits with the idea that larger bodies may be more costly to maintain and repair over time. The weight of evidence thus suggests that short men and women are at a general disadvantage with respect to overall health and survival in Western populations, while taller people may experience health risks of a more specific nature. Too few studies have addressed these same issues in non-Western populations to draw any meaningful conclusions on this front (Sear, 2010).

(b) *Height and infant mortality*

In addition to their greater mortality risks, shorter women are also at a disadvantage with respect to successfully raising

children to adulthood: both child morbidity and mortality are negatively related to maternal height (Subramanian & Ackerson, 2009). Shorter women have been shown to be at a higher risk for complications during pregnancy and delivery, such as stillbirth (Bresler, 1962), failure to progress in labour (Sheiner *et al.*, 2005), and the need for Caesarean sections (Kirchengast & Hartmann, 2007). Stulp *et al.* (2011) found that shorter British women (i.e. 1 S.D. below mean height) were one and a half times more likely to need an emergency Caesarean section than taller women (1 S.D. above mean height) when giving birth to their first child. Furthermore, over half of British women under 150 cm needed a Caesarean section. In addition to maternal height, the difference in height between parents was also linked to a greater risk of birth complications in this study: the likelihood of an emergency Caesarean section was significantly higher in cases where the father was very much taller than the mother (particularly when the baby is heavy) (Stulp *et al.*, 2011). Thus, one could argue that the birth complications suffered by shorter women, often due to obstructions of the birth canal, represent a significant selection pressure on height, given that pelvic dimensions increase with stature. A recent study by Kurki (2007), however, argues that, in short-statured populations, pelvic geometry (size and shape) has shifted to protect the obstetric canal; that is, selection has acted on pelvis shape to reduce the risk of birth complications, rather than selecting against short stature. It is also the case that, when birth complications do arise, this is often due to deformation of the pelvis due to malnutrition, rather than because of short stature *per se* (Wells *et al.*, 2012).

The adverse effects of short stature during pregnancy and parturition also appear to carry through to newborn health. Shorter women are more likely to give birth to infants with relatively low birth weights (Camilleri, 1981) and relatively low Apgar scores (a health assessment administered directly after delivery: Camilleri, 1981; Casey, McIntire & Leveno, 2001), both of which are predictors of child morbidity and mortality (McIntire *et al.*, 1999; Casey *et al.*, 2001). Maternal height is also negatively related to child mortality both in developing (Monden & Smits, 2009) and low-to middle-income countries (Özaltin, Hill & Subramanian, 2010); indeed, the literature shows very clearly that taller women have increased child survivorship in non-Western populations. Even in a contemporary, industrialized population with very low levels of child mortality, shorter mothers had higher child mortality than taller mothers (Stulp *et al.*, 2012*d*). Although these patterns between maternal height and child morbidity and mortality appear to be quite general, it is unclear whether they hold for all populations.

Overall, then, the evidence on mortality risks suggests that it is possible for natural selection to be acting currently on human height, and that it may do so differentially between the sexes. Effect sizes in the available studies are generally small, however, and it seems unlikely that selection could exert any influence without also influencing differential fertility in additional ways. One obvious candidate is *via* mate choice and sexual selection. Indeed, many of the health effects observed may influence mate choice processes, and it seems likely that these might exert a greater influence on fitness differentials in contemporary low-mortality populations.

(2) Sexual selection and height

Sexual selection could operate on height either by increasing competitiveness within the sexes or by increasing the probability of being selected as a mate by the opposite sex. In considering the empirical evidence for sexual selection on height in humans, some caution is warranted: most studies do not present direct evidence that greater competitiveness and/or attractiveness translates into higher reproductive success among contemporary populations. Rather, they deal only with preferences for particular traits, and place the emphasis on this as evidence of past selection, rather than attempting an assessment of the opportunity for selection in the present day.

(*a*) *Intrasexual competition*

Height has been associated with physical dominance in human males, mirroring findings from other mammals (Ellis, 1994): taller men are physically stronger (Sell *et al.*, 2009; Puts, 2010), more aggressive (Archer & Thanzami, 2009), and are argued to have better fighting ability (von Rueden, Gurven & Kaplan, 2008; Archer & Thanzami, 2009; Sell *et al.*, 2009). The physical superiority of taller men is thought to contribute significantly to their achieving greater access to resources and status within social groups. In modern Western societies, it seems unlikely that physical strength and fighting ability have any direct influence on social status and resource acquisition in terms of male–male physical combat. There are, however, clear social gradients in height that reflect differential access to resources: in a study examining 10 different European countries, Cavelaars *et al.* (2000) found that, without exception, more highly educated individuals were taller than those who were less educated, with height differences ranging from 1.2 to 3 cm. Similarly, a meta-analysis by Judge & Cable (2004) found that height was consistently positively related to leadership, income and professional achievement, all of which are proxies for access to resources. The positive relationship between height and several measures of social status has been well established (e.g. Case & Paxson, 2006; Magnusson, Rasmussen & Gyllensten, 2006; Batty *et al.*, 2009; Deaton & Arora, 2009), although relationships need not always be linear, and, as is the case for health, diminishing returns on income with increasing height have also been observed (e.g. Stulp *et al.*, 2014).

