

# The determinants of child stunting and shifts in the growth pattern of children: A long-run, global review

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## Abstract

This article explores how child growth has changed over the past 150 years and links changes in child growth to the recent decline in child stunting in low- and middle-income countries (LMICs). The article begins by defining the four characteristics of the growth pattern in height: size at birth, size at adulthood, the timing of the pubertal growth spurt, and the speed of maturation. It then shows how these characteristics have changed over time and links these characteristics to child stunting. Stunted children are too short for their age relative to healthy standards, and their share in the population is used as an indicator of malnutrition in LMICs today. The article then surveys the literature on the causes of changes in the growth pattern and reductions in child stunting, comparing research on current LMICs with historical research on current high-income countries (HICs) in the past. To limit the scope of the contemporary literature, I focus on explanations of the so-called “Indian enigma:” why Indian children are shorter than sub-Saharan African children despite India’s lead in many indicators of economic development. The article

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closes with ideas for what historical and contemporary researchers can learn from one another.

#### KEYWORDS

child health, child stunting, growth pattern, health transition, Indian enigma, living standards

#### JEL CLASSIFICATION

I10, I14, I15, J13, J16, N30, O15

## 1 | INTRODUCTION

Child growth is sensitive to the nutritional and environmental conditions to which children are exposed. Children who are malnourished or have repeated bouts of infection grow more slowly than healthy children, their scarring visible in their trajectory of growth (Steckel, 1995). Because of this sensitivity, child stunting, the share of children under age 5 who are too short for their age (formally defined below), is a leading indicator of population malnutrition and reducing stunting has been an important target in both the Millennium and Sustainable Development Goals. Although child stunting has been declining in recent years, there are still 148.1 million children in the world who are stunted (Unicef et al., 2023). Stunted children not only face poor conditions in early life, but their unhealthy growth scars them, leading to poorer labor market and human capital outcomes and affecting their health later in life (Alderman et al., 2006; Hoddinott et al., 2013; Roberts et al., 2023). Thus, there has been a large amount of research and policy focus on eradicating child stunting.

This research in nutrition, global health, demography, and development economics has helped reduce child stunting around the world, and it has sometimes even taken an explicitly historical approach focussing on the past 40 years (Nisbett et al., 2023). However, eliminating child stunting is actually a consequence of shifting children from an unhealthy growth pattern to a healthier one. The growth pattern in height is defined by four characteristics: size at birth, size in adulthood, the timing of the pubertal growth spurt and the speed of maturation (defined below). These characteristics are interrelated and determined by individual genetic variability and by adverse health shocks during sensitive periods of development that can push children away from their genetically determined path (Wells, 2017). The secular change in the growth pattern is an essential part of the health transition, which has been repeated to varying degrees and in different time periods in most populations around the world (Deaton, 2013; Floud et al., 2011). Thus, there is value in taking a long run view on the shift in the growth pattern and the causes underpinning these changes.

This review presents global changes in the growth pattern of children since the 19th century, juxtaposing research on historical periods for high-income countries (HICs) today with research on more recent periods for low- and middle-income countries (LMICs). It first presents the characteristics of the growth pattern in height of children<sup>1</sup> and discusses how researchers use growth standards and references to assess growth (Sections 2 and 3). It then shows that there are many similarities in the shift in the growth pattern across the health transition comparing countries around the world (Section 4). There are important differences as well though, especially in relation to birth size. Next, the review analyses factors that have led to changes in the growth pattern. It compares historical research with contemporary research seeking to explain the so-called “Indian

Enigma,” the fact that Indian children are shorter than sub-Saharan African children despite higher levels of development in India (Section 5). I consider among others the importance of economic growth; nutrition; water, sanitation, and hygiene (WASH); unequal allocation of household resources; maternal health; and atmospheric pollution for changes in the growth pattern and reductions in child stunting. The review then considers which periods of child development are most sensitive to health shocks and interventions and the possibility that children can recover from adverse health shocks in early life (Section 6). Finally, I explore the insights gained from comparing the literature on historical HICs with current LMICs, emphasizing unanswered questions and areas for further research (Section 7).

## 2 | UNDERSTANDING THE GROWTH PATTERN IN HEIGHT

As mentioned above, there are four characteristics of the growth pattern in height of individuals: size at birth, size in adulthood, the timing of the pubertal growth spurt, and the speed of maturation. The first characteristic of the growth pattern is the size of a child at birth. Figure 1a plots the length of children at birth according to the WHO standard, but I will also discuss birth weights as a proxy for size since birth length is subject to considerable measurement error, especially historically, and is not as widely used as an indicator. The second characteristic is the final adult height of an individual once they have stopped growing, the most commonly studied characteristic of the growth pattern among historians. These two characteristics of the growth pattern are easiest to measure and interpret because they relate to height attained at a particular age rather than changes in growth over age.

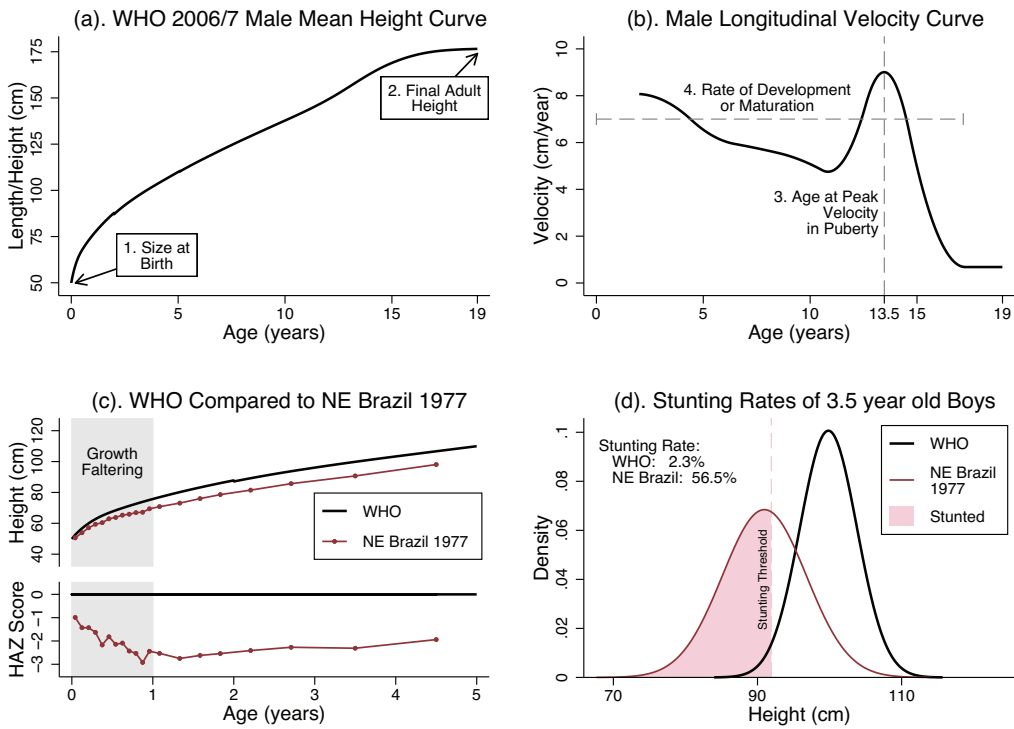
The next characteristics of the growth pattern move beyond the height curve and are based on its first derivative, the velocity curve, illustrated in Figure 1b using Tanner and Davies (1985)'s male longitudinal growth reference. The third characteristic is the timing of the pubertal growth spurt, measured by the age at peak growth velocity in adolescence. There is heterogeneity in the timing of the pubertal growth spurt across individuals, but as shown in the individual-level longitudinal curve, this is a very pronounced experience for boys with growth velocity often doubling the prespurt level at the peak of the spurt. The pubertal growth spurt is somewhat less pronounced for girls and occurs at earlier ages. The final characteristic of the growth pattern is an individual's speed of maturation or development. The rate of development will determine whether they cease to grow relatively young or continue experiencing growth into their twenties. It also determines how quickly an individual grows throughout the growing years because a person who attains their final height in 16 years will have to grow more quickly than a person who attains the same adult stature in 22 years of growth.

These four characteristics are not determined independently. Instead, they are a product of genetic endowments and biological responses to the environment in order to increase an individual's evolutionary fitness (Gluckman & Hanson, 2006a, 2006b; Wells, 2017): see Online Appendix A for more details.

## 3 | ASSESSING HEALTHY AND UNHEALTHY CHILD GROWTH

### 3.1 | The WHO 2006 growth standard and 2007 growth reference

There is considerable variation in the characteristics of the growth pattern across individuals, making it difficult to determine whether an individual child is experiencing typical growth or



**FIGURE 1** Characteristics of the growth pattern in height using males as an example. *Notes:* Panel (a) shows the WHO 2006/7 mean height for boys. Panel (b) shows the growth velocity from Tanner and Davies (1985)'s longitudinal growth curve smoothed using the SITAR growth model (Cole, 2019). Growth velocity was truncated below age 2 so that the pubertal growth spurt would be visible. Panel (c) shows growth faltering by comparing the height of boys in the WHO standard with the heights of Brazilian boys in the Northeastern Region measured in 1977 as part of the ENDEF study. Panel (d) shows predicted distributions of height of 3.5-year-old boys in the WHO standard and in the Northeastern region of Brazil in 1977. *Sources:* WHO (2006), de Onis et al. (2007), Tanner and Davies (1985), Fundação Instituto Brasileiro de Geografia e Estatística (1977, pp. 69-72). [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

not. Twin studies have shown that genetic heterogeneity can explain 70%–80% of the variation in adult height (McEvoy & Visscher, 2009; Silventoinen et al., 2008).<sup>2</sup> This means that some children will be short because they were genetically predisposed to be short whereas others may have experienced a variety of adverse health shocks that caused them to be shorter than they otherwise would have been. The difficulty is distinguishing between the two cases, and the only way to do this is to establish a growth reference, against which to compare individuals.

Growth references or standards are simply data that, in their simplest form, present the mean heights by age and sex of a large number of children. More sophisticated references or standards will also report Z-scores or percentiles of height at each age so that one can determine an individual's relative position in the distribution. There has been much debate over the last 50 years about whether an international growth standard is realistic and/or useful (Butte et al., 2007; Eveleth, 1976; Haas & Campirano, 2012; Seidell et al., 2006; Wang et al., 2006). Growth standards are meant to be prescriptive, to indicate the rate and level of growth of children “that has been associated empirically with specified health outcomes and the minimization of long-term risks of disease” (Butte et al., 2007, p. 154). Growth references, on the other hand, represent the

growth of a specific population and are meant merely as a point of comparison rather than as a recommendation for growth. Although growth references allow scholars to compare the heights of individuals within a population, they do not allow for cross-country comparisons since there would be no basis for picking one population over another as the normative standard for growth. Scholars have been creating growth references since the late 19th century (Bowditch, 1891), but the development of growth standards is more recent.<sup>3</sup>

The current WHO international growth standard and reference were constructed in the early 2000s. For children under age 5, the WHO Multicentre Growth Reference Study (MGRS), a population-based growth survey, was conducted between July 1997 and December 2003 in six cities around the world: Davis, California, USA; Oslo, Norway; Pelotas, Brazil; Muscat, Oman; Accra, Ghana, and New Delhi, India. The MGRS combined longitudinal data from ages 0 to 24 months with cross-sectional measurements of children taken between 18 and 71 months. The inclusion criteria for the MGRS were as follows: no environmental constraints on growth; full term, singleton births; mothers willing to follow WHO feeding recommendations; no significant morbidity; and a nonsmoking mother. In Muscat, Accra, and New Delhi, additional screening mechanisms were introduced to capture children whose growth had not been constrained. Thus, the goal of the MGRS was to measure the growth of healthy children to set a normative standard for children under age 5. This culminated with the publication of the 2006 WHO Child Growth Standards (WHO, 2006), which have become widely used around the world (de Onis et al., 2012).

