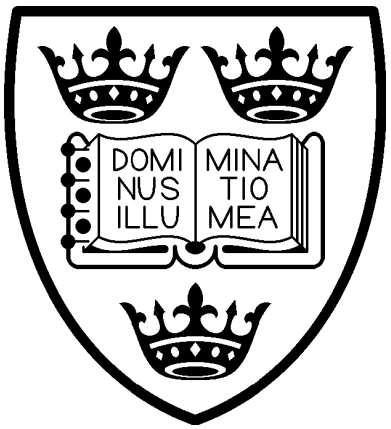

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Abstract

This paper presents a new adaptive framework for understanding children's growth in the past. Drawing upon the recent work of Gluckman and Hanson (2006) and their co-authors on adaptive responses in relation to growth, I present three prenatal and three 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 foetus develops assuming that the postnatal environment will closely match prenatal conditions. Thus, the metabolism and growth trajectory of a child is programmed during the prenatal period: children experiencing good conditions *in utero* would have a higher metabolism and growth trajectory than their counterparts facing poor conditions. Having discussed the framework and other responses in detail, I then use it to reinterpret the growth pattern of American slaves (Steckel, 1979, 1986). I argue that the mismatch between relatively good conditions *in utero* and absolutely appalling conditions in infancy and early childhood led slave children to become incredibly stunted by age three or four. However, after this age, slave children experienced rapid catch-up growth, first because their immune systems had become more developed and had adapted to the poor disease environment and later because their diet improved tremendously and hookworm exposure was reduced when they entered the labour force around age ten. Thus, American slave children were able to experience rapid 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.

[1] The author wishes to thank Deborah Oxley and Richard Follett and participants at the European Historical Economics Society Congress in London, the Centre for the Study of African Economies Conference in Oxford, the Economic History Society Conference in Warwick, and history and economics departmental seminars at the University of Sussex for helpful comments on the paper. The usual disclaimer applies.

Human growth is an extremely complex process driven by both genetic inheritance and epigenetic adaptation 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 under good nutritional and disease environments, these scholars mostly ignored genetic or 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, genetic, and other 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 incredibly 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 will first introduce the adaptive framework for understanding children's growth and then apply this framework to the 'peculiar' pattern of slave children's growth in antebellum America and the Caribbean. Later, 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. This framework points toward four important issues, which should form the basis for additional and continued research on children's growth. It highlights the importance of the nutritional environment *in utero* and maternal health for later growth; the deleterious consequences of mismatches between the prenatal and postnatal environment; the critical role morbidity may play in shaping children's growth; and the different ways in which girls and boys may adapt their growth to similar environmental conditions.

1. Children's Growth in an Adaptive Framework

1.1 Defining Adaptive Responses

Before delving into the adaptive models of children's growth in detail, 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.[3]

[2] See for instance Stinson, 'Growth Variation', p. 456; Silventoinen *et al.*, 'Genetic Regulation', p. 292.
[3] Frisancho, *Human Adaptation*, pp. 4-5; Huss-Ashmore, 'Theory', pp. 13-15.

Thus, they are adaptations that increase the probability that an organism will survive to reproductive age and produce viable offspring. Immediate adaptations confer 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.[4] Whether adaptations confer improved fitness to the organism in both the short and long run is not an absolute but a relative calculation.[5] 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.

1.2 Prenatal Growth in an Adaptive Framework

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. Barker initiated much research and debate when he published his hypothesis in 1990, sometimes known as the 'early origins hypothesis' or 'foetal programming', suggesting that foetuses limit their growth and permanently change their physiology and metabolism based on the availability of nutrients in the womb. Barker further argued that these physiological and metabolic changes could be responsible for higher incidences of coronary heart disease, hypertension, stroke, and diabetes.[6] Since then, there has been a tremendous amount of scholarship published attempting to explain the detailed mechanisms of foetal conditioning for risk of diesease, the ways nutrients reach the foetus in the womb, and the consequences of poor health conditions *in utero* for longevity, height and morbidity in later life.

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 foetus's response to poor environmental conditions in the womb. First, if nutrient conditions in the womb are extremely dire, the foetus could experience developmental disruption, which is not adaptive and can lead to severe birth defects.[7] Second, an immediate adaptive delay in growth might allow the foetus 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 foetus where nutrient conditions *in utero* are taken to be a good prediction of future conditions and the growth, metabolism, and physiology of the foetus are adapted to best fit that predicted environment. In other words, if a foetus is undernourished in the womb, epigenetic and hormonal processes adapt it to be able to survive better in a postnatal environment with limited nutrients.[8]

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[4] Gluckman et al., 'Environmental Influences', p. 672.
[5] Stinson, 'Nutritional Adaptation', p. 161.
[6] Barker, 'Fetal and Infant Origins', p. 1111; Barker, 'Maternal Nutrition', pp. 807, 812.
[7] Gluckman et al., 'Environmental Influences', p. 672.
[8] The theory of predictive adaptive responses (PAR) is not without its detractors. In a 2007 paper Bogin *et al.* argued that existing evidence does not support PAR, but most of their evidence was