Several hypotheses have been put forward to explain this relationship between stature and status including the correlated influence of parental resources on height and social success (Persico, Postlewaite & Silverman, 2004), the decreased health of shorter men and women (Silventoinen *et al.*, 1999) and the increased cognitive ability associated with height (explained by factors such as genes and/or nutrition (Case & Paxson, 2006; Silventoinen *et al.*, 2006). Intriguingly, Persico *et al.* (2004) observed that, even after controlling for all the above factors, taller individuals retained their social

advantage, raising the possibility that height has a direct influence on the ability to achieve high social status in modern Western society (see also Cinnirella, Piopiunik & Winter, 2011). Indeed, the upward social mobility of taller compared to shorter individuals (Peck, 1992; Cernerud, 1995; Krzyżanowska & Mascie-Taylor, 2011), even when familial circumstances are controlled (e.g. by examining sibling pairs: Bielicki & Charzewski, 1983; Magnusson *et al.*, 2006) suggests there is more to height than simply parental influences.

One way in which taller individuals may achieve higher social status is by increasing the probability of winning non-physical confrontations. There is currently only circumstantial evidence for this idea, including the higher proportion of popular votes that taller USA presidential candidates receive (Stulp *et al.*, 2013*d*), the increased number of unfair offers made by taller individuals in economic games within a virtual reality setting (Yee & Bailenson, 2007), the increased authority of taller football referees on the pitch (Stulp *et al.*, 2012*a*), and the greater influence of perceived taller individuals in a negotiation task (Huang, Olson & Olson, 2002). It seems possible that this occurs because taller men are perceived as more competent and authoritative (Young & French, 1996; Judge & Cable, 2004; Cinnirella & Winter, 2009; Marsh *et al.*, 2009). Whatever the mechanism, there is a clear relationship between height, resource acquisition and social status. This potentially could contribute directly to mating success *via* male competitiveness, although it seems likely to do so only under conditions where female choice is tightly constrained. In most modern settings, male height as an indicator of resource-holding potential is more likely to exert its effects *via* intersexual selection, i.e. as a component of female choice.

(*b*) *Height and intersexual preferences*

One of the central premises of intersexual selection is that traits that signal high reproductive potential should be preferred by the opposite sex (see e.g. Kuijper, Pen & Weissing, 2012). If height acts as an accurate cue to resource-holding potential by men, and if this increases the direct reproductive benefits that accrue to women, then a preference for greater height by women is potentially adaptive. In addition, as taller men are healthier and show lower mortality than shorter men then, to the extent that these traits are heritable, females will gain indirect benefits as well. There may, however, be a limit to women's height preferences, given the finding that women partnered to men much taller than themselves experience an increased risk of birth complications (Stulp *et al.*, 2011). Thus, women potentially face a trade-off between the benefits associated with increased partner height and the cost of increased mortality risk (it should be noted that we are speaking here from a functional evolutionary perspective, and not from a proximate, conscious decision-making perspective, although the latter will factor into this). Moreover, there may be diminishing returns on height, which could mean that men of above-average height do not provide substantively more benefits than average-height men (see e.g. Krams *et al.*, 2014; Stulp *et al.*, 2014).

From the male point of view, we have already noted that short women are at a disadvantage when it comes to health, mortality, obstetric problems, and offspring survival. A preference for average height or taller women could, therefore, be considered adaptive, but again there may be trade-offs involved: there is some evidence to suggest that shorter women are genetically predisposed to display an earlier age at first birth, which potentially compensates for their (children's) higher mortality risks (Stearns *et al.*, 2012; Stulp *et al.*, 2012*d*). Given the complexity of the potential selection pressures operating on humans, and the limits on males as well as females to invest in young, predicting the height preferences of each sex with respect to adaptive outcomes is therefore complex, and not always obvious. In addition, as we have noted, most studies in this area have focused largely on mate preferences, and take no account of the subsequent processes of mate choice and pair formation. As the latter can lead to significant deviations from the stated preference of each sex, the lack of information on these processes is problematic. With these additional caveats in mind, we now review the available evidence relating to intersexual preferences for height.

Height is a partner characteristic known to be valued by both men and women – although women value height more highly (Pierce, 1996) – and preferences for partner height have been well studied (reviewed by Courtiol *et al.*, 2010*b*). In general, average-height women and above-average-height to taller men are considered most attractive by the opposite sex in laboratory-based preference studies. In both men and women, questionnaire data also suggest that, as the height of a given individual increases, so does the preferred height of their partner, indicating preferences for assortative mating (Courtiol *et al.*, 2010*b*; Stulp, Buunk & Pollet, 2013*b*: see also Kurzban & Weeden, 2007; Stulp *et al.*, 2013*c*). Men's preferences in this regard are entirely relative: they prefer their partners to be shorter, but show no preference for shortness *per se* (Courtiol *et al.*, 2010*b*). By contrast, women seem to value male height in a more 'absolute' sense; accordingly, a male-taller preference is more pronounced in women (Stulp *et al.*, 2013*b,c*). Men should not be too tall compared to their female partners, however: excessive height differences are considered unacceptable, and several studies have reported a maximum acceptable height difference of around 25–26 cm (Pawlowski, 2003; Fink *et al.*, 2007; Salska *et al.*, 2008).