Conducting a similar international study for children over the age of 5 was deemed infeasible, so an expert group sought to construct a new reference for school-aged children from historical data. After reviewing hundreds of studies, they decided to use the United States National Center for Health Statistics (NCHS) study, which was conducted in the mid-1970s and was ethnically diverse and representative of the United States population. However, the NCHS data were adjusted so that it would accord with the growth standard for children under 5 and not understate obesity in the BMI growth curves. The result was the 2007 WHO growth reference for school-aged children and adolescents (de Onis et al., 2007). Note that the 2007 reference is not meant to serve as a normative standard of growth. There are differences in adolescent growth across populations and historically that are not adequately captured by the reference. Most importantly, differential timing of the pubertal growth spurt across individuals (and populations) can lead to faulty comparisons with the references in adolescence (Tanner & Davies, 1985). For instance, because children in historical populations experienced the pubertal growth spurt at older ages, they appear to fall behind the WHO reference when the modern children are experiencing their pubertal growth spurt and catch up at later ages as the historical children continue to grow.<sup>4</sup>

While the WHO growth standard/reference has become widely used around the world (de Onis et al., 2012), they are not without their critics. Some have argued that genetic differences between populations make the standard a poor proxy for malnutrition (Panagariya, 2013). However, while there are clear population differences in growth in adolescence, a recent meta-analysis that compared the height growth of children under 5 for healthy and well-off subgroups across a wide range of countries found that most had HAZ scores within  $\pm 0.5$  standard deviations of the WHO standard (Natale & Rajagopalan, 2014). Pacific Islander, Dutch and Finnish children were positive outliers, whereas Saudi and Indian children were negative outliers (outside the  $\pm 0.5$  sd range). While these results may suggest that genetic differences matter, as discussed in Section 5.1 below, there are many factors detrimental to health that would affect all people in a society and could explain why even well-off children would experience growth outside the modern norm. Despite these critiques, the WHO standard is largely trusted for gauging population-level malnutrition.

### 3.2 | Growth faltering and stunting

If we had longitudinal measures for individuals, we could track individual children's growth relative to the standard to get a sense of whether they were catching up to or falling behind healthy children. However, longitudinal measures of child growth are rare today and in history, so typically researchers analyze cross-sectional measures of children's heights by age. The assumption is that systematic deviations between the mean height of healthy children (the WHO 2006 standard) and the population being analyzed will show that on average children in the population are experiencing unhealthy growth. Figure 1c shows an example of this, comparing the growth of healthy children according to the WHO growth standard (black line) to children in the Northeastern region of Brazil measured in 1977 (maroon line) (Fundação Instituto Brasileiro de Geografia e Estatística, 1977).<sup>5</sup> The Brazilian children did not grow as quickly on average as healthy children (top graph), which means that they fell behind the modern standard in relative terms (bottom graph). In the bottom graph, the  $y$ -axis is expressed in height-for-age  $Z$ -scores (HAZ) where healthy children's mean growth is standardized at 0 across all ages and the mean height of Brazilian boys is expressed as the number of standard deviations away from the mean of the standard:

$$HAZ_a = \frac{\bar{X}_a^B - \bar{X}_a^S}{s_a^S} \quad (1)$$

where  $\bar{X}_a^B$  is the mean height of Brazilian boys at age  $a$ ,  $\bar{X}_a^S$  is the mean height of the standard at age  $a$ , and  $s_a^S$  is the standard deviation of the growth standard at age  $a$ . This pattern of falling behind in the first 2 years of life is called growth faltering, and growth faltering is common in populations that suffer from nutritional deficiencies or high burdens of chronic infection (Victora et al., 2010). It is also one of the reasons for the emphasis on health conditions in the first 1000 days (from conception to age two) in the nutrition and development economics literature.

Comparisons with a growth standard also allow us to assess the distribution of heights in a population. Figure 1d shows the expected height distribution of healthy 3.5-year-old boys according to the WHO 2006 standard in black. Note that even among healthy boys, there is substantial variation in height, highlighting the importance of genetic variability. However, in order to diagnose children with unhealthy growth, it is necessary to set a threshold for unhealthy growth. The stunting threshold has been set at two standard deviations below the mean of the healthy distribution, that is, a HAZ score of  $-2$  or lower. In a healthy population, we would expect 2.3% of children to have heights below this threshold, but in present-day and historical populations, we observe far higher percentages of children with heights below the threshold. For instance, the distribution of heights of 3.5-year-old Brazilian boys in the Northeast region in 1977 (Figure 1d) has a lower mean and greater dispersion than the healthy standard, and accordingly 56.5% of boys are shorter than the stunting threshold. Given that we would only expect 2.3% of healthy children to have heights below this threshold, a high stunting rate suggests that at least 54% of children are likely to be experiencing abnormal, unhealthy growth.

Finally, it is worth considering how child stunting and growth faltering relate to the characteristics of the growth pattern described above. Growth faltering occurs when children grow too slowly in the first 2 years of life and therefore is connected to the speed of development (Ohuma et al., 2021). Stunting is a product of growth faltering (and thus the speed of development) and also of size at birth since populations with low mean height initially will have higher levels of

stunting. However, the process of growth faltering and stunting also appears to reduce subsequent adult height as well.<sup>6</sup> Thus, child stunting is related to three characteristics of the growth pattern, which means that changes in child stunting reflect changes in the characteristics of the growth pattern for these populations. These connections mean that studies of historical changes in the growth pattern and studies on changes in child stunting rates are fundamentally analyzing the same phenomenon: how the growth pattern shifts from an unhealthy to more healthy form.

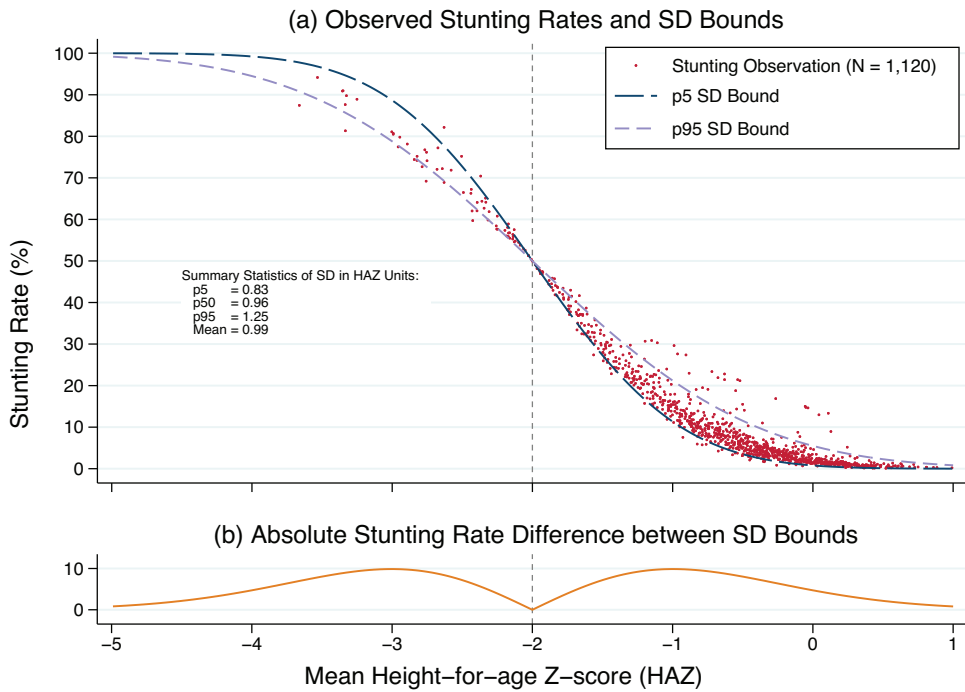
### 3.3 | Criticism of child stunting as an indicator

The widespread implementation of the WHO 2006 child growth standard has enabled child stunting to become the most important indicator of both individual- and population-level malnutrition. However, it is important to understand what stunting can and cannot reveal as an indicator. The stunting threshold itself is arbitrary: a child with a HAZ score of  $-1.99$  does not have markedly healthier growth than a stunted child with a HAZ score of  $-2.01$ . In addition, nonstunted children could have still experienced growth faltering for instance if they fell from a HAZ score of 1 at birth to  $-1$  at age 5. Thus, child stunting rates are a proxy for the extent of growth faltering in a population but should not be read as the share of children experiencing abnormal growth.

Another complication with the stunting rate is that the age composition of children in a survey can distort the stunting rate. Given the widespread pattern of growth faltering illustrated in Figure 1c, stunting increases with age until perhaps the age of 1 or 2 depending on the population. Thus, the share of children at each age is important (Aiyar & Cummins, 2021). These age differences also potentially matter when trying to assess the causes of child stunting. Alderman and Headey (2018) show that the socioeconomic correlates of stunting are stronger after age 2 when growth faltering has largely concluded. Clearly, researchers need to consider the age structure of their samples when computing the stunting rate and also when seeking to explain stunting (Aiyar & Cummins, 2021).

There are also hidden tensions in the idea that stunting is both an individual- and population-level measure of malnutrition. Those taking a more individualistic approach might argue that policymakers should find stunted children in their population and target them for interventions, that is, reduce inequality in height. On the other hand, those taking a population-based perspective might argue that stunting is indicative of population ill health and therefore interventions should be wide reaching, that is, increase the mean height of all children in the population.

Fortunately, we can test these perspectives with data. Figure 2a graphs 1120 stunting observations from the late 19th century to the present from the Worldwide Historical Stunting Dataset (Schneider et al., 2023). The  $x$ -axis plots the mean HAZ score for the population while the  $y$ -axis plots the stunting rate. There is a very clear and strong negative relationship between the mean HAZ score and the stunting rate as one might expect, but it is also interesting that the stunting observations fall within a relatively narrow band. This band is related to the effect of the dispersion in HAZ on the stunting rate because the range of the standard deviation of the HAZ distribution used to compute the stunting rate is rather narrow. The dashed lines in the figure use the 5th and 95th percentile values of the standard deviation to predict the stunting rate across the range of values of mean HAZ scores. These predictions assume that the HAZ distribution is normally distributed, but this is a very sound assumption given that height is almost always normally distributed. Figure 2b shows the absolute difference in the stunting rate between the two bounds at each level of mean HAZ. This captures the stunting change that would occur from shifting from one extreme of inequality to another.<sup>7</sup> The greatest reduction in stunting that



**FIGURE 2** Bounds in inequality in child stunting. *Sources:* Worldwide Historical Child Stunting Database presented in Schneider et al. (2023). [Colour figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com/doi/10.1111/joes.12901)]

could arise from reducing inequality would be 9.9 percentage points, which is small relative to the very large changes in stunting that occur with changes in mean HAZ. Thus, reductions in child stunting appear to be driven by changes in mean height rather than reductions in height inequality.

These findings based on historical data are reinforced by studies of more recent child stunting, analyzing large numbers of LMICs using Demographic and Health Survey (DHS) data. Roth et al. (2017) find that growth faltering is a population phenomenon with children at all levels of initial HAZ falling behind rather than stunting being concentrated in subpopulations that face the worst health conditions. Thus, the eradication of stunting seems to occur by changing the population distribution of height, not by targeting stunted children.