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. The foetal predictive adaptive mechanism permanently changes the child's physiology, altering the velocity and timing of growth and maturation. 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 foetus reaches reproductive viability early and has a better chance of reproducing before it dies.[9]

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 increased the prevalence of heart disease, stroke, and diabetes in later life.[10] Cohorts exposed to poor conditions *in utero* have also experienced non-adaptive scarring such as cognitive impairments and lower educational attainment and earnings.[11]

Clearly, impaired prenatal growth can have many serious consequences for the postnatal life of the foetus. However, these consequences, some more negative than others, do not preclude an adaptive framework from the prenatal predictive response. If the foetus 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 foetal 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.[12]

1.3 Postnatal Growth in an Adaptive Perspective

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 nutrition or disease insult. However, there is an important difference between prenatal and postnatal adaptive responses.

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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 Bogin *et al.* ascribe to it. Bogin *et al*. 'Life History'; Strauss and Thomas, 'Health over the Life Course', pp. 3429-3432. See web appendix for more information.

[9] 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.

[10] Barker, 'Maternal Health', pp. 811-12; Jirtle and Skinner, 'Environmental Epigenomics', p. 253; Doniloy and Jirtle, 'Environmental Epigenomics', pp. 4-5.

[11] Silventoinen *et al.*, 'Genetic Regulation', p. 292; Gluckman and Hanson, 'Consequences', p. 8; Almond and Currie, 'Killing Me Softly'.

[12] Gluckman and Hanson, 'Consequences', p. 10.

Because phenotypic plasticity, 'an environmentally based change' in physical characteristics, is greatest during the embryonic and foetal period of a child's development, prenatal responses can have long lasting if not entirely permanent effects on the child's physiology and growth.[13] 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.[14]

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, a child who undergoes postnatal stunting is neither healthy nor normal. The child is at a higher risk of disease because malnourishment weakens the immune system, and the child is knocked off its genetically and epigenetically defined growth curve.[15]

In order to understand whether these adaptations are beneficial to the child, the costs of the trait must be measured and compared with alternate outcomes. Shorter and underweight children have a higher risk for morbidity and mortality at all ages.[16] However, before completely ruling out the adaptive nature of the response, one must consider the alternative to stunting in the face of a nutritional or disease insult: the child continues to grow in the poor environmental conditions and needs to consume more and more calories to continue growing. When these calories are not available, the child would be more susceptible to disease and begin to starve with horrible consequences. The stunted child would therefore have a higher relative level of fitness than the child who continued to grow.

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 by the female to postpone reproduction hoping that environmental conditions would improve before she incurred the high energetic cost of pregnancy and lactation.[17] 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.

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. 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

[13] Via et al., 'Adaptive Phenotypic Plasticity', p. 212; Feinberg, 'Phenotypic Plasticity', p. 438.
[14] Gluckman and Hanson, 'Consequences', p. 11; Proos and Gustafsson, 'Early'.
[15] Shetty, 'Malnutrition and Undernutrition', pp. 525-28; Schaible and Kaufmann, 'Malnutrition and Infection', pp. 806-7.
[16] Fogel, *Escape from Hunger*, pp. 23-27; Barker, 'Maternal Nutrition', pp. 807, 812; Barker, 'Fetal'.
[17] Gluckman and Hanson, 'Evolution', p. 9.

cessation of growth. Type C catch-up growth includes both type A and type B catch-up growth.[18] 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 appears to be differences in the type of catch-up growth children who faced different forms of deprivation can achieve. Gluckman and Hanson discovered that sheep exposed to intrauterine undernutrition experienced less catch-up growth following a postnatal nutritional insult than sheep who had a normal, healthy prenatal environment. In addition, 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 happened more slowly and was smaller on an absolute scale than normal birth weight children.[19] Prenatal predictive adaptive responses also influence final height potential. Karlberg and Luo found in a study of healthy, full-term Swedish babies that foetal size, measured as length at birth, along with the genetic height potential, measured as midparental height, played a strong role in defining the final height potential of children.[20] 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 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 period may trigger earlier pubertal development, limiting the amount of type B catch-up growth children could achieve.[21] 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. In this sense, 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.[22] However, other factors such as disease, work, and climate 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.[23] In addition, diarrhoea can significantly decrease dietary energy and nutrient absorption further exacerbating the problems of malnutrition.[24] Child labour

[18] Boersma and Wit, 'Catch-up Growth', pp. 647-648; Stinson, 'Growth Variation', pp. 451-452.
[19] Gluckman and Hanson, 'Consequences', p. 8.
[20] Karlberg and Luo, 'Foetal Size', pp. 632, 635-36.
[21] Proos, 'Growth', pp. 647-649.
[22] Shetty, 'Malnutrition and Undernutrition', p. 524; Dauncey and Pell, 'Genetic Regulation', pp. 143-144; Noguchi, 'Protein Nutrition', p. s241.
[23] Schaible and Kaufman, 'Malnutrition and Infection', p. 806.
[24] Marino, 'Water', pp. 1930-32; Caulfield *et al.*, 'Undernutrition', p. 197.

can also influence growth patterns if large amounts of energy are required to work.[25] Finally, climate can also influence growth because if a child lives in a cooler climate, he/she will have to expend higher amounts of energy to maintain his/her body temperature during cold winters.[26] 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 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.

