Eric B. Schneider

Children's growth in an adaptive framework: explaining the growth patterns of American slaves and other historical populations

Article (Accepted version)
(Refereed)

CHILDREN'S GROWTH IN AN ADAPTIVE FRAMEWORK: EXPLAINING THE GROWTH PATTERNS OF AMERICAN SLAVES AND OTHER HISTORICAL POPULATIONS

Eric B. Schneider[1]

Assistant Professor, Economic History Department
London School of Economics and Political Science

e.b.schneider@lse.ac.uk

1 September 2016

Abstract

This paper presents a new adaptive framework for understanding children's growth in the past. Drawing upon the recent work on adaptive responses in relation to growth, I present prenatal and postnatal adaptive mechanisms that affect the growth patterns of children. The most novel adaptive response to the historical literature is the prenatal predictive adaptive response where the metabolism and growth trajectory of a child is programmed to match predicted conditions later in life. Having discussed the framework in detail, I then suggest a reinterpretation of the growth pattern of American slaves. It seems likely that a mismatch between relatively good conditions *in utero* and absolutely appalling conditions in infancy and early childhood led slave children to become extremely stunted by age three or four. However, after this age, slave children experienced catch-up growth because their immune systems had become more developed and because their diet improved tremendously and hookworm exposure was reduced. Thus, it seems that American slave children may have experienced substantial catch-up growth because they were prenatally programmed for a higher metabolism and growth trajectory. The paper concludes by setting out some stylized facts about children's growth in the past and pointing toward areas of future research.

Human growth is an extremely complex process driven by both genetic inheritance and epigenetic adaptation (in the widest sense) to environmental conditions. Growth is influenced for instance by parental growth patterns, the pelvis size of the mother, maternal health and nutrition during pregnancy, morbidity and infection, physical exertion, elevation, and exposure to cold environments. Human biologists, anthropologists, economists and anthropometric historians have tended to treat these inherited and environmental factors as intrinsically separate. Because environmental factors explained more of the variation in mean heights than genetic differences within a like population, these scholars mostly ignored inherited influences on height to focus on the effects of varying environmental conditions on nutritional status.[2] This omission may be a useful heuristic device in studying variation in adult stature, but it seriously oversimplifies the complex interactions between environmental and inherited characteristics and their influence on human growth.

In order to understand these interactions, we must begin considering adaptive models of prenatal and postnatal growth. Within an adaptive framework, it is possible to understand how environmental conditions trigger certain genetically and epigenetically encoded life strategies, which, in turn, affect final height, longevity, and morbidity throughout the life cycle. An adaptive framework for growth can also help historians to make sense of the varied patterns of growth in historical populations. For instance, it can help explain why the timing of the pubertal growth spurt varies widely across populations and why some groups of children experience catch-up growth when others do not.

In this paper, I use an adaptive framework for understanding children's growth to reinterpret the 'peculiar' pattern of slave children's growth in antebellum America and the Caribbean. In a series of papers in the 1970s and 1980s, Steckel traced the basic outline of slaves' growth pattern. He imputed slave birth weights from other evidence arguing that they

---

[2] Stinson, 'Growth Variation', p. 456; Silventoinen *et al.*, 'Genetic Regulation', p. 292.

were extremely low compared to modern populations. However, despite slave children's low starting point, they experienced remarkable catch-up growth after entering the labour force around age ten, attaining final adult heights taller than many of their European working-class contemporaries.[3] In essence, I argue that the tremendous catch-up growth experienced by slaves is inconsistent with Steckel's assertion that slave birth weights, a proxy for health *in utero*, were incredibly low. Adaptive theories of human growth suggest that poor conditions *in utero* have long-lasting effects on the growth pattern of children, especially in limiting catch-up growth. Thus, it is more likely that slave children experienced relatively good conditions *in utero* and had higher birth weights than Steckel argued. Terrible health conditions in infancy and early childhood led the children to become extremely stunted, but the combination of prenatal adaptations for a tall adult height and improvements in their diet and environment made their remarkable catch-up growth possible. After discussing this case study, I will draw on secondary literature to develop four stylized facts about children's growth patterns in the past and place these stylized facts within the adaptive framework of growth. The paper concludes by offering some ideas about methods and topics ripe for future research.

I

Before delving into the prenatal and postnatal adaptive models of children's growth in detail (presented schematically in Figure 1), it is first necessary to explain the concept of adaptation from a biological perspective. Functional adaptations are adaptations that improve an organism's fitness or functional viability within a specific environment during an organism's lifetime.[4] Thus, they are adaptations that increase the probability that an organism will survive to reproductive age and produce viable offspring. Immediate adaptations confer

---

[3] Steckel, 'Peculiar Population'; Steckel, 'Birth Weights'.
[4] Frisancho, *Human Adaptation*, pp. 4-5; Huss-Ashmore, 'Theory', pp. 13-15.

immediate functional benefit to the organism but could have long-term costs for fitness later in life. Predictive adaptive responses attempt to align the organism's physiology with later environmental conditions, conferring long-term advantages, but not necessarily improving the immediate fitness of the organism.[5] Whether adaptations confer improved fitness to the organism in both the short and long run is not an absolute but a relative calculation.[6] The adaptive mechanisms presented in this paper increase a person's chances of surviving to reproductive age and reproducing, but they also have some negative long-term consequences on health after the typical period of reproduction. Thus, functional adaptations do not necessarily maximize life expectancy or health over the entire life cycle.

Insert Figure 1 here

There has been growing evidence in the past decade that conditions in the prenatal period are extremely important to growth and health later in life. David Barker initiated much research and debate when he published his hypothesis, sometimes known as the 'early origins hypothesis' or 'fetal programming', suggesting that fetuses limit their growth and permanently change their physiology and metabolism based on the availability of nutrients in the womb.[7] Barker further argued that these physiological and metabolic changes could be responsible for higher incidences of coronary heart disease, hypertension, stroke, and diabetes.[8] Since then, there has been a tremendous amount of scholarship published attempting to explain the consequences of poor health conditions *in utero* for longevity, height and morbidity across the life course.[9]

---

[5] Gluckman *et al.*, 'Environmental Influences'.
[6] Stinson, 'Nutritional Adaptation', p. 161.
[7] Barker, 'Fetal and Infant Origins'.
[8] Barker, 'Maternal Nutrition'.
[9] Gluckman and Hanson, 'Consequences'; Almond and Currie, 'Killing'; Quaranta, 'Early Life Effects'.

The complex and varied results of this research agenda allowed Gluckman and Hanson to place Barker's Hypothesis in an adaptive framework. They argued that there are three ways of interpreting a fetus's response to poor environmental conditions in the womb. First, if nutrient conditions in the womb are extremely dire, the fetus could experience developmental disruption, which is not adaptive and can lead to severe birth defects. Second, an immediate adaptive delay in growth might allow the fetus to survive a short-term nutritional insult that would have killed it if it did not curtail its growth. Third, low nutrient levels in the womb could trigger a predictive adaptive response in the growing fetus where nutrient conditions *in utero* are taken to be a good prediction of future conditions and the growth, metabolism, and physiology of the fetus are adapted to best fit that predicted environment.[10] In other words, if a fetus is undernourished in the womb, epigenetic and hormonal processes adapt it to be able to survive better in a postnatal environment with limited nutrients.[11]

There are two sets of long-term consequences of predictive adaptive responses *in utero*: the first affects the child's growth pattern during the growing years. Children exposed to poor conditions *in utero* have a slower metabolism, reach a shorter adult stature that requires fewer calories to maintain and experience faster maturation and an earlier age at menarche so that the fetus reaches reproductive viability early and has a better chance of reproducing before death.[12] The second set of long-term consequences of a prenatal predictive adaptive response influence children's long-term health and productivity. As mentioned above, Barker and others have found that exposure to poor conditions in early life

---

[10] The theory of predictive adaptive responses (PAR) is not without its detractors. Bogin *et al.*, 'Life History' argued that existing evidence does not support PAR, but most of their evidence was drawn from the INCAP Guatemalan intervention studies, which have experimental design flaws that make the results difficult to interpret and undermine many of the inferences that they ascribe to it. See also Strauss and Thomas, 'Health', pp. 3429-3432 and web appendix for more information.

[11] Gluckman *et al.*, 'Environmental Influences'.

[12] Gluckman and Hanson, 'Consequences', pp. 6-8; Gluckman and Hanson, 'Evolution', pp. 8-9; Gluckman *et al.*, 'Environmental Influences', pp. 672-4; Godfrey, *et al.*, 'Epigenetic Mechanisms', pp. 5-7.

increased the prevalence of heart disease, stroke, and diabetes in later life.[13] Cohorts exposed to poor conditions *in utero* also experience non-adaptive scarring such as cognitive impairments and lower educational attainment and earnings.[14]

Clearly, impaired prenatal growth can have many serious consequences for the postnatal life of the child. However, these consequences, some more negative than others, do not preclude an adaptive framework from the prenatal predictive response. If the fetus predicts that its nutritional environment will be poor in its postnatal life, then limiting its body size so that it requires fewer calories and nutrients to survive is a beneficial response despite some of the negative consequences listed above. The problem arises when there is a 'mismatch' between the fetal predictive response and the actual conditions of the postnatal environment, for instance, a better nutritional environment in postnatal life. This mismatch could increase the risk of developing metabolic diseases, obesity and diabetes. However, significant long-term improvements in environmental conditions were rare at the time this trait evolved, so the prenatal predictive adaptive response was beneficial in its original context.[15]

In addition to the prenatal adaptive responses, there are three potential adaptive responses to environmental conditions in the postnatal period: an immediate adaptive response of limiting growth to minimize the basal metabolic rate, an immediate adaptive response of delaying the pubertal growth spurt and menarche in females in order to postpone sexual maturity; and catch-up growth when environmental conditions improve following a nutritional or disease insult. However, there is an important difference between prenatal and postnatal adaptive responses. Because phenotypic plasticity, 'an environmentally based change' in physical characteristics, is greatest during the embryonic and fetal period of a

---

[13] Barker, 'Maternal Nutrition'; Jirtle and Skinner, 'Environmental Epigenomics'; Dolinoy and Jirtle, 'Environmental Epigenomics'.
[14] Silventoinen *et al.*, 'Genetic Regulation'; Almond and Curry, 'Killing'.
[15] Gluckman and Hanson, 'Consequences', p. 10.

child's development, prenatal responses can have long lasting if not entirely permanent effects on the child's physiology and growth.[16] However, postnatal immediate adaptive responses in growth tend to be more temporary mechanisms to deal with a poor nutritional or disease environment and do not have the same long-lasting effects.[17]

The first postnatal immediate adaptive response is a slowing of growth during a nutritional or disease insult. This is a beneficial adaptive response because the child limits increases to its basal metabolic rate that come with growth in order to conserve energy. However, there are costs to this response, and a child who undergoes postnatal stunting is not healthy. Shorter and underweight children are at a higher risk of disease because malnourishment weakens the immune system.[18] The fitness of a stunted child is not higher than a child who continued to grow normally. Instead, the stunted child has a higher relative level of fitness compared to a child who continued to grow normally despite poor environmental conditions.

The second postnatal adaptive response is to delay the pubertal growth spurt and sexual maturity during times of hardship. Despite the fact that menarche would be programmed to arrive earlier by a poor prenatal nutritional environment, Gluckman and Hanson have argued that menarche can be delayed during periods of poor nutrition in the postnatal environment. This was an immediate adaptive response to postpone reproduction until environmental conditions improved when the high energetic cost of pregnancy and lactation would be less damaging. It would also have the unintended consequence of delaying or slowing the pubertal growth spurt, extending the length of the growing period, and lowering final adult height.[19]

---

[16] Via *et al.*, 'Adaptive Phenotypic Plasticity'; Feinberg, 'Phenotypic Plasticity'.
[17] Gluckman and Hanson, 'Consequences'; Proos and Gustafsson, 'Early Puberty'.
[18] Shetty, 'Malnutrition and Undernutrition', pp. 525-28; Schaible and Kaufmann, 'Malnutrition and Infection', pp. 806-7.
[19] Gluckman and Hanson, 'Evolution'.