The criticisms presented in this section do not invalidate child stunting and the stunting rate as an indicator of malnutrition, but they do suggest that researchers need to be careful when interpreting child stunting and consider these limitations.

#### 4 | STYLIZED FACTS OF THE CHANGE IN THE GROWTH PATTERN

Having covered the basic characteristics of the growth pattern and discussed how researchers measure healthy and unhealthy growth, this section presents descriptive results about how the growth pattern has changed across the four main characteristics. It will also discuss evidence on how child stunting rates have changed over time reflecting multiple characteristics of the growth pattern.<sup>8</sup>



## 4.1 | Birth weight and length

Before describing change in birth size over time, it is important to note that there are substantial differences in mean birth weights and low birth weight (LBW) percentages<sup>9</sup> around the world with South Asia, Africa, and Southeast Asia having especially low mean birth weights and high LBW percentages (Blencowe et al., 2019; Kramer, 1987). The causes of these variations are usually attributed to differences in maternal nutritional status, exposure to infectious diseases such as HIV and malaria, and exposure to airborne pollution (Accrombessi et al., 2017; Amegah et al., 2014; Eisele et al., 2012). It is also possible that there are genetic differences in birth weight between populations, but inherent differences have been challenged by the INTERGROWTH-21st Group (Villar et al., 2014).

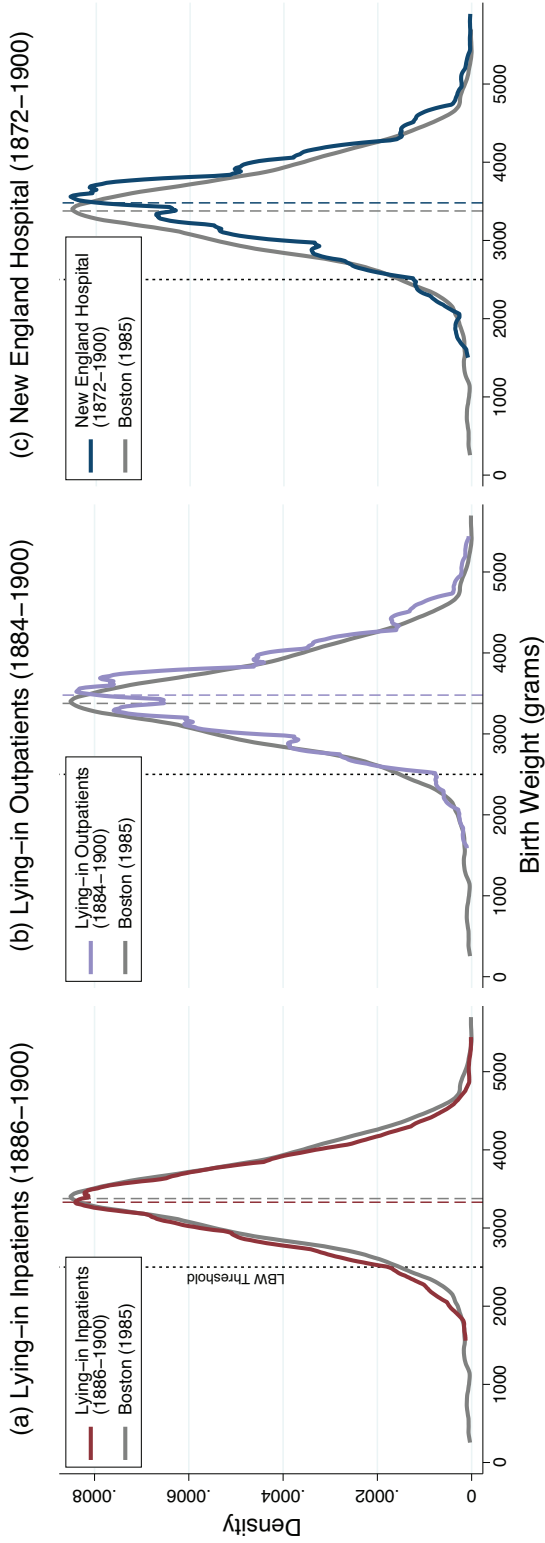
The variation in mean birth weights globally and the relationship between birth weight and maternal nutritional status might lead one to assume that birth weights have increased in Western countries as they experienced their health transitions from the mid-19th century onwards. However, this was not the case. While there have been some increases in birth weight since the late 19th century (Ward, 1993, 2016) and some in the 20th century as well (Ghosh et al., 2018; Kramer et al., 2002), Cole (2003), Costa (2015, 1998) and Schneider (2017b) have emphasized that these changes were small relative to the increases in adult stature over the same period and compared to the variation in mean birth weights across countries. On the whole, birth weights and their distributions in the late 19th century were remarkably similar to modern distributions in North America, Western Europe, Australia, and New Zealand (Galofré-Vilà & Harris, 2021; Roberts & Wood, 2014; Schneider, 2017b). Figure 3 presents the birth weight distributions in three Boston maternity hospitals at the end of the 19th century and compares them with white singleton births, the closest comparison group, in 1985. The distributions are very similar and the mean birth weights are even with or slightly above the 1985 means. The fact that these results are common across three institutions with different patient selection criteria and are also mirrored in historical comparisons from other cities and countries suggests that they are robust. Thus, an increase in birth weight and possibly birth length<sup>10</sup> was not a part of the historical change in the growth pattern in North America and Europe.

Outside the Western world, it is more difficult to find long run trends in birth weight. For Japan, mean birth weights appear to have increased by *c.* 250 g between the early 20th century and the 1970s, a significant increase four times larger than the increases in birth weight in Western countries cited above (Kato et al., 2021; Misawa, 1909). However, since the 1970s, mean birth weights in Japan have fallen by 200 g nearly returning to their early 20th century level (Kato et al., 2021).

Despite these declining trends in Japan, there are signs that birth weights have been increasing in recent years in regions where mean birth weight is low. Blencowe et al. (2019) show improvements in birth weight in South Asia, Southeast Asia, and Africa between 2000 and 2016, though the progress is relatively slow. Likewise, Headey et al. (2019) compare change in neonatal size across DHS surveys in a number of countries and show that there have been substantial increases in neonatal length in South Asia and East Africa in the past 20 years. Thus, the change in the growth pattern, with respect to birth weights, may be different in regions with low mean birth weights than in current HICs.

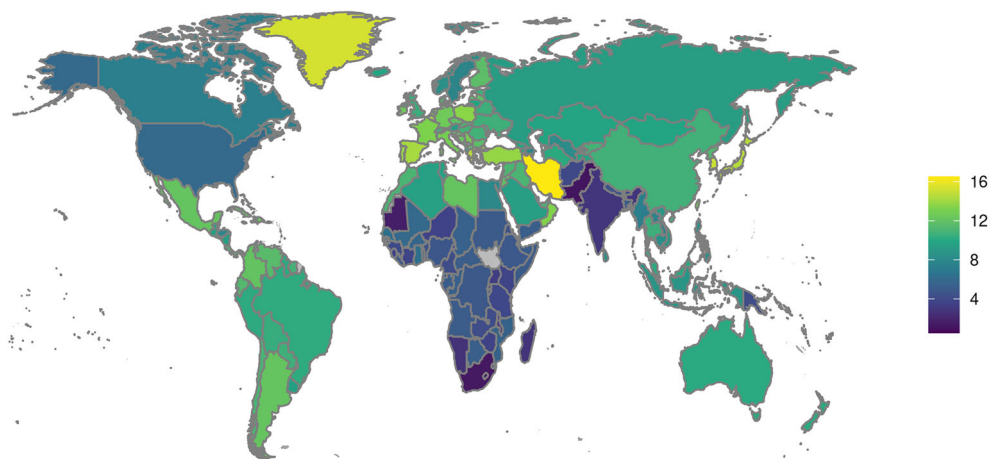
## 4.2 | Adult stature

The secular increase in adult stature since the mid-19th century is well-known and well-studied: see Steckel (1995), Steckel (2009), Hauspie et al. (1997), Komlos and Baten (2004), and Harris

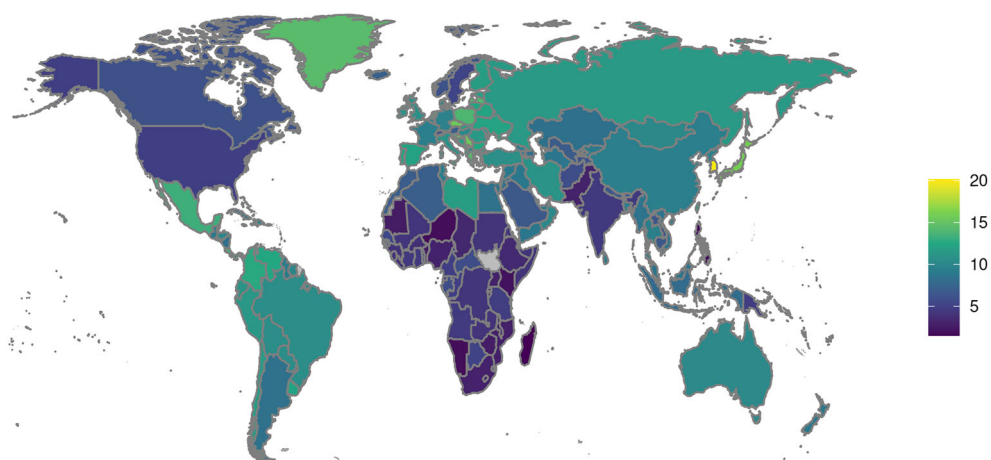


**FIGURE 3** Birth weight distributions in three 19th-century Boston hospitals compared with the birth weight distribution of Boston in 1985. Notes: Dashed lines mark the means of the late-19th century and 1985 distributions. The short dashed line is the low birth weight (LBW) threshold of 2500 g. Sources: Ward and Gagné (2012a), Ward and Gagné (2012b), Ward and Gagné (2012c), National Center for Health Statistics (1986). See Schneider (2017b) for a complete discussion. [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

## Change in mean male adult stature, 1896 to 1996



## Change in mean female adult stature, 1896 to 1996



**FIGURE 4** Increase in adult stature between the 1895 and 1995 birth cohorts.

Source: Bentham et al. (2016).

[Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

(2021) for earlier reviews. Bentham et al. (2016) recently estimated the secular increase in height between the 1895 and 1995 birth cohorts for most countries around the world.<sup>11</sup> Figure 4 presents their findings. They show that nearly all countries have experienced an increase in adult stature over the past 100 years, but the magnitude of the increase varies substantially across countries. The largest increases in stature for women occurred in South Korea (20.1 cm) and Japan (16.0 cm) and for men in Iran (16.5 cm) and South Korea (15.1 cm). Adult stature has increased by at least 5 cm in most regions with the exception of South Asia, Sub-Saharan Africa, and the USA and Canada.

While the Bentham et al. (2016) data paints an excellent picture of changes across a century, the careful work of economic historians allows us to understand when and how this increase in height occurred. Hatton and Bray (2010) present trends in male adult stature from military records

and survey data in 15 European countries from the 1850s to the 1970s. They find that Northern and Western European men experienced the most rapid gains in adult stature between the 1910s and 1950s, a period that included two world wars and the Great Depression. Southern European countries experienced the most rapid gains somewhat later between the 1950s and 1970s. Baten and Blum (2012) conducted a similar study collecting adult male stature for 156 countries around the world. They found that adult male stature was relatively stable at a low level in the 19th century with the biggest increases occurring in the 20th century. They also highlighted that men in the United States, Canada, New Zealand, and Australia were substantially taller than men in the rest of the world in the 19th century.