2. Applying the Adaptive Framework: The Peculiar Growth Trajectory of Slaves in Antebellum America and the Caribbean

2.1 Background on the growth of slaves in the antebellum South

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. This section seeks to demonstrate the utility of this framework by using it to reinterpret 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.

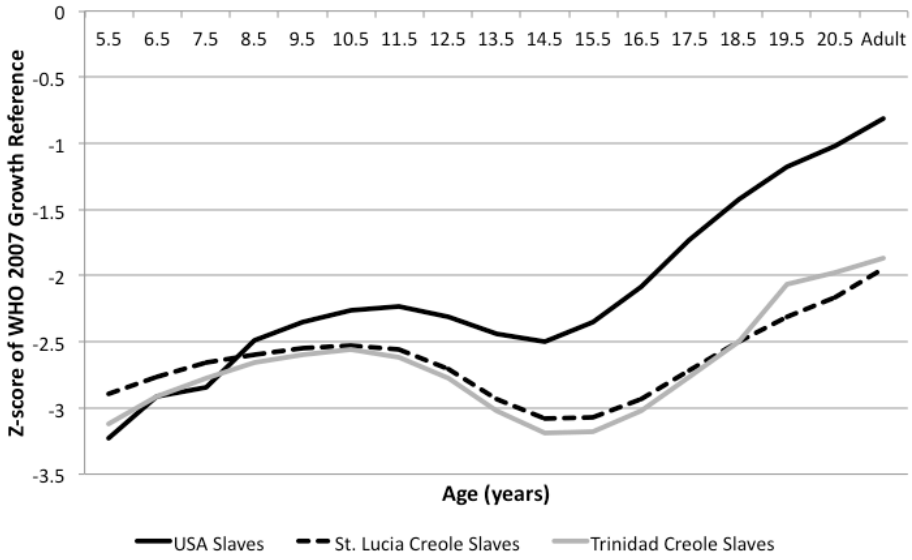
Steckel was the first to study the heights of slave children and compare them with modern growth standards. To do this, he drew upon a large number of ship manifests created to monitor the coastwise slave trade after the Atlantic slave trade was abolished in 1807. Steckel found that slave children had extremely low birth weights and were very stunted in early life with males at 3.23 standard deviations below modern standards and females at 2.89 standard deviations below modern standards (recalculated according to 2006/7 WHO standards) at age 5.5. However, by the time that the slave children had stopped growing after age 21, the average person was only 0.81 and 0.67 standard deviations below modern growth standards for men and women respectively.[27] 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.[28] Steckel argues that the 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.[29] Explaining the poor growth outcomes at younger ages and improving health outcomes by the time slaves reached adulthood has been more difficult. Steckel argued that slave owners had different incentives to invest in

[25] Steckel, 'Heights and Human Welfare', p. 10.
[26] Komlos, 'Anthropometric History', pp. 178-9.
[27] Steckel, 'Peculiar Population', pp. 724-725. These Z-scores were calculated from the heights that Steckel reported using the WHO 2007 growth reference.
[28] Prentice *et al.*, 'Critical Windows', pp. 914-916.
[29] Steckel, 'Peculiar Population', pp. 724-726.

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.[30] 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.[31]

Steckel's original argument has been challenged on several fronts. Pritchett and Freudenberger and Bodenhorn *et al.* have argued that the slave heights in coastwise manifests were upwardly biased compared to the rest of the slave population because masters preferred to sell and ship taller slaves. This bias was especially prominent among young children in the slave manifests, but was not quite so important for adults.[32] This is confirmed by a later study, which showed that slave children shipped alone were taller than slave children shipped with their mothers.[33] Thus, it seems that the average height of slave children in the population was lower than the figures that Steckel reports especially at younger ages. This means that Steckel underestimated the children's catch-up growth, which was even greater than what is reported above.[34] However, Komlos and Alecke argue that slaves in Maryland were taller than the slaves in the coastwise manifest, so they argue there was no clear upward bias in the coastwise manifest.[35]

Figure 1: Heights of slave boys in the US and Caribbean reported as Z-scores of the WHO 2007 reference.