The third postnatal adaptive response is catch-up growth that occurs when environmental conditions improve during the growing years. Boersma and Wit defined three types of catch-up growth.[20] Type A catch-up growth is a period of rapid growth after a nutritional insult that attempts to bring the child back in line with its genetically and epigenetically defined growth curve. Type B catch-up growth does not involve growth at more rapid than normal levels. Instead, the growth period is extended longer than normal so that children can attain a higher height-for-age at the cessation of growth. Type C catch-up growth includes both type A and type B catch-up growth.[21] Catch-up growth occurs most commonly among children who are substantially below modern standards in terms of height-for-age and when the nutrition and the disease environment they are exposed to improves during the growing years.

There also appear to be differences in the type of catch-up growth children who face different forms of deprivation can achieve. Low birth weight children, a proxy for health *in utero*, are less responsive to hormones signalling growth, i.e. insulin, growth hormone (GH) and IGF-I, when these hormones are excreted at higher levels to spur catch-up growth following a nutritional insult; in other words, their catch-up growth happens more slowly and is smaller on an absolute scale than normal birth weight children.[22] Prenatal predictive adaptive responses also influence final height potential. Karlberg and Luo found in a study of healthy, full-term Swedish babies that fetal size, measured as length at birth, along with the genetic height potential, measured as midparental height, plays a strong role in defining the final height potential of children.[23] Therefore, it appears that children facing growth retardation and predictive adaptive responses *in utero* have a different growth pattern than children exposed to healthy conditions with earlier maturation, different responses to

---

[20] Boersma and Wit, 'Catch-up Growth'.
[21] Stinson, 'Growth Variation', pp. 451-52.
[22] Gluckman and Hanson, 'Consequences', p. 8.
[23] Karlberg and Luo, 'Foetal Size'.

hormonal growth signalling and a lower final height potential. Finally, several recent studies suggest that improving nutrition and type A catch-up growth in children who suffered malnutrition in both the prenatal and postnatal periods may trigger earlier pubertal development, limiting the amount of type B catch-up growth children could achieve.[24] In any case, catch-up growth is also adaptive because it helps children attain their height potential and confers upon them the advantages of larger body size: greater productivity, health and self-defence.

These adaptive responses in the postnatal period are triggered by environmental conditions that influence the energy and nutrient consumption of the child. General food calorie consumption and a balanced diet are essential for maintaining the basal metabolic rate and sustaining growth. There are different negative effects from protein deficiencies and general shortfalls in dietary energy in part because growth hormones respond differently to each form of poor nutrition.[25] However, other factors such as disease and work can also affect energy and nutrient consumption. Even if a child were not particularly undernourished before contracting a disease, the immune response to the disease would raise his/her energy requirements so that the child must consume more food than before to stay alive and remain healthy.[26] In addition, diarrhoea can significantly decrease dietary energy and nutrient absorption further exacerbating the problems of malnutrition.[27] Child labour can also influence growth patterns if large amounts of energy are required to work.[28] All of these environmental factors can trigger an immediate adaptive response to slow or increase growth velocity.

Clearly, genetic and environmental influences cannot be seen as separate spheres of influence on growth and final adult height. They are both a part of a wider evolutionary

[24] Proos, 'Growth'; Proos and Gustafsson, 'Early Puberty'.
[25] Shetty, 'Malnutrition and Undernutrition', p. 524; Dauncey and Pell, 'Genetic Regulation', pp. 143-144.
[26] Schaible and Kaufman, 'Malnutrition and Infection', p. 806.
[27] Marino, 'Water', pp. 1930-32; Caulfield *et al.*, 'Undernutrition', p. 197.
[28] Steckel, 'Heights and Human Welfare', p. 10.

context. Adaptive mechanisms are built into human physiology to adapt individuals to poor nutritional and disease conditions in both the prenatal and postnatal growth periods. Anthropometric historians can benefit from understanding the conditions that lead to adaptive responses and evaluating what the benefits and costs of any adaptation could be.

II

The previous section set out an adaptive framework for understanding children's growth and attempted to explain how some of these mechanisms might have worked. The next three sections seek to demonstrate the utility of this framework by using it to suggest a reinterpretation of the growth pattern of slaves in antebellum America and in the Caribbean. The analysis will build upon Steckel's original pioneering research as well as more recent debates about slave children's health.

Steckel was the first to study the heights of slave children and compare them with modern growth standards, reconstructing the growth pattern of slave children. To do this, he drew upon a large number of ship manifests created to monitor the coastwise slave trade after the Atlantic slave trade was abolished in 1807.[29] These manifests provide cross-sectional height measurements for all slaves shipped, including children. Steckel found that slave children were very stunted at birth and in early life. Slave boys and girls were 3.23 and 2.89 standard deviations below modern standards (recalculated according to 2006/7 WHO standards) respectively at age 5.5.[30] In addition, because there is no evidence of slave birth

---

[29] There has been a lengthy discussion in the literature about potential selection bias in these sources. However, the general finding is that the magnitude of the catch-up growth would be the same as or larger than Steckel described. Thus, for the purposes of this paper, any potential selection bias will not influence the arguments. Pritchett and Freudenberger, 'A Peculiar Sample'; Pritchett, 'Interregional'; Calomiris and Pritchett, 'Preserving'; Komlos and Alecke, 'Economics', pp. 455-457; Bodenhorn *et al.*, 'Problems'; Steckel and Ziebarth, 'Trader Selectivity'; Pritchett and Freudenberger, 'Reply'.

[30] Steckel, 'Peculiar Population', pp. 724-25. The WHO Z-scores are calculated from the WHO's 2006 growth standards for preschool age children ages 0-5 and 2007 growth references for preadolescent and adolescent children ages 5-19. The 2006 growth standard was based on the WHO Multicentre Growth Reference Study (MGRS), which compared the growth of high SES, healthy children in Brazil, Ghana, India, Norway, Oman and the United States. The 2007 growth reference is based on the 1977 NCHS reference for US

weights, Steckel used the heights of slave children at ages three and four to predict their birth weights using the log linear relationship between mean heights at ages three and four and mean birth weights in poor modern populations. This procedure yielded an extremely low mean birth weight of 2,320 grams compared to current international standard of 3,380 grams.[31] However, despite these very poor early life health indicators, by the time that the slave children had stopped growing after age 21, the average man and woman was only 0.81 and 0.67 standard deviations below modern growth standards respectively. This is a massive gain in height relative to modern standards, much larger than any gain that has been measured for similarly deprived populations, including slave populations in the Caribbean analysed by Higman.[32]

Steckel's analysis is sound, but it is worth noting that by using percentiles to understand growth trajectories, he only described the relative catch-up growth of the population not the absolute catch-up, which is best measured using a Z-score. A population moving from the 1st percentile to the 2nd percentile of modern growth standards has made a much larger gain in absolute height than a population moving from the 49th percentile to the 50th percentile.[33] This difference is important because it leads Steckel to understate the catch-up growth of American slaves before the age of 10 (figures 2 and 3) and the catch-up growth of slaves in the Caribbean.[34]

Steckel argues that the American slave children began experiencing catch-up growth early in childhood, but the most sustained catch-up growth occurred after age 16.5 for boys and 13.5 for girls. Explaining the poor growth outcomes at younger ages and improving

---

children, adjusting thresholds for underweight, overweight and obese downward since the obesity epidemic had already started in the United States. The 2006 standard and 2007 reference are now widely accepted as a benchmark for children's growth and have been used to study populations around the world (WHO MGRS 'Assessment'; Butte *et al.*, 'Evaluation'; Wang *et al.*, 'Limitations'; Dercon and Singh, 'Nutrition').

[31] Steckel, 'Birth Weights', pp. 180-2; Villar *et al.*, 'International Standards', p. 864.

[32] Steckel, 'Peculiar Population', pp. 724-25; Prentice *et al.*, 'Critical Windows', pp. 914-916; Higman, *Slave Populations*, pp. 534-535.

[33] Wang *et al.*, 'Limitations', p. s180.

[34] Steckel, 'Peculiar Population', p. 728; Higman, *Slave Populations*, pp. 534-535.

health outcomes by the time slaves reached adulthood has been more difficult. Steckel argued that slave owners had different incentives to invest in children versus adults. Until children could join the productive workforce, after approximately age 10, slave owners fed children low cost and low nutritional quality grains, only providing meat and protein for children after they started working in the fields. Steckel also points out that slave mortality rates after age 10 were relatively low and stable, suggesting that the catch-up growth after age 10 would not be influenced by survival bias.[35]

Insert Figure 2 here

Coelho and McGuire have challenged Steckel's explanation of the catch-up growth, arguing that slow growth before the age of ten can be better explained by hookworm exposure than poor nutrition. They argue that because children were kept near slave quarters during the day and did not have good control over their bowels, they were at much higher risk of becoming infected with hookworm than adults who spent most of the time in the fields away from the unsanitary living quarters. Thus, when children began working around the age of ten, their exposure to and morbidity from hookworm decreased significantly, and they were able to attain high levels of catch-up growth. They estimated that hookworm could account for 31 per cent of the shortfall in children's heights.[36] Steckel challenged their claims in a response, arguing that they overestimated the effect of hookworm and that constant levels of childhood mortality among slaves aged seven and above suggest that improvements in the disease environment could not explain slave children's remarkable catch-up growth.[37]

Insert Figure 3 here

---

[35] Steckel, 'Peculiar Population', pp. 724-726, 735-36.
[36] Coelho and McGuire, 'Diets', pp. 239-241.
[37] Steckel, 'Diets', pp. 249-254.

<center>**III**</center>

Although these authors' arguments are interesting, there is quite an intriguing puzzle hidden in their extensive work. Steckel argues that slaves had the lowest average birth weights of any population ever studied, suggesting very poor early life health conditions.[38] Yet American slaves experienced dramatic catch-up growth. If prenatal conditions are extremely important for future growth and may even trump catch-up growth, then this is a real conundrum that needs to be solved, especially since modern populations suffering malnutrition *in utero* do not experience catch-up growth like the slaves did.[39]

However, an interpretation of slave children's growth in an adaptive framework would perhaps better explain the pattern of growth that Steckel observes (see figure 4 for a schematic view). I will first present the framework for American slaves before also applying it to slaves in the Caribbean as a robustness check. The argument turns around one key revision of Steckel's research on slave health, namely that slave birth weights were *not* extremely low. Thus, the argument would follow that adult slave women had relatively good nutrition and health. This ensured that prenatal conditions were fairly good for the growing fetus compared to other historical populations. Relatively good conditions *in utero* provoked a predictive adaptive response. Slave fetuses developed as if conditions in the postnatal period would closely mirror the conditions faced by their mothers in adulthood; they were programmed for a tall stature and a relatively high metabolism.

However, after the children were born, they experienced a brutal mismatch between the prenatal and postnatal environment: conditions were far worse for the children in the

---

[38] Steckel, 'Birth Weights'.
[39] Proos and Gustafsson, 'Early Puberty'.

early postnatal period than the prenatal period. The combination of a high metabolism set in the prenatal period and poorer nutritional and disease conditions in the postnatal period took a striking toll on the children's health and prompted an immediate adaptive response in the children, slowing their growth rate relative to children in modern populations. This is corroborated by evidence that the children were severely stunted when the first reliable estimates of their height are available at age three and four.[40] The children first started experiencing catch-up after age 3.5, but persistently poor conditions likely also triggered the immediate adaptive response in the children to delay the pubertal growth spurt since their level of nutrition was still quite low in early adolescence. Thus, the second bout of catch-up growth was possibly sparked by the children's entry into the labour force, which provided them with a more ample and balanced diet and reduced their hookworm morbidity. These two positive shocks forced the children back onto the healthier growth trajectory that was set during the prenatal period, allowing them to experience catch-up growth. The immediate adaptive response to delay the pubertal growth spurt also meant that the growth continued for a much longer period than would occur in modern populations.