A conflict over height preferences between the sexes has also been observed, with women preferring larger partner height differences than men (Courtiol *et al.*, 2010*b*; Stulp *et al.*, 2013*a,b*). This can lead to 'sub-optimal' pair formation, where both sexes are forced to compromise on their preferred height difference (Stulp *et al.*, 2013*a*). Preferred height differences are also dependent on an individual's own height: studies using French, Polish, Dutch and USA samples have shown that both shorter men and taller women prefer smaller partner height differences than do taller men and shorter women (Pawlowski, 2003; Courtiol *et al.*,

2010*b*; Stulp *et al.*, 2013*a*,*b*). It should be noted that while studies on height preferences from industrialized populations are fairly consistent, studies examining non-industrialized populations can be strikingly different (e.g. Sorokowski *et al.*, 2011; Sorokowski & Butovskaya, 2012) Currently, we cannot explain such differences, but they may be contingent on ecological differences between populations, and the 'optimal' height within such populations. Alternatively, methodological differences may be responsible.

The real question here, of course, is whether people's preferences translate into mate selection. There is some suggestion that it does. Male height has been shown to be positively related to the likelihood of having a date (Cawley, Joyner & Sobal, 2006), dating frequency (Shepperd & Strathman, 1989), having a partner and multiple long-term relationships (Nettle, 2002*a*), being married (Harper, 2000; Pawlowski, Dunbar & Lipowicz, 2000; Herpin, 2005), having multiple wives (Mueller & Mazur, 2001) and having a partner who scores more highly on relevant mate-choice characteristics (Oreffice & Quintana-Domeque, 2010; Stulp *et al.*, 2014). Curvilinear effects of height on the age at marriage and probability of marriage are also observed (Stulp *et al.*, 2012*c*; Manfredini *et al.*, 2013). For women, shorter individuals have been shown to be less likely to date (Cawley *et al.*, 2006), but also to be more preferred as dates or more frequently dated (Shepperd & Strathman, 1989). Furthermore, taller women have been reported to have lower probabilities of marriage (Harper, 2000; Stulp *et al.*, 2012*d*) and a later age at marriage (Stulp *et al.*, 2012*d*). Also average-height women have been observed to have the highest number of relationships (Nettle, 2002*b*). As observed for the mate preferences with respect to stature, results are not consistent across the globe: whereas in African pygmies and rural Gambian men, height is positively related to number of marriages (Sear, 2006; Becker *et al.*, 2012), among the Hadza height was not related to probability of marriage (Sear & Marlowe, 2009).

If we look at patterns of height within couples, we also find that many of the above preference rules are realized in actual couples, suggesting that laboratory-based preferences play out in the real world. For instance, assortative mating is widely observed in industrialized populations (Gillis & Avis, 1980; Spuhler, 1982; McManus & Mascie-Taylor, 1984; Silventoinen *et al.*, 2003*a*; Oreffice & Quintana-Domeque, 2010; Courtiol *et al.*, 2010*a*; Stulp *et al.*, 2013*c*). Furthermore, both the male-taller and male-not-too-tall norms are found in actual couples (Gillis & Avis, 1980; Sear, 2006; Becker *et al.*, 2012; Stulp *et al.*, 2013*c*). In addition, partner height differences have been shown to be dependent on an individual's own height in a sample of actual British couples (Stulp *et al.*, 2013*c*).

There are two relevant points to add. First, realized associations with respect to height between partners are much weaker than the associations found in preference studies (Courtiol *et al.*, 2010*a*,*b*; Stulp *et al.*, 2013*c*), particularly for women (Courtiol *et al.*, 2010*a*) implying that such preferences are only moderately realized in actual couples. In addition, non-random pairing with respect to height is not always apparent in non-industrialized populations. Although there is support for assortative mating and the male-taller norm in some non-Western populations (Mexico: Malina *et al.*, 1983; Bolivia: Godoy *et al.*, 2008; Pakistan: Ahmad, Gilbert & Naqui, 1985; Cameroon: Pieper, 1981; Becker *et al.*, 2012), there are also a number of studies that show no evidence of these effects (Korea: Hur, 2003; Gambia: Sear, Allal & Mace, 2004; Hadza: Sear & Marlowe, 2009).

As already noted, it seems likely (and indeed obvious) that individuals take into account a variety of other characteristics besides height when choosing a mate. Having said this, although the effects are modest, height does play a role in the partnering process in at least some populations. Combined with the evidence relating to resource acquisition, health and mortality risks, it is therefore worth asking whether height significantly influences reproductive success in contemporary humans.

(3) Does height influence reproductive success?