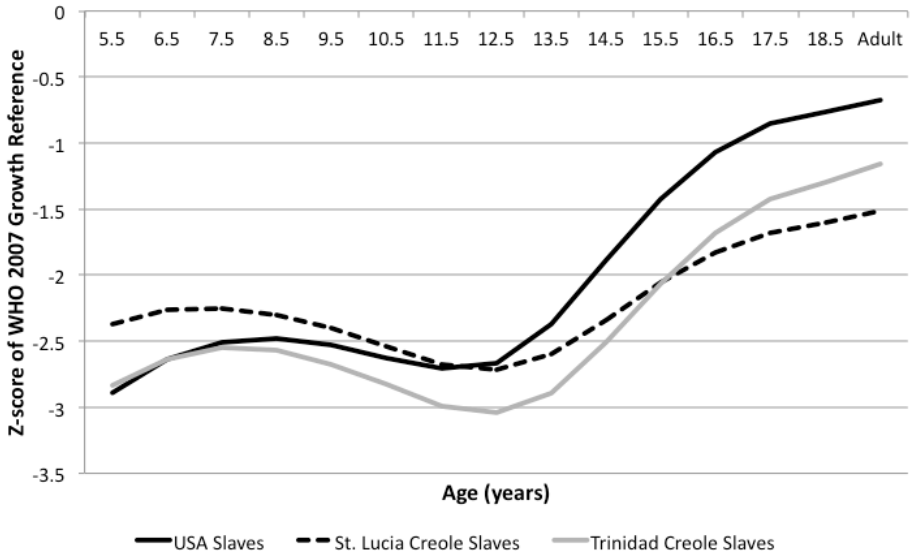
Notes: The Z-scores were calculated based on the WHO 2007 growth reference from the Preece-Baines smoothed figures reported in Steckel, 'Growth Depression'.

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

[30] Steckel, 'Peculiar Population', pp. 735-736.
[31] Steckel, 'Peculiar Population', pp. 732-733.
[32] Pritchett and Freudenberger, 'A Peculiar Sample', pp. 125-126; Pritchett, 'Interregional Slave Trade', pp. 83-85; Bodenhorn *et al.*, 'Problems', pp. 29-33.
[33] Calomiris and Pritchett, 'Preserving Slave Families'.
[34] Pritchett, 'Interregional Slave Trade', pp. 83-85; Bodenhorn *et al.*, 'Problems', pp. 29-33.
[35] Komlos and Alecke, 'Economics', pp. 455-457.

Coelho and McGuire have also challenged Steckel's conclusions arguing that slave children's slow growth can be better explained by hookworm exposure rather 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]

Figure 2: Heights of slave girls in the US and Caribbean reported as Z-scores of the WHO 2007 reference.



Notes and Sources: see figure 1.

Steckel's analysis is sound, but it is worth noting that by using percentiles to understand growth trajectories, he is only able to describe the relative catch-up growth of the population not the absolute catch-up, which is best measured using a Z-score. This is because 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.[38] The difference between percentile change and Z-score change is important because it leads Steckel to understate the catch-up growth that occurs for American slaves before the age of 10 (figures 1 and 2). It also leads Steckel to be somewhat dismissive

[36] Coelho and McGuire, 'Diets', pp. 239-241.
[37] Steckel, 'Diets', pp. 249-254.
[38] Wang *et al.*, 'Limitations', p. s180.

of the catch-up growth of slaves in the Caribbean when their catch-up was actually quite impressive.[39]

2.2 Slave children's growth in an adaptive framework

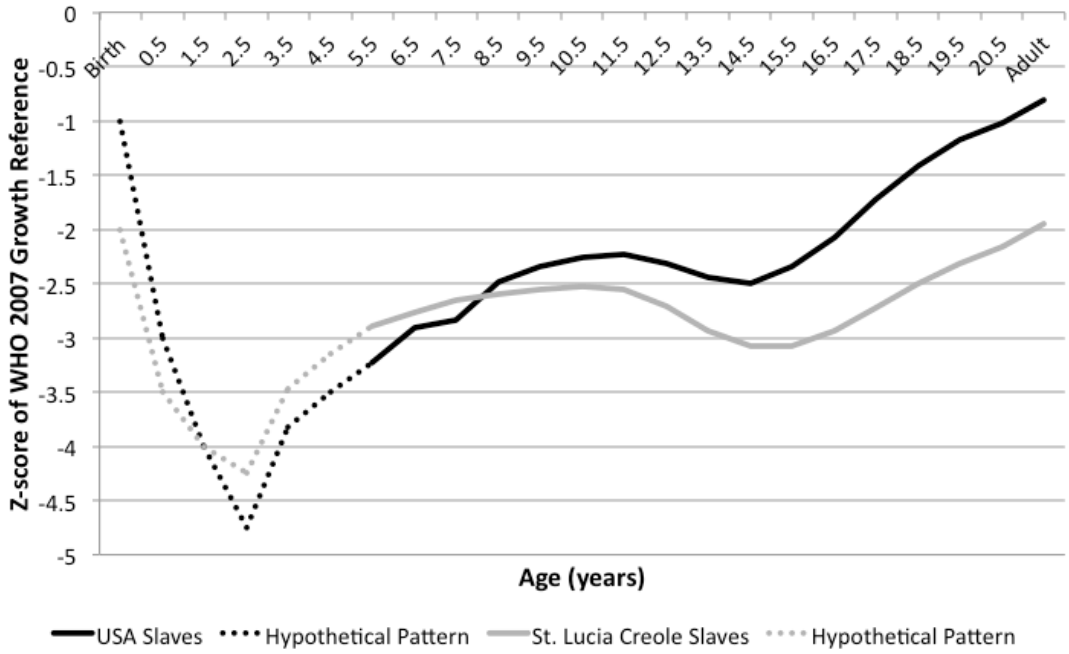
Although these authors' arguments are interesting, there is quite an intriguing puzzle hidden in their extensive work. In another paper, Steckel argues that slaves had the lowest average birth weights of any population ever studied, suggesting very poor early life health conditions. Yet American slaves experienced incredibly dramatic catch-up growth.[40] If prenatal conditions are extremely important for future growth and may even trump catch-up growth are correct, 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.[41]