This new interpretation is built on four key assertions about slavery in antebellum America that have to be carefully justified: first, slave women had to be healthy and well fed relative to women in other historical populations; second, conditions for slave children had to be very bad in infancy and early childhood and improve thereafter; third, slave children's birth weights could not have been as low as Steckel estimated; and finally, catch-up growth could not have occurred on the scale observed if the children experienced poor conditions in the prenatal period. I will deal with each of these in turn; however, it is important to note that slavery was a very heterogeneous institution with different practices based on the size of the plantation and the primary crop. This makes it difficult to precisely determine factors

---

[40] Steckel, 'Birth Weights', p. 178.

influencing the pace of catch-up growth across childhood and adolescence, especially acceleration at a given age, but it is possible to highlight changes in slave nutrition and disease exposure that could have sparked such tremendous catch-up growth.

Insert Figure 4 here

The first assertion that must be proven in order for the adaptive framework explanation of slaves' growth to hold true is that slave women had to have been healthy relative to other historical populations. There is considerable debate about whether adult slaves received adequate nutrition for the amount of work that they were required to perform. In the controversial *Time on the Cross*, Fogel and Engerman argued that slaves were given adequate nutrition to carry out their required tasks.[41] However, Sutch disputed Fogel and Engerman's findings, arguing that although slaves were provided a diet with enough energy to carry out their required tasks, their diets lacked variety and key nutrients.[42] Kiple and Kiple also dispute that adult slaves had adequate nutrition arguing that their diets were deficient in iron and calcium.[43] However, it is unclear how much worse this would have been relative to other historical populations, who also had inadequate diets in the early and mid-nineteenth century. Gibbs et al. agree, in a study of South Carolina, Georgia and Florida, concluding that 'the diet of slaves in this ecozone appears to have met the nutrient needs of the population in a manner that permitted continued high work outputs as well as substantial population growth'.[44]

[41] Fogel and Engerman, *Time*.
[42] Sutch, 'Treatment', pp. 359-60, 382-86.
[43] Kiple and Kiple, 'Slave', p. 287.
[44] Gibbs *et al.*, 'Nutrition', p. 251.

Despite the contrary evidence, in a recent paper Jasienska concludes that 'slavery was associated with poor nutritional status in utero, childhood, and adulthood'.[45] This is certainly true when comparing slaves to modern populations, but this statement does not conform to the anthropometric evidence that is available for slaves relative to other historical populations. At 158.9 cm tall (in the coastwise manifest), American female slaves were substantially taller than working-class women in other contemporary populations. Female convicts transported from England and Ireland to Australia were significantly shorter—156.6 cm for rural English women, 154.3 cm for urban English women, 155.7 cm for rural Irish women and 155.3 cm for urban Irish women.[46] Even women imprisoned in the nineteenth-century Netherlands were shorter at 156.7 cm than American slave women, and the Dutch are now the tallest population in the world.[47] In addition, the size of catch-up growth that slaves of both genders experienced is evidence that slaves were given more than enough nutrients to grow despite their high work requirements in adolescence.[48] Finally, Steckel shows that while there were substantial differences in mortality rates of slaves and other Americans in childhood, these had largely disappeared by age 20-24.[49]

It is thus credible that aside from their heavy labour, adult female slaves experienced fairly good conditions relative to other historical populations. This does not mean that slave women were healthy or fit relative to modern populations. It only means that the conditions that pregnant slave woman and their developing children experienced *in utero* were superior to the conditions that slave children faced in the first few years of life.

The second key assertion is that the environmental and nutritional conditions that slave children faced in infancy had to be exceptionally bad and there had to be improvement

---

[45] She argues that the persistent (and econometrically unexplainable) gap in birth weights between European Americans and African Americans descended from slaves in the US may be a residual effect of the terrible conditions of slavery (Jasienska, *Fragile*, p. 126, quote; Jasienska, 'Low').
[46] Johnson and Nicholas, 'Male', p. 211, 219; Nicholas and Oxley, 'Living Standards', p. 733.
[47] de Beer, 'Physical Stature', p. 64.
[48] Steckel, 'Peculiar Population'.
[49] Steckel, 'Dreadful Childhood'.

over childhood and adolescence. There is substantial evidence to support this claim, though the evidence on slave children's health and nutrition is sparse and fragmented at best. While mothers tried to breastfeed their children for at least a year, this could prove difficult depending on owners' labour requirements. Agricultural journals and plantation rule books generally suggest that slave mothers were given one month out of work following a birth, allowing them to recover and practise exclusive breastfeeding, but after this month and even during the first month in times of peak labour demand, it became more difficult to maintain an exclusive breast milk diet for their babies. On smaller plantations, infants might be brought with their mothers into the fields where they could be nursed regularly. However, on larger plantations using gang labour or where the slave owners wished to extract more productive labour, infants would be kept either in the slave quarters or in a nursery with mothers returning two to three times during the day to breastfeed their children.[50] The rigid schedules and limited feeding, especially during times of peak labour demand, forced slaves to begin supplementing infants' diets from an early age, depriving slave children of key nutrients, protein and anti-bodies from their mother's milk and also making them more susceptible to diseases from contaminated food.[51] Slaves supplemented breast milk with potlicker (broth from cooking beans), mush and skimmed milk, or clabber (soured thick milk with a yoghurt-like consistency).[52] Slave children were fully weaned probably around one year, though mothers were given less time to breastfeed after 8 months.[53]

After weaning slave children were given a very poor diet of mush, corn, hominy and fat, lacking in protein and micronutrients necessary for growth. This poor diet left slave

---

[50] Schwartz, *Born*, pp. 56/7, 60-64.
[51] Kiple and Kiple, 'Slave', p. 288.
[52] Schwartz, *Born*, pp. 67/8.
[53] Schwartz, *Born*, pp. 82/3; Klein and Engerman, 'Fertility'; Kiple and Kiple, 'Slave'.

children especially prone to protein-calorie malnutrition.[54] Slave infants also suffered from acute deficiencies in calcium, magnesium, and iron, which greatly increased infant mortality.[55] This poor diet was even less plentiful in the first two years of a child's life since slave holders often denied weekly food rations to children under the age of two, forcing parents to feed young children from their own rations.[56] This deficit in rations is important since the FAO guidelines suggest that children aged 1-2 require around 900 kcal per day, perhaps around a quarter of a slave's daily allotment.[57] If slaves were not able to sacrifice this level of calories or if they were not aware that toddlers needed so much energy, then their children could have suffered a lot in these early years.

Finally, slave children had increased exposure to disease because of open defecation and unsanitary and cramped living quarters. Thus, slave children likely experienced high levels of morbidity from malaria, hookworm, diarrhoea, and respiratory diseases despite the fact that they may have had some level of immunity to hookworm and malaria.[58] Pneumonia, hookworm and malaria were particularly important as the eradication of these diseases in the South in the early twentieth century led to significant increases in human capital.[59]

Despite these very poor conditions in infancy and very early childhood, there is substantial evidence to suggest that conditions improved for children in later childhood and adolescence. Before children began working in the fields around age 10, there were likely three main factors driving catch-up growth. First, there may have been some selection of weak, shorter children in early childhood since mortality rates were quite high during this period. Second, slave children's diets improved even before they went to the fields as full

---

[54] Interestingly, Forrester *et al.*, 'Prenatal factors' found that children with poor fetal nutrition were less likely to develop marasmus and kwashiorkor in childhood, bolstering evidence presented below that Steckel's birth weights were likely too low.

[55] Kiple and Kiple, 'Slave', pp. 285-286, 289, 296-297.

[56] Schwartz, *Born*, p. 68.

[57] FAO, 'Human', pp. 26/7.

[58] Savitt, *Medicine*, pp. 57-73; Kiple and Kiple, 'Slave', pp. 298/9; Coelho and McGuire, 'Biology'.

[59] Bleakley, 'Disease'; Bleakley, 'Economics'; Bhalotra and Venkataramani, 'Captain'.

labourers. Slave children began to receive their own food rations after age two, but more importantly, they also helped the household supplement its income and diet by fishing, trapping or hunting small game, helping out in the garden and making cloth. These tasks were difficult for the parents to carry out regularly when working in the fields and caring for younger children.[60] Finally, modern evidence suggests that children may develop progressive immunities that prevent them from getting sick as often. For instance, Prentice *et al.* attributed catch-up growth between the ages of 3 and 7 in Gambian children to a reduction in growth-impairing infections as 'the normal postnatal maturation of the children's immune systems' proceeded and the children developed 'a broad repertoire of adaptive responses against previously encountered pathogens'.[61]

After the children began labouring in the fields, their condition improved further. According to agricultural journals and guides to plantation management, children working in the fields received substantially larger rations with more meat than children not working in the fields. They also received better clothes more frequently and new shoes and blankets.[62] Schwartz describes these perks as an inducement to get children to begin working in the fields, but they could fit Steckel's model of slave owner investment as well. An increase in the quantity and quality of food would have dramatically improved the health of slave children. Receiving shoes would have also significantly reduced their exposure to hookworm, which was often contracted through bare feet.[63] Clearly, environmental and nutritional conditions for slaves in infancy and early childhood were very bad and could easily have been worse than the conditions they faced in the prenatal period, but there was a marked improvement across childhood and adolescence that sparked catch-up growth.

---

[60] Schwartz, *Born*, pp. 132-34.
[61] Prentice *et al.*, 'Critical Windows', p. 914.
[62] Schwartz, *Born*, pp. 138-43; Steckel, 'Peculiar Population', pp. 735/6.
[63] Coelho and McGuire, 'Diets', p. 240.

Third, it seems likely that Steckel significantly underestimated slaves' birth weights. As mentioned above, Steckel estimated birth weights by assuming that the log linear relationship between birth length (or height) and birth weight in poor modern populations would be the same as in historical populations.[64] All in all, this is not a bad assumption given the paucity of historical data. However, because it is very difficult to measure height for children who cannot walk, Steckel used the average heights of three and four-year-olds relative to modern standards to predict the slaves' birth weights. This assumption is slightly more problematic because it requires the slave children to have followed the same growth trajectory from birth until the point of measurement as the reference populations used to predict the birth weights. Growth faltering, or stunting in early life, is common in impoverished populations, and likely occurred in Steckel's reference populations.[65] However, the evidence presented above suggests that infancy and early childhood were periods of extremely poor nutrition and health for slave children, and likely poorer than Steckel's reference populations. Thus, it seems plausible that slave children would have become severely stunted by the time they reached the age of three or four, especially since growth velocities are highest before the age of three.[66]

In addition, the birth weights that Steckel predicted were extremely low. At 2,320 grams, they are a full 1,060 grams below the current international standard of 3,380 grams.[67] The only populations in the world with mean birth weights of a similar level are tribal populations in New Guinea. In the 1960s, these children had birth weights between 2,400 and 2,600 grams, experienced slow growth throughout their growing years, and attained extremely short final adult statures.[68] The Lumi of New Guinea, the population with the lowest mean birth weight that Steckel includes in his regression to predict slaves' birth

[64] Steckel, 'Birth Weights', p. 181.
[65] Victora et al., 'Worldwide'.
[66] Boersma and Wit, 'Catch-up Growth, p. 647.
[67] Villar et al., 'International Standards', p. 864.
[68] Malcom, Growth; Ulijaszek, 'Evidence'; Tracer et al., 'Two'.

weights, had a mean birth weight around 2,400 grams, but at maturity women were only 147 cm tall, nearly 12 cm shorter than female slaves in the US South.[69] Aside from these populations, which may differ genetically from other populations, the lowest observed mean birth weights are 2,700 to 2,800 grams. Indeed, the African countries from which most US slaves were drawn had mean birth weights much higher in the 1970s: between 2,880 and 3,117 grams in Nigeria and 3,163 grams in Zaire. These African birth weight figures compare well with the mean birth weights of African American children born in the early twentieth century at Johns Hopkins Hospital in Baltimore, 3,183 grams.[70] Thus, while it is impossible to know the birth weights of slaves, this evidence suggests that Steckel's predicted birth weights are implausibly low and that slave birth weights were likely substantially higher.