Before we address whether height is associated with reproductive success, we should briefly mention a few further caveats relating to methodological issues. There are, for example, problems of non-representative samples (e.g. the inclusion of only healthy individuals and those that have not yet completed their reproductive lifespan, the exclusion of childless individuals, delinquents, twins) that may affect results, although the exact manner in which they may do so is often unclear. There is also a lack of control of potentially confounding variables in some studies (e.g. education and income), and many have small sample sizes. As selection gradients are typically low (Kingsolver *et al.*, 2001), substantial samples are required to detect any effect; some studies, therefore, may simply be underpowered. Thus, a certain degree of caution is needed when interpreting many of the available studies. For a more thorough discussion of these issues, see Stulp *et al.* (2012*b*,*c*,*d*).

(a) *Female height and reproductive output*

The relationship between female height and reproductive output was assessed in 29 populations [Table 1; this is an extension of that in Stulp *et al.* (2012*d*), see their study for further details]. Even brief inspection of Table 1 reveals that the results are highly variable, with null, positive, negative and curvilinear effects all reported. This may arise at least partly from methodological factors, most notably variability in age ranges and whether only parous women were included in the sample. These caveats aside, it is clear that most variability is found among non-Western nations, whereas among Western populations, there is a more consistent negative association between female height and reproductive success (in those cases where a significant relationship is found) (Stulp *et al.*, 2012*d*).

High levels of variability in the relationship between height and reproductive success across populations are perhaps also to be expected (if they are not simply the result

Table 1. A summary of studies on the relationship between height and reproductive success

Study	Population	Female height				Male height			
		N	Effect	Average	Optimum	*N*	Effect	Average	Optimum
Lasker & Thomas (1976)	Mexicans who have lived in USA	147	–	∼151		∼215	–	∼162	
Shami & Tahir (1979)	Pakistan[a]	827	Curv	∼152–153	154–155	860	Curv	∼164–165	166–167
Mueller (1979)	Columbia[a]	349	–	?		338	Curv	?	?
Mueller, Lasker & Evans (1981)	Mexicans in Mexico and USA	121	–	?		159	–	?	
Martorell *et al.* (1981)	Indians in Guatamala[a]	380	Pos	142	153				
Brush, Boyce & Harrison (1983)	Papua New Guinea	152	Curv	151	151				
Goldstein & Kobyliansky (1984)	Mexicans in Mexico and USA	230	–[b]	?		230	–	?	
Devi *et al.* (1985)	India	291	Neg	161	140				
Baqui *et al.* (1994)[b]	Bangladesh	2417	–	149					
Winkler & Kirchengast (1994)	!Kung san from Namibia					114	–	161	
Kirchengast & Winkler (1995)	Urban !Kung san					59	Neg	171	159
Kirchengast & Winkler (1995)	Rural !Kung san					78	Pos	171	182
Kirchengast & Winkler (1996)	!Kung san from Namibia	93	Neg	149	139				
Kirchengast & Winkler (1996)	Kavango from Namibia	85	Neg	160	150				
Kirchengast (2000)	!Kung san from Namibia	65	Neg	149	149	103	–	160	
Sear *et al.* (2004) and Sear (2006)	Rural Gambia	216	Pos	158	∼167	303	–	168	
Pollet & Nettle (2008)	Rural Guatamala	1989	Curv[c]	147	148				
Fielding *et al.* (2008)	China	6709	Neg	154	144	2620	Neg	165	153
Sorokowski, Sorokowska & Danel (2013)[b]	Yali West Papua	54	–	151		52	–	141	
Becker *et al.* (2012)	Baka pygmies and	∼99	–	146		∼99	–	156	
	Nonpygmy Nzimé			158				168	
Courtiol *et al.* (2013)[b, d]	Rural Gambia	2818	Var	∼158					
Clark & Spuhler (1959)	USA	324	–	?		136	Curv	∼172	∼172[e]
Damon & Thomas (1967)	USA					2616	Curv	∼173	178
Bailey & Garn (1979)	USA	∼1261	Neg	158	∼146				
Scott & Bajema (1982)	USA	600	–	162		621	Curv	177	∼176
Pawlowski *et al.* (2000))	Poland					3201	Pos	172	∼185
Mueller & Mazur (2001)	USA					322	Pos	∼178[f]	∼190[f]
Nettle (2002*a*,*b*)	UK	3554	Neg	162	151	4586	No	177	
Deady & Law Smith (2006)	Caucasians; UK, USA, Canada, Australia	315	Neg	164	150				
Helle (2008)	Finland	271	No	164					
Genovese (2008)	USA					192	–	?	
Nenko & Jasienska (2009)	Rural Poland[a]	328	–	159					
Byars *et al.* (2010) and Stearns *et al.* (2012)[b]	USA	2227	Neg	161	148	2655	Curv	174	∼174
Stulp *et al.* (2012*c*,*d*)	USA	4069	Neg	164	152	3578	Curv	179	177
Stulp *et al.* (2012*b*)[b,g]	USA	1853	Neg	164	152	1669	Curv	179	177
Silventoinen *et al.* (2013)[a]	Swedish twins	4236	–	164		3600	Curv[h]	177	∼177

See Stulp *et al.* (2012*a*,*b*) and original studies for more elaborate description of samples and how the effect of height was established. Studies above the middle line are considered non-industrialized and non-Western.

Effect refers to the effect of height on reproductive success (Pos: positive; Neg: negative; Curv, curvilinear; –, no effect observed; Var, variable selection pressures over time observed).