However, an interpretation of slave children's growth in an adaptive framework would better explain the pattern of growth that Steckel observes (see figure 3 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 once they entered the labour force.[42] This ensured that prenatal conditions were fairly good for the growing foetus compared to other historical populations, though obviously malaria exposure and the strenuous labour slaves had to perform would have still made the conditions less than ideal. Relatively good conditions *in utero* provoked a predictive adaptive response. Slave foetuses 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 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.[43] The children first started experiencing catch-up after age 3.5, perhaps because their immune systems became better adapted to the disease environment or because shorter children died at higher rates during this period.[44] But, persistently poor conditions likely also

[39] Steckel, 'Peculiar Population', p. 728; Higman, *Slave Populations*, pp. 534-535.
[40] Steckel, 'Birth Weights'.
[41] Proos and Gustafsson, 'Early Puberty'.
[42] Steckel, 'Peculiar Population', pp. 736-740.
[43] Steckel, 'Birth Weights', p. 178.
[44] Prentice *et al.* found catch-up growth between the ages of 3 and 7 in Gambian children. They believed 'that a combination of the normal postnatal maturation of the children's immune systems and the development of a broad repertoire of adaptive responses against previously encountered pathogens reduces the frequency and severity of growth-impairing infections'. Prentice *et al.*, 'Critical Windows', p. 914.

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 sparked by the children's entry into the labour force, which provided them with a more ample and balanced diet, most importantly including meat 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.

Figure 3: 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.

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; 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.

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.[45] 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.[46] Kiple and Kiple also dispute that adult slaves had adequate nutrition arguing that they were deficient in iron and calcium, but it is unclear how much worse this would have been relative to other populations, which also had inadequate diets in the early and mid nineteenth century.[47] However, in a study of South Carolina, Georgia and Florida, Gibbs *et al.* concluded 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'.[48]

Despite the contrary evidence, in a recent paper Jasienska concludes that 'slavery was associated with poor nutritional status in utero, childhood, and adulthoood'.[49] 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 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.[50] 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.[51] In addition, the timing and size of catch-up growth that slaves of both genders experienced is evidence that once they entered the labour force, they were given more than enough nutrients to grow despite their high work requirements.[52] 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.[53]

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.

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[45] Fogel and Engerman, *Time on the Cross*, pp. 107-144.
[46] Sutch, 'Treatment', pp. 359-360, 382-386.
[47] Kiple and Kiple, 'Slave Child Mortality', p. 287.
[48] Gibbs *et al.*, 'Nutrition', p. 251.
[49] 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 Wisdom*, p. 126; Jasienska, 'Low Birth Weight'.
[50] Johnson and Nicholas, 'Health', pp. 211, 219; Nicholas and Oxley, 'Living Standards', p. 733.
[51] de Beer, 'Physical Stature', p. 64.
[52] Steckel, 'Peculiar Population'.
[53] Steckel, 'Dreadful Childhood', pp. 427-429.

The second key assertion is that the environmental and nutritional conditions that slave children faced in infancy had to be exceptionally bad. There is substantial evidence to support this claim. Steckel notes that 'it is doubtful that populations of the past could have been much, if any, worse off than slaves as young children'.[54] Slave children were weaned incredibly early, probably around three months and certainly within one year. Early weaning not only deprived slave children of key nutrients and protein but also made them more susceptible to diseases since they were not receiving anti-bodies through their mothers' milk and could be served contaminated food.[55] In addition, slave children were given a very poor diet of corn, hominy and fat, lacking in protein and micronutrients necessary for growth. This poor diet left slave children especially prone to protein-calorie malnutrition. Slave infants also suffered from acute deficiencies in calcium, magnesium, and iron, which greatly increased infant mortality.[56] Finally, slave children were left unsupervised during the day, increasing their exposure to diseases especially in the cramped and unsanitary dwellings in which they were forced to live. Thus, they 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.[57] Clearly, environmental and nutritional conditions for slaves in infancy and early childhood were horrendous and could easily have been worse than the conditions they faced in the prenatal period.

Third, it seems fairly likely that Steckel significantly underestimated slaves' birth weights. Because there is no reliable evidence on slaves' birth weights, 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.[58] 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 children to have followed the same growth trajectory from birth until that point; they cannot have experienced any catch-up growth or stunting relative to their prenatally programmed growth trajectory. This would seem unlikely given the evidence presented above that suggests that infancy and early childhood were periods of extremely poor nutrition and health for slave children. 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.[59]

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[54] Steckel, 'Birth Weights', p. 740.

[55] Steckel, 'Birth Weights', p. 732; Steckel, 'Dreadful Childhood', p. 449; Klein and Engerman, 'Fertility Differentials'; Kiple and Kiple, 'Slave Child Mortality', p. 288.