These patterns in the economic history literature are fascinating, but unfortunately, they are solely based on patterns of male height. Finding long-run series of heights for women is far more difficult since women did not serve in the military. However, scholars who have reconstructed trends in female adult stature have shown that they do not always neatly follow the trends in men's heights (Carson, 2011; de Beer, 2010; Koepke et al., 2018; Ridolfi, 2023). Likewise, different racial or ethnic groups often follow different trends requiring separate analysis (Carson, 2009; Inwood et al., 2015; Mpeta et al., 2018). Thus, reconstructing historical height trends for women and other under-represented groups continues to be a priority for future research.

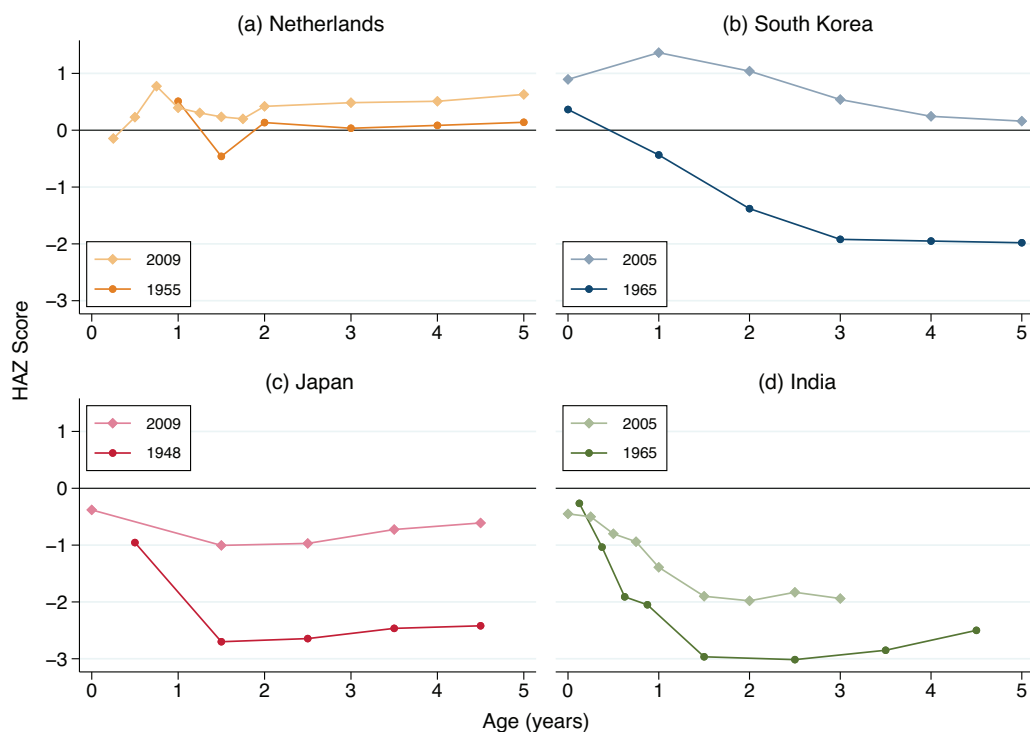
### 4.3 | Timing of the pubertal growth spurt and the speed of development/maturation

Change in the final two characteristics of the growth pattern are perhaps best discussed together since they are related. We can track these characteristics with indicators of growth in three different age periods. First, growth faltering captures slower than normal growth in the first 2 years of life. Second, growth in adolescence reveals the timing of the pubertal growth spurt as well as the velocity of growth in adolescence. Finally, studying growth in late adolescence and early adulthood reveals the age at which individuals cease growing, which again proxies the speed of development. I will deal with each of these in turn.

While drawing on the literature to help answer these questions, I have also collected child and adolescent growth curves for six countries, the Netherlands, Czech Republic, Costa Rica, India, South Korea, and Japan. These countries have two complete sets of height measurements for each sex one from the 1950s or 1960s and another from the early 2000s.<sup>12</sup> The sources of the data and some potential limitations are discussed in the notes of Figure 6. These data help to illustrate changes in the growth pattern across the second half of the 20th century.<sup>6b</sup>

#### 4.3.1 | Early life growth faltering

As presented above in Figure 1c, growth faltering occurs when children do not grow fast enough in early life and therefore fall behind relative to the WHO growth standard. Victora et al. (2010) show that in the early 2000s, growth faltering was common in many LMICs. Children were born with an average HAZ score of around  $-0.4$  and fell to a HAZ score of approximately  $-1.75$  by age 2. Figure 5 presents the change in growth faltering over time for four countries. Clearly, reductions in growth faltering have been a common occurrence across the 20th century. South Korea and Japan have seen large declines in growth faltering over time, and even India, which still has a high child stunting rate, has seen some reductions in growth faltering since the 1960s. Only the Netherlands



**FIGURE 5** Change in growth faltering across the late 20th century in the Netherlands, South Korea, Japan, and India. *Notes:* HAZ scores relative to the WHO 2006 Standard. *Sources:* Netherlands: 1955 data from de Wijn and de Haas (1960, pp. 26, 28) and 2009 data from Schönbeck et al. (2012), South Korea: Data from Kim et al. (2008), Japan: 1948 data are from the National Health and Nutrition Survey, Ministry of Health, Labour, and Welfare (1948) and 2009 data are from the Ministry of Health, Labour, and Welfare (2009), birth length in 2009 from Itabashi et al. (2014), India: 1965 data from Indian Council of Medical Research (1972) and 2005 data from Mamidi et al. (2011). [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

did not experience large changes in growth faltering, but this was because growth faltering had likely already been eliminated by the mid-20th century.<sup>13</sup> These reductions in growth faltering over time are confirmed by studies of the Gambia (Nabwera et al., 2017) and historical studies looking at changes in early child growth from the early 20th century to the present (Roberts & Warren, 2017; Weaver, 2010).

#### 4.3.2 | The pubertal growth spurt

In addition to growth faltering, the timing of the pubertal growth spurt and the velocity of growth during the pubertal growth spurt have changed dramatically over time. Figure 6 presents smoothed growth velocity curves for each country showing how the pattern of growth velocity above the age of 5 changed over time. The figure shows that the pubertal growth spurt is more pronounced and peaked for boys than girls, as expected. In all countries, the age at which peak growth velocity was reached in puberty has declined over time as suggested by earlier literature (Ali et al., 2000; Cole, 2003; Cole & Mori, 2017; Hauspie et al., 1997; Komlos, 1986; Komlos et al., 1992; Steckel, 1987). In some cases, the decline was very large: in South Korea, the age at peak

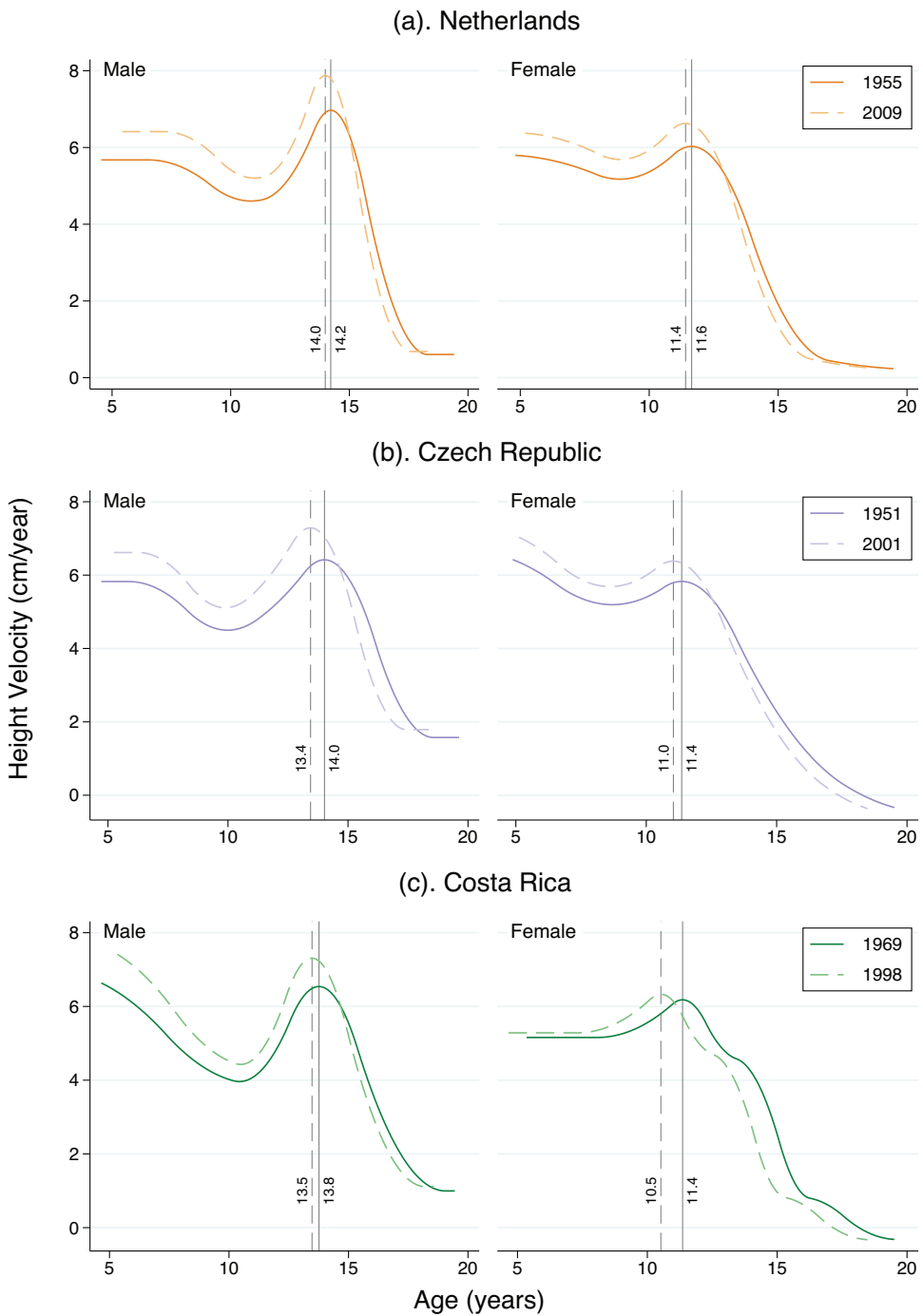
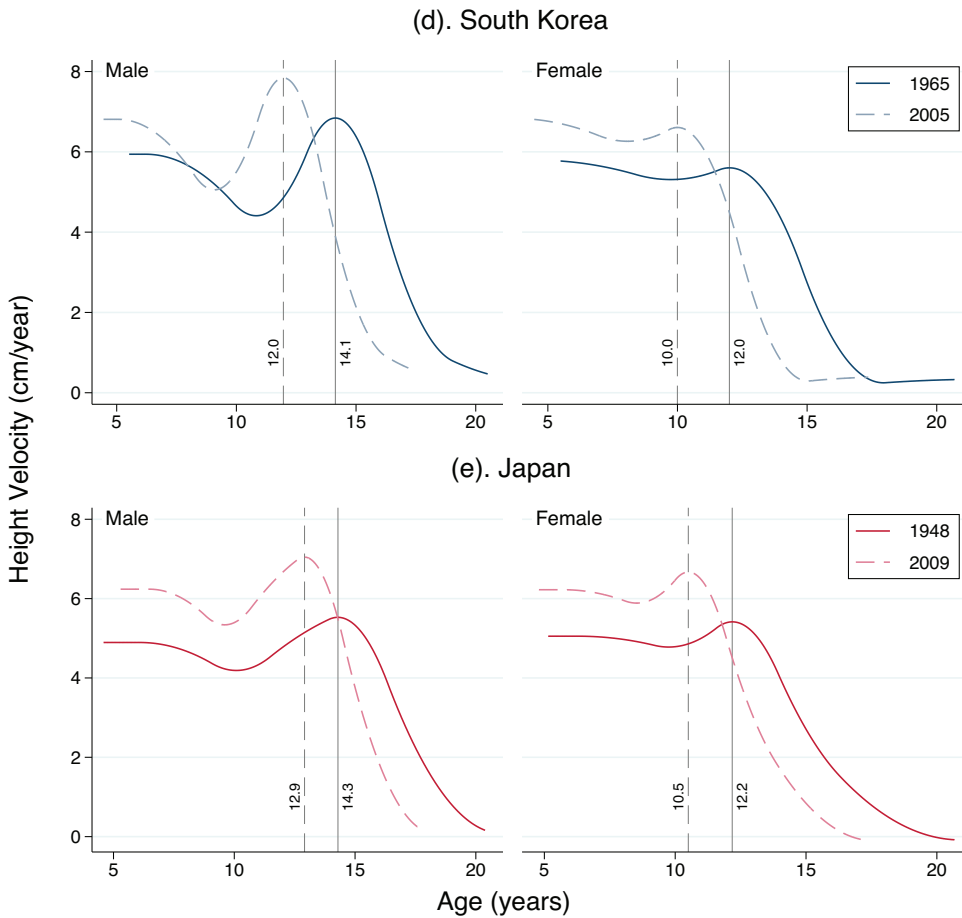