Optimum refers to those heights that were associated with highest reproductive success, either by determining optimum of curve, or identifying height bin associated with highest fertility. When a positive/negative association of height was observed, heights of 2 S.D. above or below the means are reported as the optimum.

[a]Only individuals with at least one child were included.

[b]Not included in reviews of Stulp *et al.* (2012*a*,*b*).

[c]In Stulp *et al.* (2012*a*) it was wrongly concluded that the effect was positive and that the optimum was at tall heights (this held true for child survival, not for the number of children ever born). On the basis of the regression equation provided in Pollet & Nettle (2008), it is clear that average-height women have higher fertility with an optimum at 151 cm.

[d]This study used similar data to those used in Sear *et al.* (2004), although the analyses were very different (see text for discussion).

[e]Estimate based on graph as provided by Mitton (1975) who reanalysed the data of Clark & Spuhler (1959).

[f]Actual heights were not available, but height was measured in percentiles. Height admissions for the military were between 157 and 198 cm, and we determined heights on the basis of these values.

[g]This study is not independent of Stulp *et al.* (2012*a*,*b*) since it uses data on brothers and sisters.

[h]Poisson regressions revealed no effects, but men (male twins) in the third quintile were more likely to have a child, and to have one or two children. The effect of height was very weak and almost non-existent in the graph.

of methodological differences) because selection pressures are also likely to differ substantially across space and time (Siepielski, DiBattista & Carlson, 2009). Moreover, from a life-history perspective, a single height optimum across all environments is inherently unlikely: the trade-offs between growth, survival and reproduction should give rise to variable body size optima that are dependent on environment-specific levels of resource abundance and mortality regimes (Sear, 2010). That is, the height that maximizes reproductive success within a population should be expected to differ from other populations, given local mortality regimes and ecological conditions. It is, however, interesting to consider how the heights that maximize reproductive success vary in absolute terms as this helps us appreciate that what constitutes tall or short height in one population may not do so in another. In addition, it allows us to gain some insight into whether female reproductive success is maximized within a particular range of heights across all populations, regardless of the nature of the relationship within a particular population.

Inspection of Table 1 reveals that there is perhaps less variability in the absolute heights that maximize reproductive success than one might imagine, given the variability in the direction of the association across populations. 'Average-height' to 'short' women in a Western context, for example, would not be considered short relative to their counterparts in other regions, and would even be considered as relatively tall in some of them. Equally, among those non-Western populations where women of average height show the highest reproductive success, such women would be considered relatively short in a Western context; indeed the height of such women approximates the height of the shortest, most reproductively successful women in the USA (Stulp *et al.,* 2012*d*). Similarly, 'tall' women who do better reproductively in a non-Western context fall within the range of average to short height in a Western context. Although speculative, one can hypothesize that the trade-off between growth and reproduction may therefore reflect some upper physiological limit that applies across all populations, regardless of ecological conditions, in much same way that the mechanical limit of pelvis size is hypothesized to constrain the minimum height that women must achieve. Although good conditions may permit growth to continue beyond the minimum threshold needed to support reproductive functioning and so increase productivity, it may be possible to extend such growth only so far before it begins to have a negative effect on reproductive output because of the delay in sexual maturity. This is most likely to be due to a reduction in the overall length of the reproductive lifespan, which is far less flexible at the tail-end with respect to the onset of menopause, than at the beginning with the onset of menarche (Morabia & Costanza, 1998; Burger, DeLong & Hamilton, 2011). Growing very much taller than the minimum height required for successful reproductive functioning (i.e. tentatively, beyond the heights of 1.60–1.70 m seen here) may therefore require exceptional environmental conditions that allow for more rapid growth both prior to and after the threshold is reached (i.e. a faster growth velocity prior to menarche, and a slower decline in growth once this has been reached). It is possible that conditions in some Northern European countries, particularly the Netherlands, where the secular trend in height apparently is on-going (Fredriks *et al.,* 2000; but see Schönbeck *et al.* 2012), approximate such conditions. Here, women may be able to grow far beyond the minimal pelvic threshold to a height that significantly reduces the risk of obstetric problems *via* increased pelvic width, while simultaneously allowing them to benefit from the increased fecundity associated with larger body size.

Outside of such exceptional conditions, where reproductive-success-maximizing heights are short relative to the population average, an earlier start to reproduction appears to compensate for increased mortality risk among short women. In line with this, in some high-income populations, shorter women tend to reach menarche earlier and reproduce at a younger age than women of taller stature, suggesting they are 'ready for reproduction' at an earlier age than their taller counterparts. As age at first birth is a crucial factor in determining overall reproductive success among women, especially in low-fertility industrial nations (for a list of studies, see Stearns *et al.,* 2010), selection may thus favour women who terminate growth earlier, and so achieve a younger age at first birth. Further weight is added to this argument by the observation that a genetic correlation exists between female height and age at first birth. That is, women who bear their first child at an early age also show a genetic predisposition to be shorter than average (Stearns *et al.,* 2012). Thus, as Stearns *et al.* (2012) point out, both the phenotypic (i.e. the life-history trade-off) and genetic correlation between height and age at first birth may explain why shorter women often attain higher reproductive success. This seems to be particularly true in contemporary industrial societies, where this head-start more clearly outweighs the higher mortality risks they experience; possibly due to advanced medical care which helps to minimize these risks.