Analysing the potential causes of low slave birth weights also leads to suspicion of Steckel's figures. If we consider the factors contributing to low birth weights that Steckel mentions, the lower average height of slave populations, presence of sickle cell anemia and maternal exposure to malaria could explain 380 grams of the shortfall between Steckel's predicted birth weights and international standards, but this still leaves a 680 gram deficit.[71] Steckel attributes this 680 grams to the hard labour that slave women were forced to carry out during their pregnancies and their upright posture during work, which can reduce blood flow to the placenta.

There is some evidence that slaves' work influenced the mortality outcomes of their children and thus might have influenced the slave children's early life health and potentially birth weights. Steckel found that neonatal mortality was higher during periods of high labour demand and Campbell found that women given more days off work in the first and second trimesters had lower rates of infant mortality among their children.[72] These studies are

---

[69] Steckel, 'Birth Weights', p. 181; Malcolm, *Growth*, p. 77.
[70] Costa, 'Race', p. 1065.
[71] Steckel, 'Birth Weights', pp. 185-88.
[72] Steckel, 'Dreadful Childhood'; Campbell, 'Work'.

interesting, but their connections between maternal work and fetal health conditions are somewhat tenuous for several reasons. First, it seems likely that on average pregnant women were given less physically demanding tasks, especially once the pregnancy could be verified. Agricultural journals and a general attitude of paternalism among slaveholders suggest that this was a strong norm. This does not mean that women were never mistreated or forced to perform hard labour during the final months of pregnancy. Cotton picking rates suggest that women were picking only around 20 per cent less in the third trimester than they would in a normal year. However, cotton picking was not considered particularly arduous, and the presence of 'trash gangs' on large plantations and slaveholders' desire to increase their slave population likely meant that overall pregnant women were relieved of the heaviest tasks.[73]

Second, more recent historical and medical research has shown that early neonatal mortality (deaths in the first seven days) is a better predictor of fetal health than infant mortality or neonatal mortality (deaths in the first 28 days), which are often driven by infectious disease exposure rather than congenital weakness.[74] If we look at early neonatal deaths in Steckel's sample, they are only higher during the planting and preparation season on cotton plantations from February to April.[75]

Third, a large percentage of stillbirths and early neonatal deaths historically were caused by complications during childbirth that had little to do with fetal health and thus predictive adaptive responses.[76] In fact, early neonatal deaths from intrapartum complications may help explain why early neonatal deaths were higher in February to April than during the rest of the year. Modern studies of the effects of maternal exercise on birth weights and pregnancy outcomes have generally found that maternal exercise does not influence either if the mother has adequate nutrition. However, heavy exercise can exacerbate intrapartum

---

[73] Schwartz, *Born*, pp. 25-27; Steckel, 'Dreadful Childhood', p. 440.
[74] Woods, *Death*, pp. 95-98; WHO, *Neonatal*.
[75] Steckel, 'Dreadful Childhood', p. 443.
[76] Woods *et al.*, 'Two Hundred'; Løkke, 'Antibiotic Transformation'.

complications such as placenta praevia and placental abruption.[77] Thus, higher levels of early neonatal mortality during times of peak demand for hard physical labour may reflect greater rates of intrapartum complications.

Finally, Campbell's finding that work release in the first two trimesters (and especially the first trimester) was most important in reducing the risk of infant death is perplexing.[78] Given that the evidence on women's exercise and infant mortality and birth weight is mixed at best and effects should be stronger later in pregnancy, it seems that the link Campbell finds may be indirect rather than direct. Perhaps the number of days off in the first trimester is a proxy for the woman's bargaining power with the overseers and slaveholder more generally. Women who were able to negotiate more time off early in pregnancy may have been able to secure better food and less strenuous work at the end of their pregnancy and after birth, which Campbell could not observe directly.[79] In conclusion, it seems likely that maternal work effort cannot explain Steckel's extremely low average slave birth weight, bolstering my claim that birth weights and early life health conditions were better than he thought.

Finally, the magnitude of the catch-up growth that the slave children achieved is unprecedented and would seem implausible if slave children were exposed to such terrible conditions *in utero*. The slave boys in Steckel's sample experienced a 2.4 standard deviation increase and the girls a 2.2 standard deviation increase in height-for-age relative to modern standards. Modern studies have mostly found catch-up growth of less than one standard deviation relative to the modern mean.[80] Thus, it is clear that the catch-up growth of American slaves was truly remarkable, especially considering that the intervention sparking

---

[77] Kramer, 'Determinants'; Artal and O'Toole, 'Guidelines'; Tafari, 'Effects'.
[78] Campbell, 'Work'.
[79] Schwartz, *Born*, pp. 24-27.
[80] Prentice *et al.*, 'Critcal Windows'.

catch-up growth occurred in the first half of the nineteenth century when even the best nutritional and environmental conditions were far below modern levels.

The slaves' catch-up growth is even more puzzling because there is substantial evidence that catch-up growth is different for children exposed to a poor environment *in utero*. For example, Proos found that Indian children with birth weights under 2,000 grams adopted in Sweden had significantly lower catch-up growth than children adopted with higher birth weights. In addition, although nearly all of the adopted Indian girls experienced rapid type A catch-up growth after their move to Sweden, they also experienced early onset of the pubertal growth spurt and menarche and did not reach final heights significantly taller than their counterparts in India.[81] This pattern has been observed with malnourished children from other countries adopted in developed countries as well.[82] Thus, it seems that for these adopted children poor conditions in the prenatal period triggered a predictive adaptive response that lowered metabolism and height potential and accelerated maturation. Further poor conditions in early childhood led to additional stunting as many of the children were malnourished and suffering from chronic diseases before being adopted. However, when the children were adopted and given the best nutrition and health care available, they experienced rapid type A catch-up growth that restored them to their prenatally determined growth trajectory. Thus, they had an earlier age at menarche than Swedish girls who were not deprived *in utero* and their Indian counterparts, who had experienced an immediate adaptive response in early adolescence to delay the pubertal growth spurt. Although the adopted children experienced type A catch-up growth, they were not able to continue to grow at relatively high velocities into their early twenties.

The direct implications of these studies for slaves' growth are slightly difficult to understand because the effectiveness of the intervention for the Indian girls was substantially

---

[81] Proos, 'Growth'.
[82] Proos and Gustafsson, 'Early Puberty'.

greater than the effectiveness of the intervention for slaves. Gluckman and Hanson also emphasize that the postnatal immediate adaptive response to delay maturation until conditions improve is probably more powerful than the prenatal predictive adaptive response to poor nutrition *in utero* to advance maturation.[83] However, the evidence presented above suggests that catch-up growth may be limited for children who have experienced poor prenatal conditions and have a low birth weight, especially if they experience an intervention that would spark type A catch-up growth.[84] Thus, the low average birth weight of American slaves seems inconsistent with their remarkably high level of catch-up growth. Their unique pattern of growth would make more sense if slave children developed and set their metabolism and growth trajectory in a relatively good uterine environment, then faced severe deprivation in infancy before experiencing catch-up growth as conditions improved in late childhood and adolescence.

IV

The adaptive framework seems plausible in explaining the growth pattern of American slaves, but explaining why Caribbean slaves would also experience substantial catch-up growth is more difficult. In terms of maternal health, slave women in the Caribbean were substantially shorter than their American slave counterparts and shorter than or equal to women in England and Ireland.[85] Women born in St. Lucia and Trinidad were 153.3 cm and 155.6 cm tall respectively, and slave women born in Africa were 1-2 cm shorter.[86] Caribbean slave women were also forced to work long hours and were given rum as part of their

---

[83] Gluckman and Hanson, 'Evolution', p. 9.
[84] Proos and Gustafsson, 'Early Puberty'.
[85] Higman's data is drawn from censuses of slaves taken in Trinidad in 1813 and St. Lucia in 1815. These censuses included all slaves and were verified by the colonial administrators in charge of the registration. Thus, they do not feature the potential for selection bias that the coastwise manifest sample does (Higman, *Slave*, pp. 6-11, 22/3).
[86] Higman, *Slave*, p. 281; Steckel, 'Growth Depression'.

rations.[87] Infant mortality rates were very high in St. Lucia and Trinidad, 583 and 479 deaths per 1,000 according to model life tables, suggesting poor health conditions *in utero* as well as a bad disease environment in infancy.[88] These are much higher even than Steckel's upper bound estimate of 350 deaths per 1,000 for American slaves based on his predicted birth weights.[89] This evidence suggests that conditions *in utero* in the Caribbean were poorer than in the United States. However, in terms of child health, slave mothers tended to wean their children at later stages in the Caribbean than in the United States, which may have given the Caribbean slave children an advantage if they survived the first month of life.[90] These practices may have contributed to the lower levels of childhood and adolescent mortality in the Caribbean when compared with the antebellum American South.[91] Slaves in the Caribbean and in the US South were given similar similar types of food with nutrition improving after slaves entered the workforce in both places.[92] Thus, it seems likely that prenatal conditions in the Caribbean were worse than in the US South, but conditions in childhood were perhaps slightly better.

Placing Caribbean slave children's growth in the adaptive framework (figure 4), slaves in the Caribbean achieved substantial catch-up growth because their material conditions did improve across their growing years, but their potential for catch-up was limited by the prenatal predictive adaptive response, which set their growth trajectory and metabolism at a lower level. Although conditions were still probably worse in the postnatal period than the prenatal period, the mismatch was smaller, which meant that Caribbean slave children did not fall behind modern standards as quickly as American slave children. Their

---

[87] Steckel shows that alcohol consumption levels were lower for slaves in the US (Steckel, 'Peculiar Population', p. 729; Higman 1984, pp. 205, 211).

[88] Higman, *Slave*, p. 319; Floud *et al.*, *Changing Body*, p. 16; Wrigley, 'British Population', p. 73.

[89] Steckel, 'Peculiar Population', p. 733.

[90] Klein and Engerman, 'Fertility Differentials'; Higman, *Slave*, pp. 353/4.

[91] Higman, *Slave*, pp. 654-57; Meredith John, 'Plantation', pp. 170/1; Steckel, 'Slave Mortality', p. 92.

[92] Kiple and Kiple, 'Deficiency Diseases', pp. 200-3; but see Higman, *Slave*, pp. 204-205 for a more sceptical opinion.

metabolism better matched their postnatal experience. Clearly, an adaptive framework for growth can be instructive when trying to understand the growth trajectories of different populations.

<p style="text-align:center">**V**</p>

With the lessons of the previous case study in mind, we can now explore the implications of an adaptive framework of growth to changes in the growth pattern of human populations over the past 150 years. Unfortunately, this discussion will have to be rather speculative because a lot more primary work remains to be done on how the growth trajectories of children have changed over time. However, a perusal of the secondary literature does allow for the development of four preliminary stylized facts that could guide a more systematic and robust analysis: 1) Early life health conditions have been relatively stable over the past 150 years in the Western World; 2) There is considerable heterogeneity in early life health around the world today with some developing countries experiencing very low average birth weights; 3) Children in the past experienced the pubertal growth spurt at later ages than modern populations; and 4) There has been a secular decrease in the age at menarche for girls in the West over the past 100 years.

The first stylized fact is that at least from the mid-nineteenth century onward, it appears that early life health conditions in the West were fairly good. Where birth weight evidence is available, mostly for working class populations, it seems that in the second half of the nineteenth and early twentieth centuries, average birth weights in North America had already reached their modern levels (table 1). Birth weights in Europe improved over the twentieth century somewhat, but they were already at a relatively high level. If we use a low birth weight (LBW) percentage of 10 per cent to distinguish developed from developing countries as Ward suggests, then clearly Edinburgh, Vienna and African Americans in

Boston showed room for improvement in the late nineteenth and early twentieth centuries.[93] Of course looking only at averages may hide considerable change in the underlying birth weight distributions, and birth weight is at best an imperfect proxy for fetal health conditions.[94] However, the stability of birth weights over time is a striking result that needs to be analysed in more detail in the future.