[56] Steckel, 'Birth Weights', p. 189; Kiple and Kiple, 'Slave Child Mortality', pp. 285-286, 289, 296-297.

[57] Savitt, *Medicine and Slavery*, pp. 57-73; Kiple and Kiple, 'Slave Child Mortality', pp. 298-299; Coelho and McGuire, 'Biology'; Coelho and McGuire, 'Diets'.

[58] Steckel, 'Birth Weights', p. 181.

[59] Boersma and Wit, 'Catch-up Growth', p. 647. Steckel reports that children ages one and two were even shorter relative to modern standards than children aged three and four. However, as Steckel notes, taking proper measurements for children who cannot stand is very difficult. Therefore, it is likely that the average heights for ages one and two significantly understate the children's actual length. Steckel, 'Birth Weights', p. 177.

In addition, the birth weights that Steckel predicted were incredibly low. At 2,320 grams, they are a full 1,180 grams below the modern standard of 3,500 grams. Of the poor populations that he uses to predict the birth weight only one population, the Lumi of New Guinea, had a birth weight below 2,840 grams.[60] In addition, by the early twentieth century, African Americans children born at Johns Hopkins Hospital in Baltimore had a mean birth weight of 3,183 grams.[61] 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, but this still leaves an 800 gram deficit.[62] Steckel attributes this 800 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.[63] However, it seems unlikely that slaves' work could have such a large effect on birth weight for two reasons. First, the largest measured effect of strenuous work during pregnancy on birth weight was a deficit of 210 grams, far lower than 800 grams.[64] And second, in a meta-analysis of 34 studies that measured the influence of maternal work on birth weight, Kramer found 'the effect of maternal work on intra-uterine growth [to be] uncertain'. A negative effect was certainly consistent with biological principles, but Kramer seemed to believe that maternal work would be most detrimental if the mother were malnourished.[65] Thus, it seems likely that slave children had substantially higher birth weights than Steckel estimated and thus better early life health conditions.

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.[66] Even Indian girls adopted in Sweden in the second half of the twentieth century, probably the most optimal conditions imaginable, only experienced type A catch-up growth of 1.55 standard deviations relative to modern standards in two years.[67] Thus, it is clear that the catch-up growth of American slaves was truly remarkable, especially considering that the intervention sparking 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 low birth weight[68] Indian children 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

[60] Steckel, 'Birth Weights', p. 181.
[61] Costa, 'Race', p. 1065.
[62] Steckel, 'Birth Weights', pp. 185-188.
[63] Kramer, 'Determinants', p. 696.
[64] Tafari *et al.*, 'Effects', p. 222; Steckel, 'Birth Weights', p. 187.
[65] Kramer, 'Determinants', pp. 696-697; Coelho and McGuire, 'Diets', p. 239.
[66] Prentice *et al.*, 'Critical Windows', pp. 915-916.
[67] Proos, 'Growth', p. 647.
[68] Below 2,000 grams.

onset of the pubertal growth spurt and menarche and did not reach heights significantly taller than their counterparts in India. This pattern has been observed with malnourished children from other countries adopted in developed countries as well.[69] 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. This evidence is corroborated by the fact that low birth weight children tend to be less responsive to hormones controlling growth.[70]

The direct implications of these studies for slaves' growth is slightly difficult to understand because the effectiveness of the intervention for the Indian girls was substantially 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.[71] 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.[72] Thus, the low average birth weight of American slaves seems inconsistent with their incredibly 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 rapid catch-up growth once they had adapted to the disease environment, their diets had improved and their exposure to hookworm had decreased.

2.3 Caribbean slave children's growth: A robustness check

The adaptive framework seems plausible in explaining the growth pattern of American slaves, but can it also explain the similar trajectory of Caribbean slaves, who in many ways suffered worse conditions?[73] 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

[69] Proos and Gustafsson, 'Early Puberty'; Proos, 'Growth', pp. 647-649.
[70] Gluckman and Hanson, 'Consequences', 8.
[71] Gluckman and Hanson, 'Evolution', p. 9.
[72] Proos and Gustafsson, 'Early Puberty'.
[73] Even after the abolition of the Atlantic slave trade in 1807, almost all slave populations in the Caribbean had negative rates of natural increase. Higman found that this was due to a combination of high mortality and low fertility among slaves. Higman, *Slave Populations*, pp. 374-378.

American slave counterparts and shorter than or equal to women in England and Ireland. 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.[74] Caribbean slave women were also forced to work long hours and were given rum as part of their rations.[75] Infant mortality rates were incredibly high in St. Lucia and Trinidad, 583 and 479 deaths per 1,000 according to model life tables, suggesting that birth weights were quite low since birth weight is a strong predictor of perinatal mortality.[76] 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.[77] This evidence suggests that birth weights in the Caribbean were lower than in the US. However, in terms of child health, slave mothers tended to wean their children at later stages in the Caribbean than in the US, which may have given the Caribbean slave children an advantage if they survived the first month of life.[78] These practices may have contributed to the lower levels of childhood and adolescent mortality in the Caribbean when compared with the antebellum American South.[79] The diets of slaves in the Caribbean and in the US South were fairly similar with nutrition improving after slaves entered the workforce in both places.[80] Thus, it seems likely that slave birth weights in the Caribbean were lower than in the US South, but conditions in childhood were slightly better.