There is also good evidence to suggest that, for women who are tall relative to their population average, increased survivorship could potentially compensate for a delayed start to reproduction in environments where mortality is high. A positive association between female height and child survival probabilities is well established for both developing countries (42 countries: Monden & Smits, 2009) and low- to middle-income countries (54 countries: Özaltin *et al.,* 2010; but see Devi, Kumari & Srikumari, 1985). In poor-quality environments, height is likely to be a reflection of health and nutritional status (Silventoinen, 2003; Sear *et al.,* 2004), and hence a greater ability to access resources, both of which are likely to have a positive influence on childhood survival and reproductive success (Martorell *et al.,* 1981). In line with this, Sear *et al.* (2004) found that taller women in the Gambia achieved higher reproductive success than short women, despite a later age at first birth. In this case, taller women not only experienced higher rates of child survival, but were also able to compensate for their delayed start by giving birth to more children overall. It is interesting to note, however, that

Courtiol *et al.* (2013) show a different relationship between height and fertility over the same period, using the same data set. In their analyses, which tracked changes in selection across the demographic transition, they found that, over the same period studied by Sear *et al.* (2004) (~1950–1975), selection favoured shorter women with a higher BMI. From 1975 onwards, this relationship shifted, with selection favouring taller women with a lower BMI (possibly linked to the establishment of a medical facility in 1975). Comparison of these two studies is complicated by the fact that different data-selection procedures were used, which may account for the contrasting patterns seen. In essence, Sear *et al.* (2004) show that taller women born in 1925 or earlier, who survived to the age of 50, had most children, whereas Courtiol *et al.* (2013) show that, for all women born before 1975, when assessed on a yearly basis, selection appears to favour shorter women.

Although height is highly heritable, and a genetic response is likely, these kinds of phenotypic correlations with fitness do not necessarily reflect a genetic correlation with fitness (Morrissey, Kruuk & Wilson, 2010). To establish whether selection on height will generate a genetic response in the next generation, data on families or actual genomic data is needed. Byars *et al.* (2010) represents one of the only examples of such a study: using a sample of USA women from the longest running multi-generational study in medical history, they showed that natural selection is operating in women to drive height downwards.

Another factor to consider when discussing how height might influence reproductive success is the ability to attract a mate and form a reproductive partnership. Stulp *et al.* (2012*d*), for example, found that taller women in a USA population were more likely to remain unmarried and, among those that did marry, they tended to marry late. Age at marriage cannot, of course, be considered an accurate proxy of mate attractiveness or value. Rather, it may equally well signify the selectivity of the woman herself. Taller women, at least in some populations, may be more selective, and less likely to 'settle', than shorter women. Indeed, given that shorter women appear to have a genetic predisposition to bear children at an earlier age, it seems plausible that this same biological inclination affects other aspects of their physiology and psychology as well (Deady & Law Smith, 2006): shorter women perhaps possess higher fertility motivations, which manifest at an earlier age. Shorter women in industrial societies may therefore show greater motivation to find a committed partner and begin a family while still very young.

(*b*) *Male height and reproductive output*

For men, data were available for 24 different populations (Table 1). Again, as for females, a variety of effects were reported, and again, there was more consistency in Western than in non-Western populations (for full discussion, see Stulp *et al.,* 2012*c*). For USA populations, in particular, average-height men tend to show the highest reproductive success. Given the above associations between male height and enhanced access to resources and mates, a positive relationship between male height and reproductive success within certain populations is not hard to explain. Equally, given our consideration of life-history trade-offs and the potential benefits of short stature in offsetting mortality risks against future reproduction, a relationship between short stature and greater reproductive output can also be accounted for under conditions where overall mortality risks are high for particular age-classes. In addition, temperature, nutritional stress, manoeuvrability through forests, and high adult mortality have all been suggested as examples of ecological factors that may favour shorter heights (e.g. Cavalli-Sforza, 1986; Bailey *et al.,* 1989; Diamond, 1991; Perry & Dominy, 2009). Explaining why men of average height should experience higher reproductive success in contemporary societies is rather more of a puzzle.

Some clues are offered by the detailed analysis of Stulp *et al.* (2012*c*) on the Wisconsin Longitudinal Sample. Although height was not related either to being married or to the total number of marriages, average-height men married at an earlier age than shorter and taller men. Controlling for age at marriage attenuated the relationship between height and reproductive success, whereas controlling for education and income did not, suggesting that the relationship was at least partly mediated by marrying earlier and hence experiencing an earlier age at first birth.