FIGURE 6a [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]



**FIGURE 6b** Change in growth velocity curves over time. *Notes:* Height-by-age data for each country and sex were smoothed using the SITAR growth model (Cole, 2019) and the predicted, smoothed growth curves from the model are plotted here. This method assumes that the data for each country and sex share a common growth curve that is changing in size, timing of the pubertal growth spurt, and speed of maturation. See Cole et al. (2010) for more information. The vertical lines display the age at peak pubertal growth velocity predicted for each curve. *Sources:* Netherlands: 1955 data from de Wijn and de Haas (1960, pp. 26, 28) and 2009 data from Schönbeck et al. (2012), Czech Republic: Data from Vignerová et al. (2006), Costa Rica: 1969 data from Villarejos et al. (1971) and 1998 data from Fernández-Ramírez and Moncada-Jiménez (2003), South Korea: Data from Kim et al. (2008), Japan: 1948 data from the National Health and Nutrition Survey, Ministry of Health, Labour, and Welfare (1948) and 2009 data from the School Health Statistics Survey, Ministry of Education (2009). [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

pubertal growth declined by 2 years, and it was only slightly smaller for children in Japan. On the other hand, the timing of the pubertal growth spurt decreased much more modestly in the Netherlands and Czechoslovakia.

However, there are two limitations with these kinds of comparisons. First, the secular change in the growth pattern had already begun by the 1950s in all of these countries, and second, nearly all studies on historical changes in the pubertal growth spurt have been based on comparing mean,

cross-sectional growth curves, which are prone to selection bias (Schneider, 2020b), rather than studying individual-level data (though see Aksglaede et al. 2008).

To overcome some of these biases, Gao and Schneider (2021) studied individual-level, longitudinal records of British boys born from the 1850s to the 1970s and found that for boys born before 1910, the mean height velocity curve did not show a strong pubertal growth spurt. A peaked pubertal growth spurt similar to those shown in Figure 6 only appeared after the 1910 birth cohort. Careful analysis of the data and simulations suggest that the absence of a strong pubertal growth spurt in the 19th century was only partially driven by greater dispersion in the timing of the pubertal growth spurt historically. Instead, the data suggest that the speed of maturation was substantially slower in the 19th century and that there was a rather sudden change for boys born from the 1910s onward.

While Gao and Schneider (2021) could only study boys, the increased speed of maturation is also captured by the secular decrease in the age at first menstruation (menarche) for girls since the 19th century: menarcheal age is strongly linked with the timing of the pubertal growth spurt (Cole, 2003; Trussell & Steckel, 1978). Tanner (1962, p. 153) shows that the mean age at menarche had declined from as high as 16 and 17 in the mid-19th century to 13 by the mid-20th century in Northern Europe. Brundtland and Walløe (1976) show that menarcheal age was fairly constant across the 19th century in Norway at around 16 for working class girls and 14 for upper class girls. This had declined to around 14 by 1926 and to 13 by 1970 (Brundtland et al., 1980). This suggests that girls may have also experienced an abrupt increase in the speed of maturation and a decline in the age of the pubertal growth spurt at the turn of the 20th century as well, mirroring Gao and Schneider (2021)'s findings. These historical shifts are also reflected in stalling or declining age at menarche in LMICs around the world (Leone & Brown, 2020).

### 4.3.3 | Age at cessation of growth

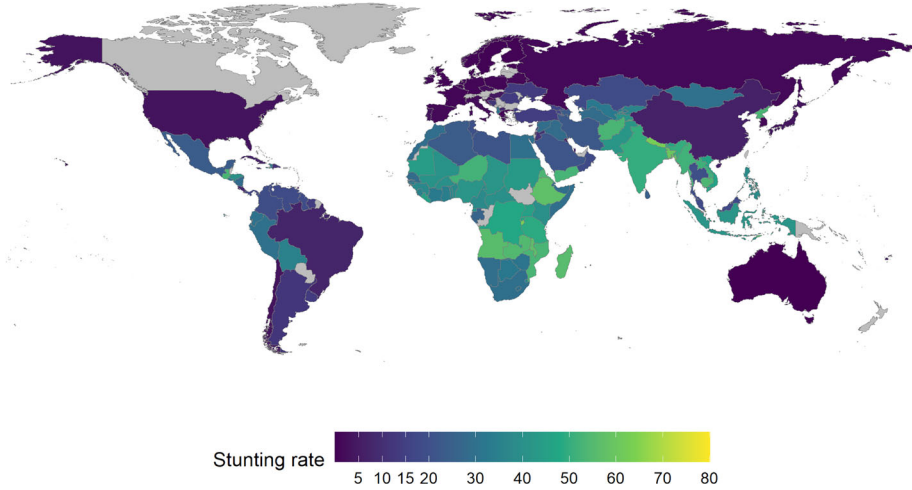
Finally, across the health transition, the speed of child development has also increased substantially so that children reach their final adult height at younger ages and grow more rapidly throughout the growth period. We can see this pattern in the velocity curves in Figure 6. In all cases, the age at which children reach zero velocities shifts to earlier ages across the second half of the 20th century. Again these findings concur with the wider literature, which shows that growth accelerated during the secular change in the growth pattern (Cole, 2003; Cole & Mori, 2017; Steckel, 1987; Schneider & Ogasawara, 2018; Schneider et al., 2021). Economic historians have also traced individuals' growth between late adolescence and adulthood showing that men in the 19th century continued growing until well into their 20s (A'Hearn et al., 2009; Beekink & Kok, 2017; Donald et al., 2022; Gauthier, 2022; Thompson et al., 2020), again suggesting that the age of cessation of growth has declined significantly over time.<sup>14</sup>

## 4.4 | Child stunting

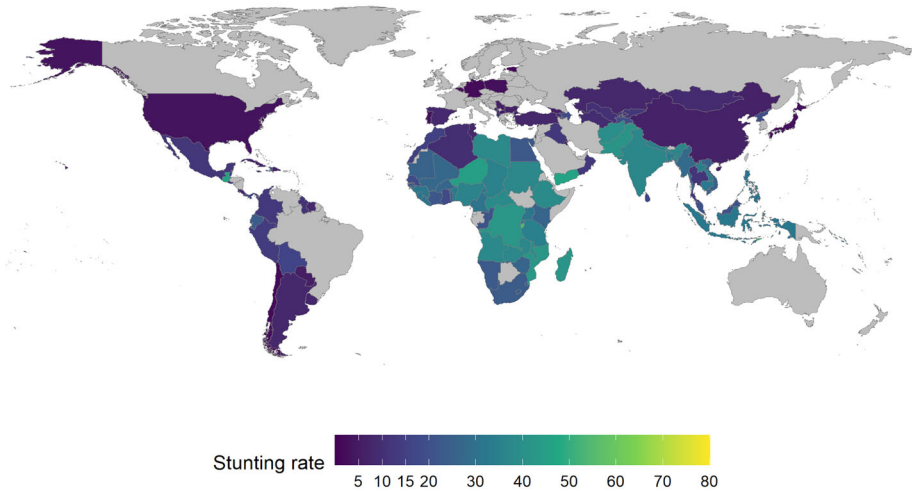
As mentioned in Section 3.2, child stunting is affected by size at birth and a slow speed of growth in early life that causes growth faltering, and it has an effect on final adult stature. Thus, with the changes described above, we would also expect to see decreases in child stunting over time even in current HICs. Child stunting has been declining around the world in the past 30 years (see Figure 7), although stunting rates remain high in sub-Saharan Africa, South Asia, and



## Stunting rate, 1990-1999



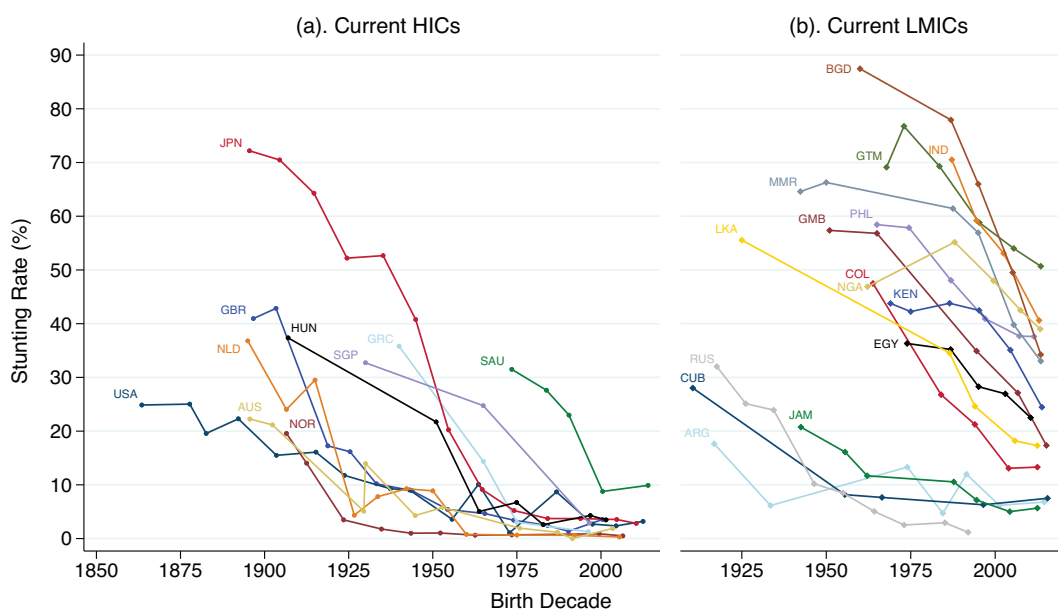
## Stunting rate, 2010-2020



**FIGURE 7** Stunting rates around the world in the past 30 years. *Sources:* UNICEF/WHO/World Bank (2023) supplemented with the Worldwide Historical Child Stunting Dataset (Schneider et al., 2023). [Colour figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com/terms-and-conditions)]

Southeast Asia (Black et al., 2013; Stevens et al., 2012). Stunting rates are generally higher in India than in sub-Saharan Africa: the so-called “Indian Enigma” (Ramalingaswami et al., 1996; Spears, 2020). There have been substantial improvements in the stunting rate in countries as varied as China, Peru, Bangladesh, Nepal, Tanzania, and Ethiopia, though some of these countries are still far from having stunting rates observed in HICs.