Insert Table 1 here

Second, there is greater heterogeneity in early life health (proxied by birth weight) around the world today than there was within Western countries over time. In the 1980s, birth weights in India and Pakistan were the lowest with means around 2,750 grams and a LBW rate of 30 per cent (table A1 in the online appendix). Some developing countries did perform better, though, with relatively high birth weights in China (3,250 grams) and Iraq (3,540 grams).[95] However, the fact that the historical samples that Ward measured were in the upper part of the 1980s global birth weight distribution suggests that children in North America and Europe were never exposed to the worst intra-uterine conditions now present in the developing world.

The third stylized fact is that children in the past had later pubertal growth spurts than healthy children in the present. Steckel has calculated the precise age at the peak of the pubertal growth spurt for a number of historical populations and found that all of the populations had much later pubertal growth spurts than modern populations.[96] Thus, there has been a transition over the past 100 years where the pubertal growth spurt has occurred earlier in children of both sexes.

---

[93] Ward, *Birth Weight*.
[94] Hanson *et al.*, 'Optimal Fetal Growth'.
[95] Kramer, 'Determinants', p. 665.
[96] Steckel, 'Growth Depression'.

Fourth and finally, there has been a clear secular decline in the age at menarche for girls over the last century in Western countries. Before the turn of the twentieth century, Norwegian, German, Finnish and Swedish girls experienced menarche between the ages of 15.5 and 17 whereas today the average age at menarche has fallen below 13 years old.[97] This is of course related to the decline in the age of the pubertal growth spurt, but may highlight that the link between growth and maturation (especially in puberty) is stronger in girls than in boys.[98]

Keeping these four stylized facts in mind, we can now attempt to interpret the differing growth trajectories of historical populations and draw some wider lessons about historical growth patterns. The suggestion that birth weights in the Western World were already quite high in the second half of the nineteenth century indicates that the negative consequences of poor early life health were not as strong in these countries as they are in some developing countries today. Thus, the predictive adaptive response was not a strong limiting factor on children's growth in North America and Europe 150 years ago as it might be in India today. This may also suggest that negative consequences of these predictive adaptive responses, such as higher risks of heart disease and diabetes in later life, may be more prominent in developing countries in the present and future than they were in developed countries in the past. Reports of age-standardized prevalence of diabetes around the world support this suspicion.[99] Good prenatal conditions in the North America and Europe also allowed these populations to experience greater catch-up growth if conditions improved. Despite relatively good early life health conditions, the delay in the timing of the pubertal growth spurt and later age at menarche in Western populations in the past suggests that these children must have been making immediate adaptive responses to delay maturation in response to poor nutrition or high levels of morbidity in the postnatal period. Thus, the

---

[97] Tanner, *Growth*, pp. 152-55; Brundtland and Walløe, 'Menarcheal Age'.
[98] Proos and Gustafsson, 'Early Puberty'.
[99] Diamond, 'Double Puzzle'.

secular increase in adult height over the past 150 years was likely caused by a reduction in postnatal stunting over time.[100]

These conclusions naturally lead to comparisons with the developing world today, but we must be careful before applying the policy implications from research conducted on Western populations to the rest of the world. Many developing countries face a double burden of prenatal and postnatal malnutrition and ill health, which may make the population's response to policy interventions somewhat different than what was experienced historically. One clear example of this is the persistence of stunting rates, the percentage of five-year-olds with heights two standard deviations below the modern standard, over the past twenty years among Indian children under the age of five despite substantial economic growth during that period.[101] Frustration over these rates has led some scholars (and Indian policy makers) to argue that Indian children are genetically different than other populations despite substantial evidence that this is not true.[102] Instead, it seems that poor conditions *in utero* are part of the mix of factors placing limits on improvements in child health (proxied by growth) over time. Poor early life health conditions in India are at least partly caused by gender inequalities in health, which were not as prominent in Europe and America in the nineteenth century.[103]

Hopefully, it is clear that analysing growth patterns in an adaptive framework can provide some useful predictions about how populations will respond to initial conditions *in utero* and nutritional and disease insults in the postnatal period. These predictions are not necessarily novel or surprising in and of themselves, but they are strengthened when placed in a unified adaptive theory based in evolutionary biology.

---

[100] Hatton, 'Europeans' finds evidence of this.
[101] World Bank World Development Indicators, http://data.worldbank.org/.
[102] Panagariya, 'India'; The WHO Multicentre Growth Reference Study (MGRS) did not find significant differences between the healthy Indian children and healthy children in the other samples (WHO MGRS, 'Assessment'). See also Eveleth and Tanner, *Worldwide*.
[103] Osmani and Sen, 'Hidden Penalties'; Horrell and Oxley, 'Bargaining'.

VI

This paper has introduced a new, adaptive framework for interpreting the pattern of children's growth in populations past and present. The adaptive framework shows how exposure to good or bad conditions at various points in the growing years can influence a child's growth pattern. Poor conditions *in utero* can trigger a predictive adaptive response that decreases a child's metabolism, sets him/her on a slower growth trajectory, and advances his/her physical and sexual maturation. There is also substantial evidence that poor conditions *in utero* limit a child's potential for catch-up growth in later life. Poor conditions in the postnatal period can spark immediate adaptive responses to delay maturation and growth until conditions improve. When these conditions do improve, catch-up growth serves as another adaptive response that attempts to move the child back to its prenatally programmed growth trajectory. All of these responses are adaptive in the sense that they increase the probability that a child will survive to reproductive age and reproduce. However, they are not without their costs. Poor nutrition *in utero* has been linked to heart disease and diabetes in later life and shorter children and adults have higher mortality risks at all ages.

I then used the framework to suggest a reinterpretation of the growth of slaves in the United States and Caribbean. These slaves appear to have experienced a mismatch between the prenatal and postnatal environment where the postnatal environment was substantially worse than the prenatal environment. This mismatch was likely greatest for US slaves because maternal health and early life health conditions were relatively good there. The mismatch caused slave infants to become severely stunted in their first few years of life because their prenatally programmed metabolism was set too high, but as conditions improved, they regained their former growth trajectory and experienced catch-up growth. Caribbean slaves did not experience the same level of catch-up growth because they had

poorer early life health, which limited their ability to respond to improving conditions. They were likely restricted by their metabolism and growth trajectory set in the prenatal period. These conclusions are tentative since the evidence on slave health is not as complete as one would hope, but this example highlights that placing children's growth in an adaptive framework can be a helpful way of categorising and explaining the growth patterns of children in the past.

Finally, the paper introduced four stylized facts of children's historical growth experience in the past 150 years. These stylized facts and the adaptive framework help to clarify some important issues that should be addressed in future research. First, this paper shows how important it is for each generation of health historians to read the latest biological literature and approach current and past debates and data to see what the newest thinking can contribute to our understanding of the past. This literature review is time consuming, but it yields interesting rewards including collaborations with medical doctors and human biologists and makes our historical work of greater relevance to the medical field. Second, the adaptive framework shows that the traditional focus on historical changes in adult height may be obscuring a clear understanding of health and child growth in the past. Growth is an enormously complex process, and by studying this process directly, the growth pattern of children, we can learn more about how child health has changed over time. This will involve new data collection efforts focused on measuring children's growth, confirming or falsifying the four stylized facts mentioned above and explaining how and why the growth pattern has changed. For instance, have early life health conditions remained constant over time? What conditions led to a shift toward an earlier maturation and pubertal growth spurt? When did children in the West achieve the modern growth pattern, and so on?

Third, the adaptive framework suggests that shifts in the growth pattern may have an influence on old age morbidity and mortality from cardiovascular disease and diabetes.

Future research could study these links more directly, especially given that age-specific cohort mortality from non-infectious diseases has fallen since the late nineteenth century.[104] Finally, understanding growth in an adaptive framework allows economic historians to link their findings more directly with health policy issues in the developing world today. Stunting rates at age five are one of the leading indicators for malnutrition and reducing stunting is in fact manufacturing a shift in the growth pattern of children. Historical comparisons may reveal key similarities and differences in these processes, yielding lessons for development policy. Clearly, an adaptive framework for child growth provides anthropometric historians with new questions and ideas to explore.

---

[104] Arora, 'Understanding Aging'.

References

Almond, Douglas and Janet Currie, 'Killing Me Softly: The Fetal Origins Hypothesis', *Journal of Economic Perspectives*, 25, no. 3 (2011), pp. 153-72.

Arora, Suchit, 'Understanding Aging during the Epidemiological Transition', *Research in Economic History*, 29 (2013), pp. 1-69.

Artal, R. and M. O'Toole, 'Guidelines of the American College of Obstetricians and Gynecologists for Exercise during Pregnancy and the Postpartum Period', *British Journal of Sports Medicine*, 37, no. 1 (2003), pp. 6-12.

Barker, David J. 'The Fetal and Infant Origins of Adult Disease', *British Medical Journal*, 301, no. 6761 (1990), p. 1111.

Barker, David J. P., 'Maternal Nutrition, Fetal Nutrition, and Disease in Later Life', *Nutrition*, 13, no. 9 (1997), pp. 807-13.

de Beer, Hans, 'Physical stature and biological living standards of girls and young women in the Netherlands, born between 1815 and 1865', *The History of the Family*, 15, no. 1 (2010), pp. 60-75.

Bhalotra, Sonia and Atheendar Venkataramani, 'The Captain of the Men of Death and His Shadow: Long-Run Impacts of Early Life Pneumonia Exposure', *SSRN Working Paper* (2012).

Bleakley, Hoyt, 'Disease and Development: Evidence from Hookworm Eradication in the American South', *Quarterly Journal of Economics*, 122, no. 1 (2007), pp. 73-117.

Bleakley, Hoyt, 'The Economics of Malaria: Economic Effects of Childhood Exposure to Tropical Disease', *American Economic Review: Papers and Proceedings*, 99, no. 2 (2009), pp. 218-23.

Bodenhorn, Howard, Timothy W. Guinnane, and Thomas A. Mroz, 'Problems of Sample-selection Bias in the Historical Heights Literature: A Theoretical and Econometric Analysis', Economic Growth Center Discussion Paper, Yale University (2013).

Boersma, Bart, and Jan Maarten Wit, 'Catch-up Growth', *Endocrine Reviews*, 18, no. 5 (1997), pp. 646-661.

Bogin, Barry, Maria Inês Varela Silva, and Luis Rios, 'Life history trade-offs in human growth: Adaptation or pathology?', *American Journal of Human Biology*, 19, no. 5 (2007), pp. 631–642.

Brundtland, G H, K Liestøl, and L Walløe, 'Height, Weight and Menarcheal Age of Oslo Schoolchildren During the Last 60 Years', *Annals of Human Biology*, 7 (1980), pp. 307-22.

Butte, Nancy F., Cutberto Garza and Mercedes de Onis, 'Evaluation of the Feasibility of International Growth Standards for School-Aged Children and Adolescents', *Food and Nutrition Bulletin*, 27, no. 4 (2006), pp. s169-s174.

Calomiris, Charles W. and Jonathan B. Pritchett, 'Preserving Slave Families for Profit: Traders' Incentives and Pricing in the New Orleans Slave Market', *The Journal of Economic History*, 69, no. 4 (2009), pp. 986-1011.

Campbell, John, 'Work, Pregnancy, and Infant Mortality among Southern Slaves', *Journal of Interdisciplinary History*, 14, no. 4 (1984), pp. 793-812.