One possibility, then, is that the life-history trade-off between growth and reproduction, which partially seems to explain female stature and reproductive success, may similarly affect male height. Selection seems to favour a younger age at first birth in men, and this may be tied to stature in a similar way as for women (including phenotypic and genetic association between height and age at first birth: Stearns *et al.,* 2012). The resulting curvilinear relationship of reproductive success and height may therefore be a consequence of both positive selection on height through, for example, health, attractiveness and income, combined with negative selection on height with respect to earlier age at first birth. This is, however, purely speculative. In addition to the relationship between growth and reproduction, height also may be (genetically) correlated to other traits under selection (Lande & Arnold, 1983). Thus, the specific relationship between stature and reproductive success may be a consequence of selection on other traits related to height. Unfortunately, these potential traits are not yet readily identified.

Another possibility that may account partially for why average-height men attain higher reproductive success, is that they not only by marry at a younger age, but also reproduce with women of higher reproductive potential. Although shorter women prefer men much taller than themselves, this does not equate to a preference for tall men *per se* (Stulp *et al.,* 2013*a*,*b*). Indeed, shorter women tend to prefer men of average height. Similarly, taller men prefer women who are shorter than they are, but not necessarily short women. These specific partner-height preferences in relation to one's own height may result in shorter women being more likely to be paired to men of average height, rather than tall men.

There is some evidence to support this notion: first, shorter women are most likely to match with average-height men at speed-dating events (Stulp *et al.*, 2013*a*) and second, shorter women are more likely to have average-height and shorter men as actual partners than taller men (Stulp *et al.*, 2013*a*,*c*). Thus, the increased reproductive success of average-height men compared to taller men may be a consequence of their increased likelihood of pairing with shorter women, who produce a larger number of children (Stulp *et al.*, 2012*d*).

Following this line of reasoning, one could then argue that shorter men, who are also most likely to be coupled to shorter women, should also have increased reproductive success, but this is not the case. Reduced reproductive success in shorter men may be a consequence of other factors disfavouring short height, such as lower health status and income, being less preferred as a mate, and obtaining a mate of lesser quality. Stulp *et al.* (2014) recently found support for these predictions, with shorter men displaying a 'double disadvantage' in terms of both their own quality and that of their spouses. In addition, Stulp *et al.* (2014) found that differences in mate quality between average-height and taller men were only marginal. Thus, the advantage accruing to height seems to come simply from not being short, rather than being taller than average. Taken together, these findings suggest that the curvilinear pattern between height and reproductive success in contemporary USA populations is unlikely to be a consequence of a single selection pressure, but is more likely to be the result of competing pressures that act differentially along the height continuum to produce the observed effects on male reproductive success.

We should again stress that the above explanations are purely speculative, and require thorough testing. Moreover, we have no evidence to suggest that such patterns extend beyond USA populations. As more and better information of this nature becomes available, it seems likely that a clearer picture of the manner in which height translates into reproductive success will arise. Even without a fully convincing explanation, however, the contrast between the selection pressures acting on female height and those acting on males in contemporary USA populations is notable, and may have a number of evolutionary consequences.

(*c*) *Intralocus sexual conflict over height*

The existence of contrasting selection pressures acting on male and female height creates the potential for intralocus sexual conflict (IASC). IASC arises when there is differential selection on a trait for males and females, but the genetic expression of a trait is determined similarly in each sex (i.e. there is a strong intersexual genetic correlation: Bonduriansky & Chenoweth, 2009). As a result, selection pressures in one sex constrain the other from achieving its sex-specific fitness optimum.

Given the high heritability of height in industrialized populations (Silventoinen *et al.*, 2001; Silventoinen, 2003) and the fact that tall parents produce tall sons and tall daughters, and short parents produce short sons and daughters, something along these lines may occur in humans. That is, whether shorter or taller height is beneficial depends on which sex expresses the trait (Rice & Chippindale, 2001). Stulp *et al.* (2012*b*) addressed this issue by investigating the reproductive success of siblings in the same USA sample that revealed contrasting selection pressures on the sexes. In short sibling pairs, greater reproductive success accrued to the female sibling compared to the male sibling, whereas in average-height sibling pairs greater reproductive success accrued to the male sibling compared to the female sibling. Sexually antagonistic selection with respect to height may, then, be a feature of the selective landscape for some modern human populations.

Having said this, it is important to emphasize that the existence of different phenotypic selection pressures between the sexes is not sufficient to demonstrate IASC; it is also necessary to show that the trait has significant genetic variation, that there is a strongly positive intersexual genetic correlation (i.e. such that independent responses to selection by each sex will be constrained), and that the genetic covariance between relative fitness and the trait in question is positive in one sex but negative in the other (Bolund *et al.*, 2013). An evolutionary response will only occur if at least part of the phenotypic relationship between the trait and fitness has a genetic basis (Morrissey *et al.*, 2010). Stearns *et al.* (2012) found that patterns of selection on height differed between the sexes, with stabilizing selection on male height and negative directional selection on female height, and a positive cross-sex genetic covariance for this trait. Despite these patterns, no significant genetic conflict for height was apparent. Thus, it is possible that the population studied by Stulp *et al.* (2012*b*) may show the same effect, with phenotypic, but not genetic, sexual conflict.

(4) Is height a target of selection?