We know relatively less about historical developments in child stunting because historical sources on child growth are rare compared with sources of adult stature. However, a recent effort by a team of 40 anthropometric historians is seeking to reconstruct stunting rates around the world from the mid-19th century to the present (Schneider et al., 2023). They have conducted a



**FIGURE 8** Stunting rates for select countries across the 20th century. *Notes:* These data are preliminary based on ongoing research. The x-axes for the two graphs are different because of the different time coverage in each, but they have the same scale, so it is possible to compare the rates of decline across the two graphs. These countries are selected to display regional variation in stunting trends and because I have relative confidence in the accuracy of the historical trends. Historical stunting estimates are far more common for children over age 2, so children under age 2 are excluded from all stunting estimates in the graphs, including the UNICEF/WHO/World Bank Joint Malnutrition Estimates, for consistency. See Schneider et al. (2023) for more detail. *Sources:* UNICEF/WHO/World Bank (2023) supplemented with the Worldwide Historical Child Stunting Dataset (Schneider et al., 2023). [Colour figure can be viewed at wileyonlinelibrary.com]

meta-analysis of child growth studies in archival and published sources in order to reconstruct trends in child stunting. The project is ongoing, but Figure 8 presents some preliminary findings. Panel (a) shows trends in child stunting in countries that are HICs today whereas Panel (b) shows trends in countries that are LMICs today. However, note that all countries were substantially poorer in absolute terms at the end of the 19th century even if European and North American countries were relatively well off by the standards of the time. The historical patterns in child stunting are far more varied than one might expect. Some current HICs such as Norway, Australia, and the United States had relatively low child stunting rates going back to the late 19th century whereas others such as the UK, Netherlands, Singapore, and Greece had considerably higher stunting rates. The timing of stunting declines was earliest in the United States, Australia, and Norway, then spreading to Western Europe, and then to Eastern and Southern Europe a few decades later. Stunting rates in Japan at the end of the 19th century were as high as stunting rates in India and Guatemala in the 1960s, and most of the decline in stunting in Japan happened after the Second World War: similar patterns exist for South Korea and Taiwan. Thus, a decline in child stunting from levels comparable to most current LMICs is a feature of the secular change in the growth pattern in current HICs.

However, the historical trends across current LMICs are also varied and interesting. Again some LMICs had very low stunting rates since the first half of the 20th century. Perhaps Argentina is not

surprising given that it was relatively wealthy in the early 20th century, but Cuba and Jamaica were very poor and yet still had very low stunting rates.<sup>15</sup> Stunting rates in other LMICs varied between 36% in Egypt to 77% in Guatemala, and declined fairly rapidly since the 1960s. The Gambia has seen a particularly large decrease in stunting of 40 percentage points since the 1950s. These trends in sub-Saharan African and South Asian countries force scholars to consider the differences in stunting rates in the two regions in a dynamic framework rather than assuming that a single mechanism can explain the Indian Enigma today.

Overall, while it is not yet possible to estimate a global stunting rate going back to the 19th century, child stunting has decreased dramatically across the 20th century and clearly reflects the changes in the growth pattern described above. Likewise, the eradication of child stunting from extremely high levels (as in Japan) suggests that eliminating child stunting is possible, even for countries with very high stunting rates today.

## 5 | DETERMINANTS OF CHANGE IN THE GROWTH PATTERN

Having shown that the decline in child stunting and the secular change in the growth pattern are part of the same phenomenon, this section attempts to connect the work of economic historians on the historical secular change in the growth pattern<sup>16</sup> with the work of development researchers on the causes of child stunting in current LMICs.

To narrow the scope of the contemporary literature, I will focus primarily on the “Indian Enigma,” which seeks to understand why young children are shorter in India than in sub-Saharan Africa. The essential fact of the puzzle has been discussed for decades (Ramalingaswami et al., 1996), but the current debate dates from the 2010s. By this point, Indian stunting rates had been persistently high despite years of strong economic growth. This fact led to a public debate in 2013 in the pages of *Economic and Political Weekly*, a weekly Indian social science journal. Panagariya (2013) instigated the debate by questioning whether the WHO 2006 standards exaggerated undernutrition among Indian children by imposing an international standard that set stunting thresholds too high for Indian children who were a genetically shorter population. Panagariya’s arguments were heavily contested in the months following his initial publication (Coffey et al., 2013; Gupta et al., 2013; Jayachandran & Pande, 2013; Lodha et al., 2013), but the discussion provided renewed interest in explaining Indian undernutrition, and it provides an exceptionally useful way of surveying the contemporary literature in a manageable way.

Thus, the following sections deal with different factors that have been considered in explaining the Indian Enigma, and then relates these contemporary debates to research on the history of current HICs. The final section covers a few additional causes of child stunting outside the scope of the Indian Enigma.

### 5.1 | Genetic differences

One potential explanation for the Indian Enigma is that Indians simply have a lower genetic height potential than Africans, which makes Indians appear more malnourished at a given stunting threshold than they really are. To support this view, Panagariya (2013) highlighted that even the wealthiest Indian children with highly educated parents, modern homes with electricity and plumbing and good access to nutrition had a stunting rate of 15%. If the stunting thresholds were increased to close this 15% gap, then the Indian Enigma would disappear. Panagariya also noted

that even though the mean height of Japanese adults has increased substantially, Japanese people were still much shorter than the Dutch, highlighting differences in genetic height potential.

Panagariya's argument has been challenged on a number of fronts, many of which are described below, but there are a few important features to mention here. First, while the Japanese are still shorter than the Dutch, as Figure 8 shows, the Japanese stunting rate has fallen from a very high level to eliminate stunting, so there is no a priori reason to believe that Indian children could not reach the same level. Second, stunting is driven by a wide range of factors beyond nutrition, and some of these, such as water, sanitation and hygiene, and atmospheric pollution, affect all people in a society, not just the poor. Finally, in a study of ethnic Indians in the UK, Alacevich and Tarozzi (2017) show that the children of ethnic Indians have similar growth to British children in early childhood despite their parents being substantially shorter than British parents. This again suggests that genetic differences cannot explain the Indian Enigma.

## 5.2 | Selective mortality

Another early explanation for the Indian Enigma was offered by Deaton (2007) who suggested that the higher stunting rates in South Asia compared to sub-Saharan Africa could in part be explained by higher infant and child mortality rates in Africa, which removed short, weak children from the survivors being measured. Bozzoli et al. (2009) developed this model further showing that when pre-adult mortality was low, the scarring effect of childhood disease was more dominant, but at higher rates of pre-adult mortality, the selection effect could become strong enough to counterbalance the increasing scarring effect. These findings were intriguing and have prompted considerable debate in both the contemporary and historical literature. From the more recent perspective, Alderman et al. (2011) showed that selective mortality could only have a strong influence on anthropometric measures like the stunting rate if mortality was high and the counterfactual heights of those who died were far lower than those who survived. Harttgen et al. (2019) extended this analysis to explicitly compare South Asia and sub-Saharan Africa and showed that mortality selection could not explain the differences in the stunting rates between regions.

The idea of mortality selection is old, dating at least to the early 20th century when eugenicists like Karl Pearson were concerned that declining infant mortality might lead to a deterioration in population health by allowing the weak to survive (Hatton, 2011). However, the evidence for selection effects related to height in history is mixed. Gørgens et al. (2012) found that mortality was selective in response to the Great Leap Forward Famine (1959–61) in China with survivors being taller than the original cohorts. It is worth noting though that this was a major mortality shock with death rates in rural areas 2.6 times higher than before the famine. Thus, the scale of mortality may not be typical of year-to-year variations present in the 19th and 20th centuries. Economic historians have not been able to find evidence for a selection effect on height in the 19th or 20th centuries in contexts as diverse as 19th-century Belgium, 1930s Japan, or 19th and early 20th-century Sweden and Britain (Bailey et al., 2016; Depauw & Oxley, 2019; Hatton, 2011; Öberg, 2015b; Schneider & Ogasawara, 2018). The lack of a strong selection result may suggest that mortality was not as selective as Bozzoli et al. (2009)'s model would suggest. The leading killers of children in the 19th and 20th centuries tended to be acute infectious diseases and many of these diseases were not selective in relation to nutritional status (Bellagio Conferees, 1983; Schneider, 2023). Thus, it seems that selective mortality is unlikely to be an important explanation of changes in the growth pattern over time.

### 5.3 | Macro income (GDP growth)

One of the initial sources of confusion in the Indian Enigma was that India had experienced rapid economic growth, but that this economic growth had not translated into reductions in child stunting. While cross-sectional studies have found a strong negative association between GDP per capita and child stunting (Headey, 2013), studies that considered a panel of regions or countries have produced smaller estimates (Vollmer et al., 2014). These null effects were present in panel analyses of Indian states (Subramanyam et al., 2011) and across sub-Saharan African countries (Harttgen et al., 2013), suggesting that the Indian Enigma was not caused by differential relationships between GDP and stunting in India and Africa. Vollmer et al. (2014) argue that the small association between GDP per capita growth and reductions in child stunting means that direct health investments are necessary to reduce child stunting. In contrast, Alderman et al. (2014) argued that GDP per capita is measured with substantial error, GDP is not a particularly good measure of average welfare in an economy and that there was a much stronger relationship between household-level wealth and child stunting (Alderman et al., 2006). Thus, they emphasize that increasing income might be an important pathway for reducing child undernutrition in the future.

These discussions in the contemporary literature mirror earlier debates about the association between GDP per capita and adult stature in historical periods. One of the most surprising findings of the then new Anthropometric History literature in the 1990s was that mean adult stature declined during the take-off of modern economic growth in 19th-century Britain and the United States (Floud et al., 1990; Fogel, 1986; Komlos, 1987, 1998). This Industrial Growth Paradox or Antebellum Puzzle spawned studies seeking to replicate the result in different historical sources for Britain and the United States and in other historical contexts (Steckel, 2009). The findings were decidedly mixed with Industrial Growth Paradoxes confirmed in Britain and the United States (Cinnirella, 2008; Zimran, 2019) and rejected in others such as France, Italy, Japan, Sweden, and Tasmania (Federico, 2003; Inwood et al., 2015; Mosk, 1996; Sandberg & Steckel, 1997; Weir, 1997).<sup>17</sup> The results confirm the uncertain relationship between GDP per capita growth and adult stature found in current LMICs. The reasons for the opposite trends in adult stature and GDP during industrialization also highlight similar issues raised in the contemporary literature. In Britain, inequality was increasing during the first phase of the Industrial Revolution (Allen, 2009) and increased urbanization led to a deterioration in the disease environment (Szreter & Mooney, 1998), indicating that GDP was not a good measure of holistic living standards during the period. It was not until workers began to benefit from industrialization and cities began to invest in public health that adult stature began to increase. In addition, despite the ambiguous relationship at the macro level, there were strong individual-level socioeconomic gradients in adult height in historical periods (Jaadla et al., 2021; Öberg, 2014; Thompson & Ophem, 2023), which mirror the importance of household wealth for child stunting in LMICs today (Alderman et al., 2006).