Caulfield, Laura E., *et al.*, 'Undernutrition as an Underlying Cause of Child Deaths Associated with Diarrhea, Pneumonia, Malaria, and Measles', *American Journal of Clinical Nutrition*, 80 (2004), 197.

Coelho, Philip R. P. and Robert A. McGuire, 'Biology, Diseases, and Economics: An Epidemiological History of Slavery in the American South', *Journal of Bioeconomics*, 1 (1999), pp. 151-90.

Coelho, Philip R. P. and Robert A. McGuire, 'Diets Versus Diseases: The Anthropometrics of Slave Children', *Journal of Economic History*, 60, no. 1 (2000), pp. 232-46.

Costa, Dora L., 'Unequal at Birth: A long-term Comparison of Income and Birth Weight', *The Journal of Economic History*, 58, no. 4 (1998), pp. 987-1009.

Costa, Dora L., 'Race and Pregnancy Outcomes in the Twentieth Century: A Long-Term Comparison', *The Journal of Economic History*, 64, no. 4 (2004), pp. 1056-1086.

Dauncey, M. J. and J. M. Pell, 'Genetic Regulation of Growth-Promoting Factors by Nutrition', in Stanley J. Ulijaszek, Francis E. Johnston, and Michael A. Preece (eds.), *The Cambridge Encyclopedia of Human Growth and Development* (Cambridge, 1998), pp. 142-44.

Dercon, Stefan and Abhijeet Singh, 'From Nutrition to Aspirations and Self-Efficacy: Gender Bias over Time among Children in Four Countries', *World Development*, 45 (2013), pp. 31-50.

Diamond, Jared M., 'The Double Puzzle of Diabetes', *Nature*, 423 (2003), pp. 599-602.

Dolinoy, Dana C. and Randy L. Jirtle, 'Environmental Epigenomics in Human Health and Disease, *Environmental and Molecular Mutagenesis*, 49 (2008), pp. 4-8.

Eveleth, Phyllis B. and J. M. Tanner, *Worldwide Variation in Human Growth* (Cambridge, 1992).

Feinberg, Andrew P., 'Phenotypic Plasticity and the Epigenetics of Human Disease', *Nature*, 447 (2007), pp. 433-40.

Floud, Roderick, Robert W. Fogel, Bernard Harris and Sok Chul Hong, *The Changing Body: Health, Nutrition, and Human Development in the Western World since 1700* (Cambridge, 2011).

Fogel, Robert William and Stanley L. Engerman, *Time on the Cross: The Economics of American Negro Slavery* (Boston, 1974).

Forrester, Terrence E, Asha V Badaloo, Michael S Boyne, Clive Osmond, Debbie Thompson, Curtis Green, Carolyn Taylor-Bryan, Alan Barnett, Suzanne Soares-Wynter, Mark A. Hanson, Alan S. Beedle, Peter D. Gluckman, 'Prenatal Factors Contribute to the Emergence of Kwashiorkor or Marasmus in Severe Undernutrition: Evidence for the Predictive Adaptation Model', *PLoS ONE*, 7, no. 4 (2012), e35907.

Food and Agricultural Organization, 'Human Energy Requirements', *Food and Nutrition Technical Report Series* (2004).

Frisancho, A. Roberto, *Human Adaptation and Accommodation* (Ann Arbor, 1993).

Gibbs, Tyson, Kathleen Cargill, Leslie Sue Lieberman and Elizabeth Reitz, 'Nutrition in a Slave Population: An Anthropological Examination', *Medical Anthropology: Cross-Cultural Studies in Health and Illness*, 4, no. 2 (1980), pp. 175-262.

Gluckman, Peter D. and Mark A. Hanson, 'Evolution, Development and Timing of Puberty', *Trends in Endocrinology and Metabolism*, 17, no. 1 (2006), pp. 7-12.

Gluckman, Peter D. and Mark A. Hanson, 'The Consequences of Being Born Small – An Adaptive Perspective', *Hormone Research*, 65, suppl. no. 3 (2006), pp. 5-14.

Gluckman, Peter D., Mark A. Hanson, Hamish G. Spencer and Patrick Bateson, 'Environmental Influences during Development and their Later Consequences for Health and Disease: Implications for the Interpretation of Empirical Studies, *Proceedings of the Royal Society: Biological Sciences*, 272 (2005), pp. 671-77.

Godfrey, Keith M., Karen A. Lillycrop, Graham C. Burdge, Peter D. Gluckman and Mark A. Hanson, 'Epigenetic Mechanisms and the Mismatch Concept of the Developmental Origins of Health and Disease', *Pediatric Research*, 61, no. 5 (2007), pp. 5R-10R.

Goldin, Claudia, and Robert A. Margo, 'The Poor at Birth: Birth Weights and Infant Mortality at Philadelphia's Almshouse Hospital, 1848–1873', *Explorations in Economic History*, 26, no. 3 (1989), pp. 360-379.

Hanson, Mark, Torvid Kiserud, Gerard H A Visser, Peter Brocklehurst, and Eric Schneider, 'Optimal Fetal Growth – A Misconception?', *American Journal of Obstetrics and Gynecology*, 213, no. 3 (2015), pp. 332-34.

Hatton, T. J., 'How have Europeans grown so tall?', *Oxford Economic Papers* (2013).

Higman, B. W., *Slave Populations of the British Caribbean, 1807-1834* (Baltimore, 1984).

Horrell, Sara and Deborah Oxley, 'Bargaining for Basics? Inferring Decision Making in Nineteenth-Century British Households from Expenditure, Diet, Stature, and Death', *European Review of Economic History*, 17, no. 2 (2013), pp. 147-170.

Huss-Ashmore, Rebecca, 'Theory in Human Biology: Evolution, Ecology, Adaptability, and Variation', in Sara Stinson, Barry Bogin, Rebecca Huss-Ashmore and Dennis O'Rourke (eds.), *Human Biology: An Evolutionary and Biocultural Perspective* (New York, 2000), pp. 1-25.

Jasienska, Grazyna, 'Low Birth Weight of Contemporary African Americans: An Intergenerational Effect of Slavery?', *American Journal of Human Biology*, 21, no. 1 (2009), pp. 16-24.

Jasienska, Grazyna, Inger Thune and Peter T. Ellison, 'Fatness at birth predicts adult susceptibility to ovarian suppression: an empirical test of the Predictive Adaptive Response hypothesis', *Proceedings of the National Academy of Sciences of the United States of America*, 103, no. 34 (2006), pp. 12759-12762.

Jasienska, Grazyna, *The Fragile Wisdom: An Evolutionary View on Women's Biology and Health* (Cambridge, 2013).

Jirtle, Randy L. and Michale K. Skinner, 'Environmental Epigenomics and Disease Susceptibility', *Nature Reviews Genetics*, 8 (April 2007), pp. 253-62.

Johnson, Paul, and Stephen Nicholas, 'Male and Female Living Standards in England and Wales, 1812- 1867: Evidence from Criminal Height Records', *The Economic History Review*, 48, no. 3 (1995), pp. 470-481.

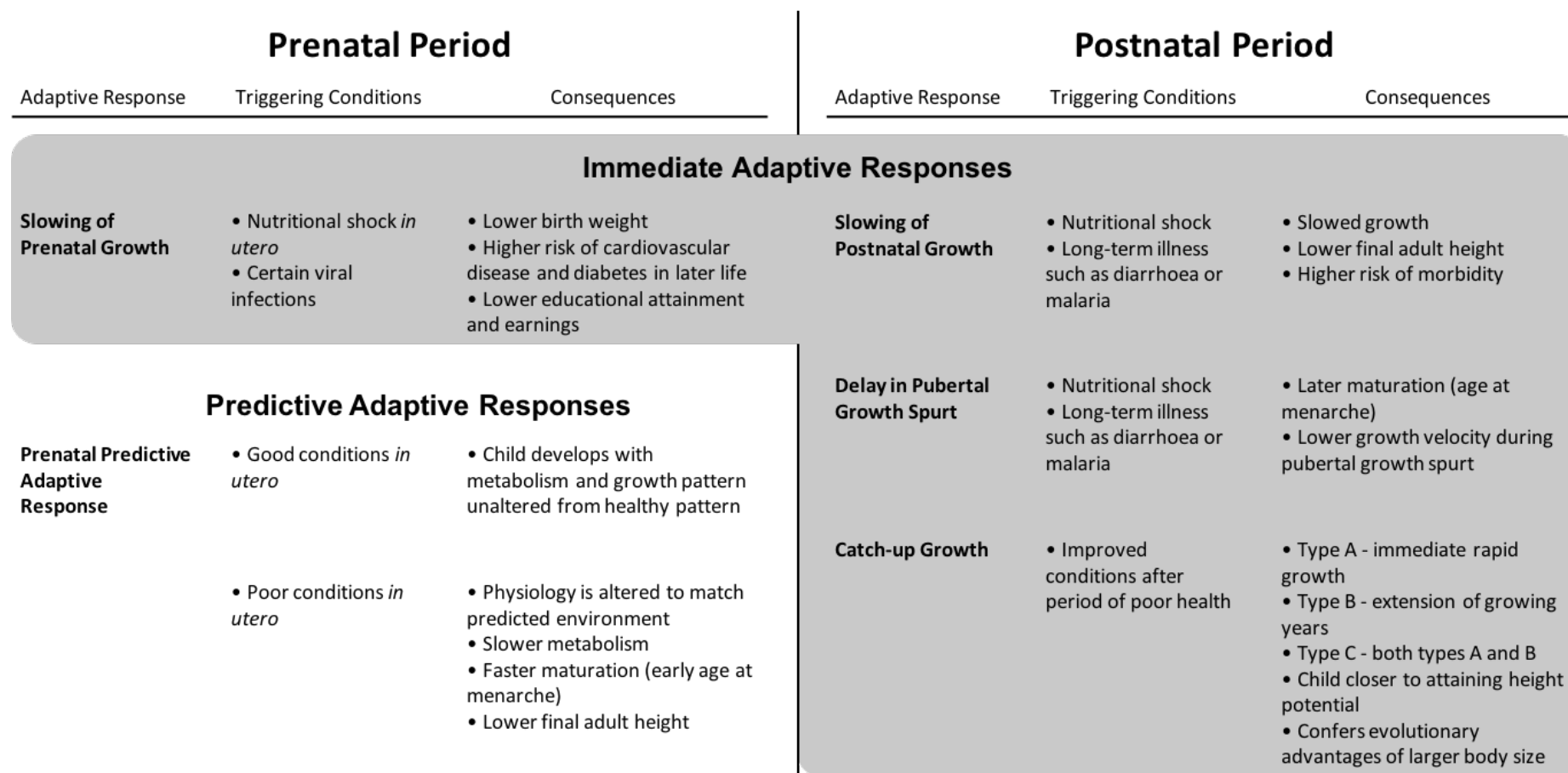Karlberg, J. and Z. C. Luo, 'Foetal Size to Final Height', *Acta Paediatrica*, 89, no. 6 (2000), pp. 632-36.

Kiple, Kenneth F. and Virginia H. Kiple, 'Slave Child Mortality: Some Nutritional Answers to a Perennial Puzzle', *The Journal of Social History*, 10, no. 3 (1977), pp. 284-309.

Kiple, Kenneth F., and Virginia H. Kiple, 'Deficiency Diseases in the Caribbean', *Journal of Interdisciplinary History*, 11, no. 2 (1980), pp. 197-215.

Klein, Herbert S., and Stanley L. Engerman, 'Fertility Differentials between Slaves in the United States and the British West Indies: a Note on Lactation Practices and their Possible Implications', *The William and Mary Quarterly*, 35, no. 2 (1978), pp. 357-374.

Komlos, John and Bjorn Alecke, 'The Economics of Antebellum Slave Heights Reconsidered', *Journal of Interdisciplinary History*, 26, no. 3 (1996), pp. 437-57.

Kramer, Michael S. 'Determinants of Low Birth Weight: Methodological Assessment and Meta-analysis', *Bulletin of the World Health Organization*, 65, no. 5 (1987), pp. 663-737.