We are now in a position to attempt an answer to the question of whether height is a trait of evolutionary significance, with adaptive value of its own. There is some evidence to suggest that variation in stature represents an evolutionary adaptation to climatic conditions, although the best current evidence suggests this applies only in the Northern hemisphere. In most cases, the evidence points to height variation as the expression of an evolved reaction norm reflecting a life-history trade-off between growth, maintenance and reproduction (Walker *et al.*, 2006). This is particularly true in the case of women, where height derives much of its evolutionary significance *via* processes relating to age at first birth and infant survival. Among contemporary populations, the lack of strong preferences among men for female height also suggests that sexual selection on height currently operating on women is unlikely to be very strong. On balance then, in terms of selective processes, natural selection acting on life-history events seems more likely to explain variation in height among women than sexual selection. At present, such inferences remain largely speculative, however, given we lack any studies that aim to test explicitly between these alternative explanations. In addition, it is of course possible that other non-selective evolutionary forces, like drift, may play a role.

For men, it is also apparent that similar life-history trade-offs to those that apply to women may also influence

stature in men, given cross-cultural relationships between height and environmental quality that are consistent with trade-offs between growth, maintenance and reproduction (Walker *et al.*, 2006). In addition, there is some evidence to suggest that the link between stature and greater reproductive success is indicative of sexually-selected female choice for height (e.g. Pawlowski *et al.*, 2000; Mueller & Mazur, 2001). Indeed, positive relationships between height, health, and various measures of social status make it at least plausible that height could be a target of selection. For certain industrialized societies, however, where males of average height are currently favoured by selection, this argument cannot apply. In such cases, taller men are preferred as partners, and are more economically successful than those of shorter stature, yet show lower reproductive success. Of course, this does not mean that sexual selection for height did not operate in the past, and present patterns cannot be used to infer past selection. What we can say, however, is that the increased reproductive success of average-height men in these populations may be tied to their early age at first birth, which in turn may be linked to an increased likelihood of pairing with shorter women, who themselves have higher fitness (Stulp *et al.*, 2012*d*). Why, then, do taller men experience a later age at first birth, particularly as they seem favoured as mates? One possibility is that age at first birth and stature may be tied to life-history trade-offs similar to those found in women. Moreover, cultural factors may promote slower life histories for taller men, and they may enter the marriage market later than their shorter counterparts. Currently, such explanations remain speculative, and the association between male stature and reproductive success across populations, as is true for women, remains something of an enigma.

These results also have to be placed into a broader perspective, as the curvilinear and negative relationships between height and reproductive success come from a limited number of Western societies, most of them in the USA. As discussed above (Section II.2*b*), Americans have been getting shorter, rather than taller, over the past century; a finding that cannot be attributed purely to increased levels of immigration (especially as the children of immigrants grow very much taller than their parents). The finding that selection consistently favours both shorter female height and average male height in this population fits well with this curious reversal in height trends, but it also suggests that, given the ongoing positive secular trend in other countries, particularly the Netherlands, selection pressures in these populations may well favour taller heights.

VII. CONCLUSIONS

(1) Although height is a trait that is easily measured and data are widely available, it is apparent that we need more consistent reporting of height across studies that will allow for more rigorous and stringent tests of evolutionary hypotheses, particularly those relating to sexual dimorphism, Rensch's rule and various aspects of life history. Both major methodological differences among studies and the various biases introduced by selection criteria and sampling procedures mean it is difficult to make any general statements about evolutionary processes.

(2) There is evidence to suggest that natural selection is acting on height in contemporary human populations, and hence that differences across populations may reflect more than just the expression of a flexible reaction norm. Effect sizes are, however, low, which raises the question of whether selection pressures are sufficiently strong to explain the patterns seen. Theoretical modelling is needed to help answer this question. More empirical studies of height are needed to assess the degree to which height is under selection in other populations, and whether selection pressures differ. In addition, such studies require large sample sizes and fewer biases in inclusion criteria than those currently available, and many existing studies require replication.

(3) Our understanding of, and attitudes toward, short stature is clearly a vexed issue. While an evolutionary approach considers it appropriate to view small bodies as the result of life-history trade-offs, this does raise questions of how this relates to adult health and socioeconomic functioning, and whether we should view short stature as adaptive or simply an accommodation to poor conditions. Equally, however, we can question ideas that 'bigger is better' should apply across the board, and that shorter stature is always an indicator of poor functioning, when viewed in a cross-cultural evolutionary perspective. This issue, in particular, highlights the fact that height cannot be considered as a purely biological trait, but one that carries significant sociocultural and political weight.

(4) Current trends relating to stature and sexual maturity across industrialized countries present an unsolved puzzle, and more and better individual-level data are needed to test the life history hypotheses put forward potentially to explain it.

(5) Work on the genetics of height has revealed that it is a truly polygenic trait, to an even greater extent than initially suspected. The advent of GWAS is making it possible to trace the biological pathways that influence height, and will help shed light on the links between environmental conditions, growth trajectories, skeletal maturity and the shift from growth to reproduction.

(6) For such a well-studied and easily measured trait, there remain some surprising gaps in our knowledge concerning the evolution and adaptive significance of height. This suggests we should be cautious in our acceptance of evolutionary (and specifically, adaptationist) claims made about other human traits, particularly those relating to our behaviour and psychology.

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