When turning from the early periods of industrialization in the 19th century to the historical period of the secular increase in height in the 20th century, the results are more clear cut. Both Hatton (2014) and Baten and Blum (2014) found that GDP per capita had a fairly robust effect on rising adult statures, but it was modest compared to changes in the disease environment proxied by infant mortality. This changing relationship between GDP per capita and secular change in the growth pattern in history suggests that perhaps GDP will have a stronger influence on child stunting once current LMICs get past the early stages of development where the costs to health of economic growth such as urbanization, crowding, and pollution potentially outweigh the benefits.

## 5.4 | Nutrition

Trends in nutrition consumption in India are also an important puzzle: calorie and protein consumption per capita declined from 1985 to 2009 even as GDP growth was strong and there was modest decline in the stunting rate (Deaton & Dréze, 2009). This highlights that changing nutrition might have a complex relationship with reductions in child stunting. Looking at micro-level evidence, some studies have found important effects for nutrition. While breastfeeding does not seem to be related to child stunting (Giugliani et al., 2015), early complementary feeding may have been more important (Panjwani & Heidkamp, 2017). Looking at DHS data from a number of countries, Headey et al. (2018) found that children who ate animal-sourced foods had lower rates of child stunting, highlighting the importance of dietary diversity (Headey et al., 2012). Likewise, the introduction of free midday school meals in India in 2003 improved children's nutritional status and mitigated the effects of an earlier severe drought (Singh et al., 2014).

However, while the magnitude of the effect of the school meals program was large, the magnitude of consuming animal-sourced food was much smaller with a shift from eating no animal-sourced foods to three animal-sourced foods only reducing stunting by 6.1 percentage points (Headey et al., 2018). In a meta-analysis on the efficacy of nutritional interventions on child malnutrition, Bhutta et al. (2013) found that existing nutritional interventions could reduce child stunting by 20%, suggesting that interventions in other areas were crucial for eliminating stunting. This somewhat pessimistic finding for nutritional interventions is shared by studies that could precisely identify the relative role of nutrition and disease, which found that disease was far more important than nutrition (De Cao, 2015).

Historical research on nutrition and the growth pattern has emphasized the importance of total calories available to the population and the quality of the diet for growth. Floud et al. (2011) and Meredith and Oxley (2014) argue that rising adult stature in England at the end of the 18th century was driven by an expansion of total calories available per capita, though both their trends in stature (Cinnirella, 2008) and in calorie availability have been challenged (Schneider, 2013). Other researchers have focussed more closely on the quality of the diet and particularly on the availability of milk and other animal products. Studying a global country-level panel of adult male stature across the 19th and 20th centuries, Baten and Blum (2014) found that proximity to and density of cattle was associated with higher statures. These findings have also been replicated with 19th-century regional data for England and Wales, Bavaria, Prussia, and France (Baten, 2009; Horrell & Oxley, 2012).

However, the importance of nutrition in the historical secular change in the growth pattern is less clear. While increases in animal protein in Japan through the school meal program following the Second World War seem to have accelerated reductions in child stunting (Schneider et al., 2021; Takahashi, 1984), macro- and micro-level nutritional evidence suggests that Britain had reached adequate levels of macronutrients by the early 20th century even if there were shortages in some key micronutrients (Floud et al., 2011; Gazeley & Newell, 2015). Although nutrition continued to improve in the early 20th century (Gazeley et al., 2022), it seems unlikely that the rapid increases in height during the interwar period nor the sudden change in the growth pattern in Britain beginning with the 1910 birth cohort were driven by radical improvements in nutrition (Hatton & Bray, 2010; Gao and Schneider, 2021). In addition, breastfeeding did not have lasting effects on the growth pattern at the turn of the 20th century (Arthi & Schneider, 2021).

Overall, the evidence for the importance of nutrition is somewhat mixed. While current and historical evidence suggests that animal-sourced foods can affect population height, it is not clear that changes in nutrition have been fundamental drivers in the secular change in the growth pattern.

## 5.5 | Water, sanitation, and hygiene (WASH)

If the evidence for nutrition is mixed, a leading contender to explain the Indian Enigma is differences in water, sanitation, and hygiene (WASH) between India and sub-Saharan Africa. WASH can affect child stunting through two key pathways (Spears, 2020). First, poor WASH increases the prevalence of diarrhoeal diseases, which prevents children from absorbing all of the nutrients they consume. Chronic infections may also lead to “environmental enteric dysfunction,” an inflammation of the small intestine that further hinders the absorption of nutrients and makes children more susceptible to other infections (Humphrey, 2009). In addition, poor WASH may increase the prevalence of parasitic worms such as hookworm, which also sap children of nutrients and cause child stunting (Stephenson et al., 1993). Second, poor WASH may have cumulative or immediate effects on the nutritional status of mothers and therefore affect the birth size of their children. WASH conditions are particularly poor in India relative to other South Asian countries and sub-Saharan African countries because of high rates of open defecation in India (Spears et al., 2013). Spears (2020) argues that nearly all of the gap in child stunting between India and sub-Saharan Africa can be explained by open defecation.

While these arguments about the importance of improving WASH conditions are persuasive, designing effective WASH interventions has been more difficult (Pickering et al., 2019). This is in part because WASH interventions require sustained individual-, household-, and community-level behavior change (Dreibelbis et al., 2013). Additionally, public improvements in water and sanitation infrastructure may be substitutes to individual- or household-level WASH behavior rather than complements as one would hope. Bennett (2012) demonstrates this in the Philippines, showing that access to piped water did not affect household’s sanitation practices, but at the community level, communities with greater access to piped water had higher rates of open defecation. In this case, the public good, piped water, led individuals to underinvest in sanitation, leading to an increase in open defecation at the community level. Thus, implementing WASH interventions is much more difficult than it might first appear.

Economic historians have also studied the effects of installing sewerage systems and treating water through filtration or chlorination in historical settings. Although early studies found that these improvements had strong effects on infant and child mortality (Cutler & Miller, 2005), more recent studies have found heterogeneous effects from large effects in Boston, England and Wales, and Germany (Alsan & Goldin, 2019; Chapman, 2018; Gallardo-Albarrán, 2020) to much smaller effects for a panel of US cities (Anderson et al., 2022). While these interventions helped to reduce typhoid mortality, it is also clear that they did not have as strong an effect on infant diarrhoeal mortality, which may have been spread by flies rather than water (Anderson et al., 2020; Davenport et al., 2019). Likewise, several studies found that clean water alone was not enough to reduce mortality since modern sewerage was needed in order to remove waste water from homes (Alsan & Goldin, 2019; Gallardo-Albarrán, 2020; Kappner, 2022). These articles point toward similar externalities as were discussed in the Philippines above.<sup>18</sup>

To date, no historical studies have tried to link anthropometric data with the precise timing of water and sewerage upgrades that have been used to test the effects of sanitation on mortality. Instead, economic historians have used infant mortality rates as a proxy for child morbidity since the diseases that killed infants such as diarrhoeal and respiratory diseases were important sources of chronic morbidity for young children. A large number of studies have shown that infant mortality affected heights of children and adults in history, including studies of country-level panels (Baten & Blum, 2014; Hatton, 2014), city-level panels in the UK (Hatton, 2011), and a registration district-level cross-section in the UK (Bailey et al., 2016). Quanjer (2023) shows that child

mortality was more strongly predictive of mean municipal conscription height in the historical Netherlands than infant mortality but again confirms the importance of childhood morbidity for height. However, these effects were not universal. Using a brother fixed-effects empirical strategy, Öberg (2015b) shows high levels of infant mortality in early life had no effect on adult stature in Sweden. Likewise, Schneider and Ogasawara (2018) analyze prefecture-level data in interwar Japan and find no effect of infant mortality in early life on subsequent child height, although they do find an instantaneous effect of infant mortality on child height in late childhood and early adolescence.

While this historical literature shows that in many contexts, child morbidity may have affected changes in the growth pattern, there are several issues that make it difficult to directly compare with the development literature above. First, infant mortality captures a much wider range of diseases than the fecal-oral diseases that were described in the current literature on India above. Diarrhoeal deaths made up approximately 20% of infant deaths in urban England in 1889 and in Japan in 1921 (Schneider & Ogasawara, 2018). Historical WASH interventions also tended to have relatively small effects on diarrhoeal mortality in infancy (Anderson et al., 2020). Thus, it is not clear that WASH interventions per se would explain as much change in the growth pattern as is suggested when proxying WASH with infant mortality. Second, none of the studies of infant mortality on the growth pattern are cleanly identified. While some studies harness annual variation in infant mortality, others rely on decadal average infant mortality, which is less precise and may be correlated with omitted confounders. Finally, none of the countries studied historically had high prevalence of open defecation in the way that is common in India at present. While no precise historical study in relation to child growth exists, Riley (2005) shows that Rockefeller Foundation interventions that taught Jamaicans to build improved latrines in the 1920s helped to reduce open defecation and led to a decrease in diarrhoeal mortality rates. Later, the first reliable child growth studies in the 1950s showed a stunting rate of around 20%. Thus, there is preliminary historical evidence to suggest that reductions in open defecation could dramatically decrease child stunting, but more historical research is needed on the shift from open defecation to the use of improved latrines.

## 5.6 | Household allocation of resources and gender bias

Another explanation for the Indian Enigma relates to the allocation of household resources among children. Jayachandran and Pande (2017) argue that half of the stunting gap between Indian and sub-Saharan African children can be explained by Indian families' preference for first-born boys over other children. Comparing children born in Africa and India, they show that the birth order gradient of child height is much steeper in India than it is in Africa, that is, higher birth order children are shorter than first-born children. They link this pattern to Hindu preferences for having a male heir to inherit property and look after the parents in old age. They perform a number of empirical exercises to demonstrate this preference: there are birth order gradients in prenatal and postnatal health inputs; the birth order gradient is steeper among Hindu children than among Muslim children in India; the gradient is less steep in Indian regions with lower son preference even though average health inputs are not higher in those regions; and there is a birth order gradient only among sons as well. Overall, their article is a powerful demonstration of the potential for gender bias and misallocation of household resources to affect child health at the population level.

Jayachandran and Pande (2017)'s findings are intriguing, but they have been challenged on a number of fronts. First, Spears et al. (2022) argue that because the DHS only collects anthropometric measures for children under age 5, there is informative censoring in the birth order effects.<sup>19</sup>



Because the fertility decline is more or less over in India, women with high fertility, and rapid enough birth spacing to have two children under 5, are negatively selected on socioeconomic status and health. However, in sub-Saharan Africa where the fertility decline is still ongoing, women with high fertility are not negatively selected. Spears et al. (2022) argue that this informative censoring explains the steeper gradient in HAZ by birth order in India, not differential resource allocation toward first-born boys. In addition, a number of studies have argued that differences in child height are mainly determined by maternal health characteristics (Aiyar & Cummins, 2021; Coffey, 2015; Grafenstein et al., 2023): see Section 5.7 for more details.

Evidence of gender bias in the allocation of household resources in the past in current HICs is mixed with household budget studies showing no or ambiguous effects (Horrell & Oxley, 2013; Logan, 2022; Saaritsa, 2017) but with estimations of sex ratios revealing “missing girls” in some European regions (Marco-Gracia & Tapia, 2021; Szoltysek et al., 2022). Only two historical studies have analyzed gender differences in child growth. Schneider (2016) finds that there were no significant differences in catch-up growth between impoverished boys and girls that entered institutional care at the turn of the 20th century in Britain and America, suggesting that even among very poor households, there was no discrimination in the allocation household resources. Horrell and Oxley (2016) on the other hand find that English girls in factory work in 1837 were shorter than boys. Other studies have analyzed trends in male and female adult stature (Carson, 2011; Koepke et al., 2018), but the fact that selection into the sources of adult stature differed by sex makes it difficult to precisely ascribe gender-specific trends to changing health conditions. Thus, gender discrimination in the allocation of household resources does not appear to have been a major factor in explaining the secular change in the growth pattern in historical HICs, though more research on girls’ growth is needed.