Placing Caribbean slave children's growth in the adaptive framework (figure 3), 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. The Caribbean children's advantage could have been caused by a better disease environment in childhood, but it could also suggest that the mismatch between the prenatal and postnatal environment was not as great for Caribbean slaves. Although conditions were still probably worse in the postnatal period than the prenatal period, the difference was smaller, which meant that Caribbean slave children did not fall behind modern standards as quickly as American slave children. Their metabolism better matched their postnatal experience. Clearly, an adaptive framework for growth can be incredibly instructive when trying to understand the growth trajectories of different populations.

3. Explaining Historical Patterns of Children's Growth in an Adaptive Framework

[74] Steckel, 'Growth Depression', p. 122; Higman, *Slave Populations*, p. 281.
[75] Steckel, 'Peculiar Population', p. 729; Higman, *Slave Populations*, pp. 205, 211. Steckel shows that alcohol consumption levels were lower for slaves in the US.
[76] Higman, *Slave Populations*, p. 319; Floud *et al.*, *Changing Body*, p. 16; Wrigley, 'British Population', p. 73.
[77] Steckel, 'Peculiar Population', p. 733.
[78] Klein and Engerman, 'Fertility Differentials'; Higman, *Slave Populations*, pp. 353-354.
[79] Higman, *Slave Populations*, pp. 654-657; Meredith John, 'Plantation Slave Mortality', pp. 170-171; Steckel, 'Peculiar Population', p. 733; Steckel, 'Slave Mortality', p. 92.
[80] Kiple and Kiple, 'Deficiency Diseases', pp. 200-203; but see Higman, *Slave Populations*, pp. 204-205 for a more sceptical opinion.

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 some 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 in the Western World.
2. There is considerable heterogeneity of birth weights 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.
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.[81] Of course looking only at averages may hide considerable change in the underlying distributions. For instance, modern medicine has allowed many low birth weight children to survive who might have been stillborn a century ago and increased use of Caesarean-section surgery may have allowed more large infants to survive childbirth. The stability of birth weights is also confounded by sharp decreases in other proxies for early life health such as stillbirths, neo-natal mortality and endogenous infant mortality over the past 150 years.[82] However, in the end it is difficult to dispute the birth weight evidence which remains quite stable.

Second, there is considerable heterogeneity in birth weights around the world. 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 2). Some developing countries did perform better, though, with relatively high birth weights in China, Chile, and Iraq.[83] However, the fact that the historical samples that Ward measured were in the upper part of the 1980s birth weight distribution suggests that children in North America and Europe were likely never exposed to the worst intra-uterine conditions now present in the developing world.

[81] Ward, *Birth Weight*, p. 133-135; Goldin and Margo, 'Poor at Birth', p. 377; Costa, 'Unequal at Birth', pp. 1005-1006.
[82] Woods, *Death*, pp. 76, 79, 96, 100; Woods, *Demography*, p. 253-55; Oris *et al.*, 'Infant and Child Mortality', p. 362; Wrigley, 'British Population', p. 71.
[83] Kramer, 'Determinants', p. 665.

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

	Historical			Modern (1970s-80s)		
Country/Place	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 at Birth', p. 992; Philadelphia – Goldin and Margo, 'Poor at Birth', p. 365; Norway – Rosenberg, 'Birth Weights', p. 281; All Others – Ward, *Birth Weight*, p. 134.

The third stylized fact is that children in the past had later pubertal growth spurts than healthy children in the present. The 2007 WHO growth reference predicts that boys and girls should reach the peak of the pubertal growth spurt at ages 13.125 and 10.875 respectively. 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.[84] Thus, there has been a transition over the past 100 years where the pubertal growth spurt has occurred earlier in children of both sexes.

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.[85] 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.[86]

[84] Steckel, 'Growth Depression', p. 127.
[85] Tanner, *Growth at Adolescence*, pp. 152-55.
[86] Proos and Gustafsson, 'Early Puberty', pp. 1800-1802.

Table 2: 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, 'Determinants', p. 665.

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 fact that birth weights in the Western World were already quite high in the second half of the nineteenth century suggests that the negative consequences of low birth weight were not as strong in these countries as they are in some developing countries today. Thus, the predictive adaptive response was likely not as powerful a constraint on children's growth in North America and Europe 150 years ago as it would 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.[87] Good prenatal conditions in the North America and Europe also allowed these populations to experience greater catch-up growth if conditions improved. Thus, these historical European populations should be considered as different than populations in developing countries today who may suffer from a double burden of poor health in both the prenatal and postnatal periods.

[87] Diamond, 'Double Puzzle', p. 601.