Løkke, Anne, 'The Antibiotic Transformation of Danish Obstetrics: The Hidden Links between the Decline in Perinatal Mortality and Maternal Mortality in the mid-Twentieth Century', *Annales de démographie historique*, 123, no. 1 (2012), pp. 205-24.

Malcolm, L. A., *Growth and Development in New Guinea: A Study of the Bundi People of the Madang District* (Chipping Norton, 1970).

Marino, Deborah D., 'Water and Food Safety in the Developing World: Global Implications for Health and Nutrition of Infants and Young Children', *Journal of the American Dietetic Association*, 107, no. 11 (2007), pp. 1930-34.

Martorell, Reynaldo, Jean-Pierre Habicht, and Juan A. Rivera, 'History and Design of the INCAP Longitudinal Study (1969-1977) and its Follow-up (1988-89)', *The Journal of Nutrition*, 125, no. 4 (1995), pp. 1027S-1041S.

Meredith John, A., 'Plantation Slave Mortality in Trinidad', *Population Studies*, 42, no. 2 (1988), pp. 161-182.

Nicholas, Stephen, and Deborah Oxley, 'The Living Standards of Women during the Industrial Revolution, 1795-1820', *The Economic History Review*, 46, no. 4 (1993), pp. 723-749.

Osmani, Siddiq, and Amartya Sen, 'The Hidden Penalties of Gender Inequality: Fetal Origins of Ill-Health', *Economics and Human Biology*, 1 (2003), pp. 105-121.

Panagariya, Arvind 'Does India Really Suffer from Worse Child Malnutrition Than Sub-Saharan Africa?', *Economic & Political Weekly*, 158, no. 18 (2013), pp. 98-111.

Prentice, Andrew M., Kate A. Ward, Gail R. Goldberg, Landing M. Jarjou, Sophie E. Moore, Anthony J. Fulford, and Ann Prentice 'Critical Windows for Nutritional Interventions against Stunting', *American Journal of Clinical Nutrition*, 97, no. 5 (2013), pp. 911-918.

Pritchett, Jonathan B. 'The Interregional Slave Trade and the Selection of Slaves for the New Orleans Market', *Journal of Interdisciplinary History*, 28, no. 1 (1997), pp. 57-85.

Pritchett, Jonathan B. and Herman Freudenberger, 'A Peculiar Sample: The Selection of Slaves for the New Orleans Market', *Journal of Economic History*, 52, no. 1 (1992), pp. 109-27.

Pritchett, Jonathan B. and Herman Freudenberger, 'A Peculiar Sample: A Reply to Steckel and Ziebarth', *Journal of Economic History*, 76, no. 1 (2016), pp. 139-62.

Proos, Lemm A. 'Growth & Development of Indian Children Adopted in Sweden', *Indian Journal of Medical Research*, 130, no. 6 (2009), pp. 646-650.

Proos, Lemm, Jan Gustafsson, 'Is Early Puberty Triggered by Catch-Up Growth Following Undernutrition?', *International Journal of Environmental Research and Public Health*, 9, no. 12 (2012), pp. 1791-1809.

Quaranta, Luciana, 'Early Life Effects across the Life Course: The Impact of Individually Defined Exogenous Measure of Disease Exposure on Mortality by Sex in 19th- and 20th-century Southern Sweden', *Social Science & Medicine*, 119 (2014), pp. 266-73.

Rosenberg, Margit, 'Birth Weights in Three Norwegian Cities, 1860-1984. Secular Trends and Influencing Factors', *Annals of Human Biology*, 15, no. 4 (1988), pp. 275-288.

Savitt, Todd L., *Medicine and Slavery: The Diseases and Health Care of Blacks in Antebellum Virginia* (Urbana, 1981).

Schaible, Ulrich E. and Stefan H. E. Kaufmann, 'Malnutrition and Infection: Complex Mechanisms and Global Impacts', *Public Library of Science Medicine*, 4, no. 5 (2007), pp. 806-12.

Schwartz, Marie Jenkins, *Born in Bondage: Growing Up Enslaved in the Antebellum South* (Cambridge, 2000).

Shetty, Prakash, 'Malnutrition and Undernutrition', *Medicine*, 34, no. 12 (2006), pp. 524-29.

Silventoinen, Karri, Kirsi H. Pietiläinen, Per Tynelius, Thorkild I. A. Sørensen, Jaakko Kaprio and Finn Rasmussen, 'Genetic Regulation of Growth from Birth to 18 Years of Age: The Swedish Young Male Twin Study', *American Journal of Human Biology*, 20 (2008), pp. 292-98.

Steckel, Richard H. 'Slave Mortality: Analysis of Evidence from Plantation Records', *Social Science History*, 3, no. 3/4 (1979), pp. 86-114.

Steckel, Richard H. 'A Dreadful Childhood: The Excess Mortality of American Slaves', *Social Science History*, 10, no. 4 (1986), pp. 427-465.

Steckel, Richard H., 'A Peculiar Population: The Nutrition, Health, and Mortality of American Slaves from Childhood to Maturity', *Journal of Economic History*, 46, no. 3 (1986), pp. 721-741.

Steckel, Richard H., 'Birth Weights and Infant Mortality among American Slaves', *Explorations in Economic History*, 23 (1986), pp. 173-98.

Steckel, Richard H. 'Growth Depression and Recovery: The Remarkable Case of American Slaves', *Annals of Human Biology*, 14, no. 2 (1987), pp. 111-132.

Steckel, Richard H., 'Diets Versus Diseases in the Anthropometrics of Slave Children: A Reply', *Journal of Economic History*, 60, no. 1 (2000), 247-59.

Steckel, Richard H., 'Heights and Human Welfare: Recent Developments and New Directions', *Explorations in Economic History*, 46 (2009), pp. 1-23.

Stinson, Sara, 'Growth Variation: Biological and Cultural Factors', in Sara Stinson, Barry Bogin, Rebecca Huss-Ashmore and Dennis O'Rourke (eds.), *Human Biology: An Evolutionary and Biocultural Perspective* (New York, 2000).

Steckel, Richard H. and Nicolas Ziebarth, 'Trader Selectivity and Measured Catch-up Growth of American Slaves', *Journal of Economic History*, 76, no. 1 (2016), pp. 109-38.

Stinson, Sara, 'Nutritional Adaptation', *Annual Review of Anthropology*, 21 (1992), pp. 143-70.

Strauss, John, and Duncan Thomas, 'Health over the Life Course', in *Handbook of Development Economics Vol. 4* (2007), pp. 3375–3474.

Sutch, Richard, 'The Treatment Received by American Slaves: A critical review of the evidence presented in *Time on the Cross*', *Explorations in Economic History*, 12 (1975), pp. 335-438.

Tafari, N., R. L. Naeye, and A. Gobezie, 'Effects of Maternal Undernutrition and Heavy Physical Work during Pregnancy on Birth Weight', *British Journal of Obstetrics and Gynaecology*, 87, no. 3 (1980), pp. 222-226.

Tanner, J. M., *Growth at Adolescence, second edition* (Oxford, 1962).

Tracer, David P., R. John Sturt, Agnes Sturt, and Lara M Braithwaite, 'Two Decade Trends in Birth Weight and Early Childhood Growth in Papua New Guinea', *American Journal of Human Biology*, 10 (1998), pp. 483-93.

Ulijaszek, Stanley J., 'Evidence for a Secular Trend in Heights and Weights of Adults in Papua New Guinea', *Annals of Human Biology*, 20, no. 4 (1993), pp. 349-55.

Via, Sara, Richard Gomulklewicz, Gerdien de Jong, Samuel M. Scheiner, Carl D. Schlichting and Peter H. van Tienderen, 'Adaptive Phenotypic Plasticity: Consensus and Controversy', *Trends in Ecology and Evolution*, 10, no. 5 (1995), pp. 212-17.

Victora, Cesar Gomez, Mercedes de Onis, Pedro Curi Hallal, Monika Blössner and Roger Shrimpton, 'Worldwide Timing of Growth Faltering: Revisiting Implications for Interventions', *Pediatrics*, 125, no. 3 (2010), pp. e473-e480.

Villar, José *et al.*, 'International standards for newborn weight, length, and head circumference by gestational age and sex: the Newborn Cross-Sectional Study of the INTERGROWTH-21st Project', *Lancet*, 384 (2014), pp. 857-868.

Wang, Youfa, Luis A. Moreno, Benjamin Caballero, and Tim J. Cole, 'Limitations of the Current World Health Organization Growth References for Children and Adolescents', *Food and Nutrition Bulletin*, 27, no. 4 (2006), pp. s175-s188.

Ward, W. Peter, *Birth Weight and Economic Growth: Women's Living Standards in the Industrializing West* (Chicago, 1993).

Woods, Robert, *Death before Birth: Fetal Health and Mortality in Historical Perspective* (Oxford, 2009).

Woods, Robert I., Anne Løkke, and Frans van Poppel, 'Two Hundred Years of Evidence-Based Perinatal Care: Late-fetal Mortality in the Past', *Archives of Disease in Childhood*, 91, no. 6 (2006), pp. F445-47.

World Bank, World Development Indicators, available at http://data.worldbank.org/, accessed 2 April 2014.

World Health Organization Multicentre Growth Reference Study Group, 'Assessment of Differences in Linear Growth among Populations in the WHO Multicentre Growth Reference Study', *Acta Paediatrica*, Supplement, 450 (2006), pp. 56- 65.

World Health Organization, *Neonatal and Perinatal Mortality: Country, Regional and Global Estimates 2004* (Geneva, 2007).

Wrigley, E. A., 'British Population during the "Long" Eighteenth Century, 1680-1840', in Roderick Floud and Paul Johnson (eds.), *The Cambridge Economic History of Modern Britain – Volume I: Industrialization, 1700-1860* (Cambridge, 2004).

Figure 1: Schematic of the adaptive framework for children's growth.

Prenatal Period

Adaptive Response	Triggering Conditions	Consequences

Immediate Adaptive Responses

Adaptive Response	Triggering Conditions	Consequences
Slowing of Prenatal Growth	• Nutritional shock *in utero* • Certain viral infections	• Lower birth weight • Higher risk of cardiovascular disease and diabetes in later life • Lower educational attainment and earnings

Predictive Adaptive Responses

Adaptive Response	Triggering Conditions	Consequences
Prenatal Predictive Adaptive Response	• Good conditions *in utero*	• Child develops with metabolism and growth pattern unaltered from healthy pattern
	• Poor conditions *in utero*	• Physiology is altered to match predicted environment • Slower metabolism • Faster maturation (early age at menarche) • Lower final adult height

Postnatal Period

Adaptive Response	Triggering Conditions	Consequences
Slowing of Postnatal Growth	• Nutritional shock • Long-term illness such as diarrhoea or malaria	• Slowed growth • Lower final adult height • Higher risk of morbidity
Delay in Pubertal Growth Spurt	• Nutritional shock • Long-term illness such as diarrhoea or malaria	• Later maturation (age at menarche) • Lower growth velocity during pubertal growth spurt
Catch-up Growth	• Improved conditions after period of poor health	• Type A - immediate rapid growth • Type B - extension of growing years • Type C - both types A and B • Child closer to attaining height potential • Confers evolutionary advantages of larger body size