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 secular increase in adult height over the past 150 years was caused by a reduction in postnatal stunting over time, and this fits the historical picture. Hatton has recently found that improvements in the disease environment and to a lesser extent increases in income and education and decreases in family size were responsible for driving the secular trend in stature across Europe.[88]

However, we must be careful before applying the policy implications from research conducted on Western populations to the rest of the world. As mentioned above, a number of 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. In addition, the double burden may mean that different factors are more important in slowing improvements in health in developing countries than existed in Western countries in the late nineteenth and twentieth centuries. One clear example of this is the persistence of stunting rates over the past twenty years among Indian children under the age of five despite substantial economic growth during that period. 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.[89] Instead, it seems reasonable to believe that poor conditions *in utero* are placing limits on improvements in child health (proxied by growth) over time. Thus, the percentage of children under 5 stunted and the percentage of low birth weight children have both remained quite stagnant, especially relative to other countries. Stunting fell from 57.1 per cent in 1992 to 47.9 per cent in 2006 and the low birth weight percentage has also remained relatively stagnant falling from 31.7 per cent in 1993 to 27.6 per cent in 2006 according to the most recent WHO/World Bank data.[90] 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.[91]

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.

4. Conclusion

[88] Hatton, 'How Have Europeans Grown so Tall?'.

[89] Panagariya, 'Does India Really Suffer'. The WHO growth reference for children under 5 is based on children's longitudinal growth measured in the WHO Multicentre Growth Reference Study (MGRS) which included healthy children from Brazil, Ghana, India, Norway, Oman and the USA. They 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'.

[90] World Bank, World Development Indicators.

[91] Osmani and Sen, 'Hidden Penalties'; Horrell and Oxley, 'Bargaining'.

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.

The utility of this framework was then tested by reinterpreting the growth of slaves in the US 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 greatest for US slaves because maternal health was relatively good there and thus birth weights were high. 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 their immune system adapted to the disease environment and they received better food, 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 restricted by their metabolism and growth trajectory set in the prenatal period. Therefore, it seems that placing children's growth in an adaptive framework can be a very helpful way of understanding growth patterns in the past.

Finally, the paper introduced four stylized facts of children's historical growth experience in the past 150 years. Analysing growth in an adaptive framework helps to clarify some important issues that should be addressed in future research. First, the link between early life health, maternal health and catch-up growth highlights the important role maternal health and the conditions a foetus is exposed to *in utero* play in determining growth outcomes. Thus, researchers should focus on measuring the relative health status of men and women and explaining any differences that may exist. Unfortunately, historical records of male height have defined large periods of anthropometric history because there is such a dearth of sources with information on female stature. In light of the adaptive framework, this male centred view is not just a one-sided perspective of living standards as many feminists have argued; it is a fundamentally skewed view because maternal conditions play such a strong role in determining growth and height potential. We also need to build upon existing research to reconstruct proxies for early life health over time. Exogenous shocks like the 1918 flu pandemic may provide robust strategies for identifying the long-term effects of poor early life health, but these shocks do not reflect normal conditions that would help us understand how predictive adaptive responses may have changed over time.

Second, mismatches between the prenatal and postnatal environment can create substantial health problems for children. If prenatal conditions were substantially better than postnatal conditions, as was the case for American slaves, then the children would experience dramatic stunting in the first couple of years of life because their metabolism was set too high for their environment. This mismatch could have modern corollaries when famine may cause postnatal conditions to be worse than prenatal conditions or when the disease environment favours high levels of morbidity and mortality in infancy and early childhood. However, the consequences for a mismatch where prenatal conditions were worse than postnatal conditions could also have health consequences. If metabolism was set low in the prenatal period but resources were more abundant thereafter, then the children would have an increased risk of developing childhood obesity and its associated health consequences.

Third, the fact that all historical populations studied experienced a later pubertal growth spurt than modern populations suggests that insults that could cross class boundaries, such as disease, were more important in triggering an immediate adaptive response to delay the pubertal growth spurt than income or nutrition. Morbidity and chronic diseases such as hookworm, diarrhoea and malaria in particular may have influenced the growth patterns of populations as much as nutrition. Thus, more work needs to be done showing how bouts with particular diseases affected children's growth and how the ecology and prevalence of diseases in different geographic areas affected children's growth.

Fourth, understanding growth in an adaptive framework highlights the need for a greater focus on gender inequality and gender differences in growth during childhood. Factors influencing the pattern of female growth and final adult height may be quite different than those of men, not just because women were treated differently than men in past societies but because different factors might trigger adaptive responses in women and men and there may be biological differences in their adaptive responses. A meta-analysis of all known historical and modern studies on children's growth might be a good starting point to make further progress on this issue.

Clearly, applying adaptive models to anthropometric history validates some older insights and raises new questions about factors affecting children's growth and later health. A clear focus in the future on foetal and maternal health, gender inequality, morbidity and mismatches between prenatal and postnatal environments will hopefully help historians formulate a more accurate and balanced account of children's health and growth in the past.

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Web Appendix

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*.[92] 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.[93] 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.[94]

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 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.[95] 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

[92] Bogin *et al.*, 'Life History', pp. 6-8.
[93] Martorell *et al.*, 'History'.
[94] Strauss and Thomas, 'Health', p. 3429.
[95] Strauss and Thomas, 'Health', p. 3429-3430.

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 required to decipher whether the PAR hypothesis holds (see Jasienska *et al.*, 2006).

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