Sources: Developed from Gluckman and Hanson, 'Evolution'; Gluckman and Hanson, 'Consequences'; and Gluckman *et al.*, 'Environmental'.
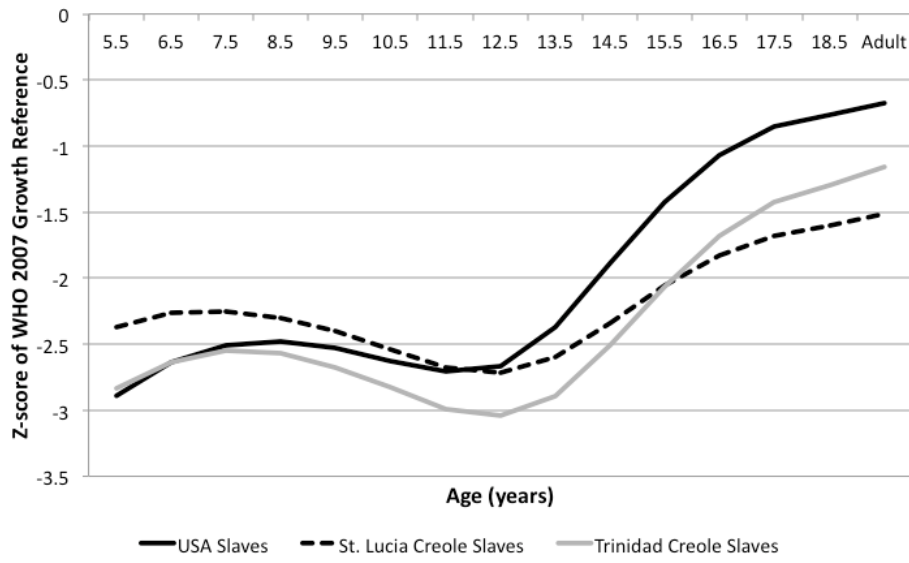
Figure 2: Heights of slave boys in the US and Caribbean reported as Z-scores of the WHO 2007 reference.
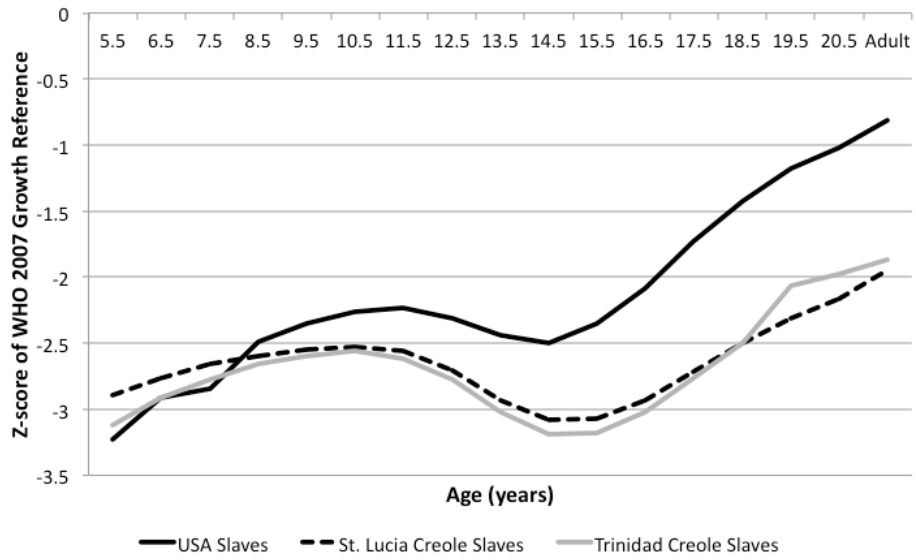
Notes: The Z-scores were calculated based on the WHO 2007 growth reference from the Preece-Baines smoothed figures reported in Steckel, 'Growth Depression'.

Sources: Steckel, 'Growth Depression', pp. 117, 122.

Figure 3: Heights of slave girls in the US and Caribbean reported as Z-scores of the WHO 2007 reference.

Notes and Sources: see figure 2.

Figure 4: Hypothetical and observed growth patterns of slave boys in the antebellum US South and the Caribbean

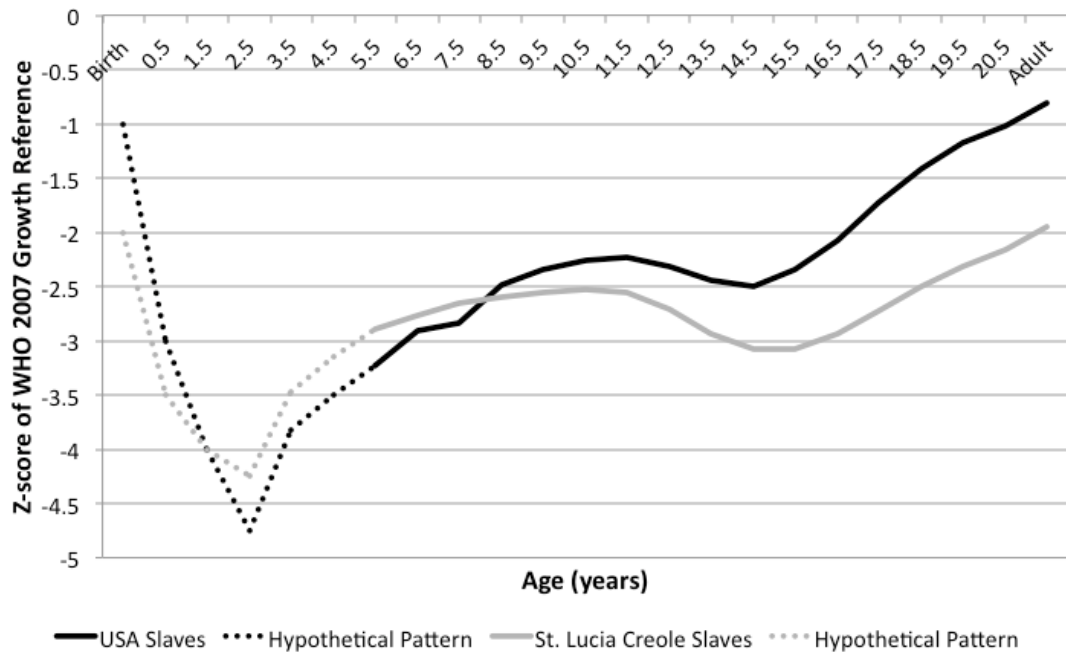*Notes*: The Z-scores were calculated based on the WHO 2007 growth reference from the Preece-Baines smoothed figures reported in Steckel, 'Growth Depression'. Hypothetical growth trajectory predicted from adaptive framework (see text).

Sources: Steckel, 'Growth Depression', pp. 117, 122.

Table 1: Birth weights of children in historical populations compared with children of like populations born in the 1970s and 1980s.

Country/Place	Historical			Modern (1970s-80s)		
	Years	Mean Birth Weight (g)	LBW (%)	Years	Mean Birth Weight (g)	LBW (%)
North America						
Boston, USA						
Whites	1872-1900	3,409	5.9	1974-77	3,480	3.6
Blacks	1872-1900	3,094	12.3	1974-77	3,230	7.7
All	1872-1900	3,397	6.2	1977	3,299	7.4
New York, USA	1910-1931	3,463	5.5	--	--	--
Philadelphia, USA	1848-1873	3,403	8.1	--	--	--
Montreal, Canada	1851-1905	3,375	5.7	1988	3,303	6.3
Europe						
Vienna, Austria	1865-1930	3,097	12.9	1978	3,320	5.8
Dublin, Ireland	1869-1930	3,282	8.0	1978-79	3,473	4.4
Edinburgh, Scotland	1847-1920	3,132*	15.2	1974	3,062*	7.7
Three Cities, Norway	1860-1920	3,400-3,500	--	--	--	--

Notes: All samples reflect mainly working class populations. LBW means low birth weight, a birth weight under 2,500 grams. *Scottish mean birth weight includes both live births and stillbirths, driving down the mean figure.

Sources: New York – Costa, 'Unequal', p. 992; Philadelphia – Goldin and Margo, 'Poor', p. 365; Norway – Rosenberg, 'Birth Weights', p. 281; All Others – Ward, *Birth Weight*, p. 134.

Response to Criticisms of the Predictive Adaptive Response (PAR) Hypothesis

Although the predictive adaptive responses (PAR) model seems to be widely accepted as a working theory by human biologists and there is mounting evidence to support it, it does have critics. Bogin *et al.* challenged the PAR hypothesis by arguing that children exposed to poor conditions *in utero* have lower survival rates, reproduction and productivity than children exposed to healthy conditions *in utero*.[1] To support these assertions, Bogin and his co-authors primarily use the famous Institute of Nutrition of Central America and Panama (INCAP) intervention studies conducted in Guatemala in the 1960s and 1970s.[2] The INCAP study was an experiment designed to test the effects of early life interventions on children's health and development. The study consisted of four Guatemalan villages (two treatment two control). In the treatment villages children under the age of seven and pregnant or lactating mothers were given a drink with 39 kilojoules of energy and 11.5 grams of protein whereas in the control villages mothers and children were given a placebo drink containing 14 kilojoules of energy. Micronutrients were added to both the treatment and control drinks after two years.[3]

While the INCAP study was extremely innovative and influential, it had some very serious experimental design flaws that suggest caution when interpreting its results. First, the researchers assigned treatment and control groups at the village level, making it impossible to control for village-level effects that may have influenced responses to the intervention. In addition, because only children under the age of seven were allowed to participate, there may have been substitution of food within households toward older children, making differences between exposed and unexposed cohorts difficult to interpret. Finally, the study did not control the amount of the drink that pregnant mothers consumed. Thus, mothers in control

---

[1] Bogin *et al.*, 'Life History'.
[2] Martorell *et al.*, 'History'.
[3] Strauss and Thomas, 'Health', p. 3429.

villages drank more than mothers in treatment villages so that the number of calories obtained was similar in both the treatment and control with mothers in control villages actually consuming more micronutrients. In the end, birth weights in the treatment and control were nearly identical, suggesting that there were not really differences in early life health across the villages.[4] These study design problems make it difficult to accept the findings that Bogin *et al.* use to contradict the PAR hypothesis.

Aside from the problems with accepting the INCAP results at face value, there are two other problems with evaluating the PAR hypothesis in the way that Bogin *et al.* do. First, in order for a trait to become prominent through natural selection over hundreds of thousands of years, it only has to be correct 51 per cent of the time. Thus, there may not be a statistically significant difference in survival between groups, but this does not mean that there is not an evolutionarily significant difference. Finally, testing the differences in survival rates, reproduction and productivity of children facing poor or favourable health conditions *in utero* does not actually provide the correct comparison to test the PAR model because there is not a proper control group. Both groups of children in the comparison would be adapted to best fit their environment, and if this were true, Bogin *et al.*'s findings would not be surprising. We would expect children who were healthy early in life to be better off later in life. However, the perfect control group would be children who faced poor conditions *in utero* but continued to grow as if they had not experienced poor conditions, i.e. children who do not experience an adaptive response in growth based on environmental conditions. Unfortunately, these children are unobservable since all children experience adaptive responses in growth. However, it is not difficult to speculate that children that experienced adaptive responses to environmental conditions would have higher survival rates and reproductive fitness than children who did not. Thus, more intricate and elaborate tests are

---

[4] Strauss and Thomas, 'Health', p. 3429-30.

required to decipher whether the PAR hypothesis holds. Two such tests have shown support for the analysis so far.[5]

Table A1: Birth weights of children around the world in the 1980s.

Country	Mean Birth Weight (g)	LBW (%)
Latin America		
Brazil	3,170-3,298	9.0
Chile	3,340	9.0
Colombia	2,912-3,115	10.0
Guatemala	3,050	17.9
Mexico	3,019-3,025	11.7
Africa		
Egypt	3,200-3,285	7.0
Kenya	3,143	12.8
Nigeria	2,880-3,117	18.0
Tunisia	3,210-3,376	7.3
United Republic of Tanzania	2,900-3,151	14.4
Zaire	3,163	15.9
Asia		
China	3,215-3,285	6.0
India	2,493-2,970	30.0
Indonesia	2,760-3,027	14.0
Iran	3,012-3,250	14.0
Iraq	3,540	6.1
Japan	3,200-3,208	5.2
Malaysia	3,027-3,065	10.6
Pakistan	2,770	27.0

Sources: Kramer (1987, p. 665).

[5] Forrester *et al.*, 'Prenatal factors'; Jasienska *et al.*, 'Fatness'.