Economic historians have also sought to understand how fertility decline would have affected the growth pattern with a number of studies showing that family size is negatively correlated with height (Bailey et al., 2016; Hatton, 2017; Öberg, 2015a, 2017; Qunjer & Kok, 2019; Ramon-Muñoz & Ramon-Muñoz, 2017; Stradford et al., 2017). Studying a cross-section of households in 1930s Britain, Hatton and Martin (2010b) instrument for family size with final twin births in order to eliminate confounding bias and find a negative relationship between family size and height. This effect remained when controlling for household income per capita, suggesting that household crowding may be another important mechanism in slowing growth. In a related paper based on similar estimates, they show that approximately a quarter of the increase in adult stature in Britain from 1906 to 1938 could be explained by reductions in fertility (Hatton & Martin, 2010a). Thus, the fertility decline may have influenced the secular change in the growth pattern both by reducing the consumption requirements of the household and by limiting crowding within households. However, this does not translate to the Indian Enigma since fertility was higher in Africa than in India.

This current debate on household resource allocation, fertility, and gender also raises questions about the ways that households respond to health interventions and shocks with respect to children. Households can respond to a health shock to one child by compensating for the shock, giving that child extra resources, or by reinforcing the shock, giving the child fewer resources perhaps because they have a lower chance of survival. Almond and Mazumder (2013) review around 20 articles and show that both compensating and reinforcing investments may be possible in the same society at the same time, making these responses dependent on the type of shock or intervention and on the outcome being measured. Reinforcing investments are found in high-income settings as well (Datar et al., 2010), so the balance of reinforcing or compensating investments does not seem to be related to the level of development.

Economic historians have also explored these issues. Parman (2015) compares individuals with siblings in utero during the 1918 flu pandemic with individuals with siblings born in other years to see whether parents compensated for or reinforced the poor initial health endowment of children affected by the pandemic in early life. He finds that parents reinforced investment in human capital, but there was no effect on adult stature. Likewise, Ogasawara (2017, 2022) shows that Japanese girls' growth was more strongly affected by being exposed to the Spanish flu pandemic or to the 1923 Kantō earthquake in utero than boys' growth. While he believes that other mechanisms could be part of this gendered effect, he argues that the results are consistent with parents reinforcing health endowments for girls and compensating them for boys. Thus, there is suggestive historical evidence for the types of resource allocation that Jayachandran and Pande (2017) found.

## 5.7 | Maternal health

Another explanation provided for the Indian Enigma is that maternal health in India is far poorer than in sub-Saharan Africa. A wide range of biological evidence and theory has suggested that maternal health capital<sup>20</sup> and health conditions in utero can have strong impacts on the growth pattern (Gluckman & Hanson, 2006b, 2006a; Wells, 2017) and later life disease (Almond & Currie, 2011).<sup>21</sup> Coffey (2015) analyzes DHS data and reweights the surveys to estimate the BMI and share underweight for Indian and African women at risk of pregnancy. She shows that women in India are much more likely to have BMIs below 18.5, the threshold for underweight, than women in Africa. In India, the mean BMI of women was 20.5 and reweighting for age and other characteristics 42.2% of women at risk of pregnancy were underweight. The corresponding figures for women in sub-Saharan Africa were 21.9 and 16.5%, respectively. This gap in maternal health may explain differences in birth weights and lengths between the two regions. In fact, Aiyar and Cummins (2021) show that the HAZ gap between India and sub-Saharan Africa is present at birth and remains largely the same across infancy and early childhood. Thus, they argue that differences in stunting were related to prenatal health investments and maternal health rather than resource allocation after children were born as Jayachandran and Pande (2017) had argued. These findings are consistent with recent longitudinal analysis of growth faltering (Mertens et al., 2023) and earlier research that showed that maternal health capital is mediated by environmental conditions around the birth (Bhalotra & Rawlings, 2011, 2013; Venkataramani, 2011).

Trying to understand maternal health capital in history is more challenging because of the paucity of sources on women's health. As shown in Section 4, women's adult stature has increased around the world, but maternal capital may be better captured as Coffey (2015) suggested through BMI and the share underweight. Historical studies of women incarcerated in local jails or penitentiaries provide evidence about female BMIs for a few countries. Carson (2018) shows that American White and Black women in penitentiaries in the late 19th and early 20th centuries had relatively high BMIs despite being shorter in stature: mean BMIs and percentage underweight for women aged 15–42 were 23.4 and 4.5% and 23.1 and 7.6% for Black and White women, respectively. Mean BMI for women aged 16–44 in England and Scotland in 1848–1882 was also high at approximately 22 and 23.9, respectively (Horrell et al., 2007; Meredith & Oxley, 2015). All of these BMI measurements are representative of working-class women who likely had lower health status than the general population, making them potentially underestimates of population values. These historical figures are far higher than what we observe for women in both India and sub-Saharan Africa today and can perhaps explain why birth weights were so high in these countries in the past. Although tentative given the limited historical sources, these data hint that the state

of maternal health during the health transition may have been very different in North America and Western Europe than in the rest of the world. They may also explain why stunting rates in these countries rarely exceeded 45%.

## 5.8 | Atmospheric pollution

Outside of China, India has some of the highest levels of atmospheric pollution in the world with concentrations far higher than in most sub-Saharan African settings (Apte et al., 2015). There is growing awareness that atmospheric pollution may affect child stunting (Sinharoy et al., 2020). This may be through two pathways: 1) high levels of pollution exposure can lead to intrauterine growth restriction and therefore reduce birth weights, and 2) chronic exposure to pollution can also lead to inflammation in the lungs, which may increase children's susceptibility to respiratory diseases and cause child stunting. Pollution can either come from ambient sources such as power plants, factories, and cars or from household sources, mainly from cooking with solid biomass fuels such as coal or wood. While modern evidence for the effect of pollution on birth outcomes is reasonably robust (Li et al., 2017), few studies have explored the links to child stunting directly. Spears et al. (2019) find that relative to children with low PM<sub>2.5</sub> pollution exposure, Indian children exposed to the highest levels of pollution in their birth month have 0.06 lower HAZ scores at age 5. They find that the strongest effects are in the 3 months before and after birth, while exposure later in infancy did not influence stunting. Although the 0.06 HAZ score effect is relatively small, it could account for half of the difference in stunting between India and Africa.

HICs also had extremely high levels of pollution during their industrialization in the 19th and early 20th centuries. Recent papers have shown that atmospheric pollution contributed substantially to infant and respiratory disease mortality in England, Germany, and the United States (Beach & Hanlon, 2017; Clay et al., 2018, 2022; Franke, 2022; Hanlon, 2022). However, there has only been one historical paper to link atmospheric pollution to the growth pattern. Bailey et al. (2018) study a cross-section of WWI recruits born in the 1890s and show that men born in registration districts with the largest numbers of workers in coal-intensive industries were 2.5 cm shorter at adulthood than men born in the least coal-intensive districts. A fundamental problem with estimating ecological effects of pollution on health is that people sort themselves into areas based on observable amenities (Heblich et al., 2021). This sorting induces pretreatment collider bias meaning that the causal effect of pollution on health is biased by the association between individuals' socioeconomic status and the other observable amenities in the area: that is, rich people choose not to live in polluted areas (Schneider, 2020a). To overcome this threat to inference, following Beach and Hanlon (2017), Bailey et al. (2018) show that pollution in the prevailing upwind direction from a district affected height in a district, but pollution downwind did not. These findings on pollution are intriguing, but more research is needed to definitively establish the importance of pollution for the growth pattern.

## 5.9 | Other factors

In addition to the areas mentioned above as part of the Indian Enigma, there are three other drivers of child stunting that have historical or contemporary importance and are worth discussing briefly. First, there is a large literature showing that war can produce substantial shocks to child health. Akresh and co-authors have shown that war increased child stunting dramatically

during civil wars or other conflicts in Nigeria, Burundi, Eritrea-Ethiopia, and Rwanda (Akresh et al., 2011; Akresh, Lucchetti, et al., 2012; Akresh, Bhalotra, et al., 2012; Bundervoet et al., 2009). However, the historical effects of war in current HICs are more mixed. For instance, the First World War had detrimental consequences for growth in Germany and Poland (Blum, 2013; Cox, 2015; Kopczyński & Rodak, 2021) but did not change the trajectory of the growth pattern in Britain (Harris, 1993). Likewise, the effects of the Second World War varied dramatically around the world. Child heights decreased in Belgium, Norway, Italy, Germany, occupied Russia, and Japan (Brainerd, 2010; Brundtland & Walløe, 1976; Daniele & Ghezzi, 2019; Ellis, 1945; Howe & Schiller, 1952; Schneider et al., 2021), but Finland, Sweden, and Denmark saw relatively little change (Angell-Andersen et al., 2004). Heights in Britain improved because rationing improved the quality of the diet for children (Harris, 1995; Magee, 1946). This historical research highlights the diversity of consequences that can arise from war depending on the historical context and cautions against simple assumptions about how war will affect the growth pattern.

A second driver of child stunting is market access. Researchers studying current LMICs tend to emphasize the positive effect of markets on child stunting because individuals with greater market access tend to have greater dietary diversity (Abay & Hirvonen, 2017; Nandi et al., 2021). Historical researchers have also found that market access improved health through nutritional pathways: Burgess and Donaldson (2010) show that access to railways in India reduced the prevalence of famine. However, other historical researchers have emphasized some downsides to market access. Zimran (2020) links changes in market access directly to the growth pattern by studying how the expansion of the railway network in the 19th century United States affected adult heights of men. He finds that increases in market access decreased heights, mainly because places with greater market access experienced more rapid population growth, had higher population densities and thus had more virulent disease environments. The different effects of market access and mechanisms through which market access affected the growth pattern in the past and present highlight how contingent this effect may be on the specific context. The negative consequences of greater exposure to infectious disease may have been greater in the past before widespread immunization campaigns, but the consequences of greater population density may explain why some studies of current LMICs have not found strong effects of market access on child stunting (Stifel & Minten, 2017).

Finally, recent research has highlighted the importance of maternal mental health in driving child stunting outcomes in LMICs. A number of studies have found that women experiencing high levels of maternal depressive symptoms are more likely to have stunted children even when controlling for a wide array of socioeconomic characteristics (Nguyen et al., 2018). A recent meta-analysis highlights several mechanisms to explain the association: mothers experiencing depression may be less likely to engage in developmentally appropriate feeding behavior, to seek medical care when necessary or to maintain high levels of sanitation and hygiene in the household. Consequently, children of mothers experiencing depressive symptoms had higher rates of diarrhoeal and respiratory infections (Asare et al., 2022). Although postpartum depression occurs in both high- and low-income settings today, it no longer has consequences for child stunting in high-income settings. This suggests that poverty and poor health conditions are likely preconditions for maternal depression to matter for child growth. Extending these findings into a historical context is exceedingly difficult however. Postpartum depression is and was stigmatized, meaning that there are likely no historical records that would provide insights into both postpartum depression and child growth. In addition, there have been major changes in psychiatric diagnosis generally over time, especially since the introduction of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM III) in the early 1980s (Mayes & Horwitz, 2005). Postpartum depression in

particular has been subject to large changes in classification over time (Godderis, 2013), making it extremely difficult to construct prevalence trends for postpartum depression. Maternal mental health is likely to be another source of individual-level heterogeneity in historical child growth outcomes but may be unobservable in a historical setting.

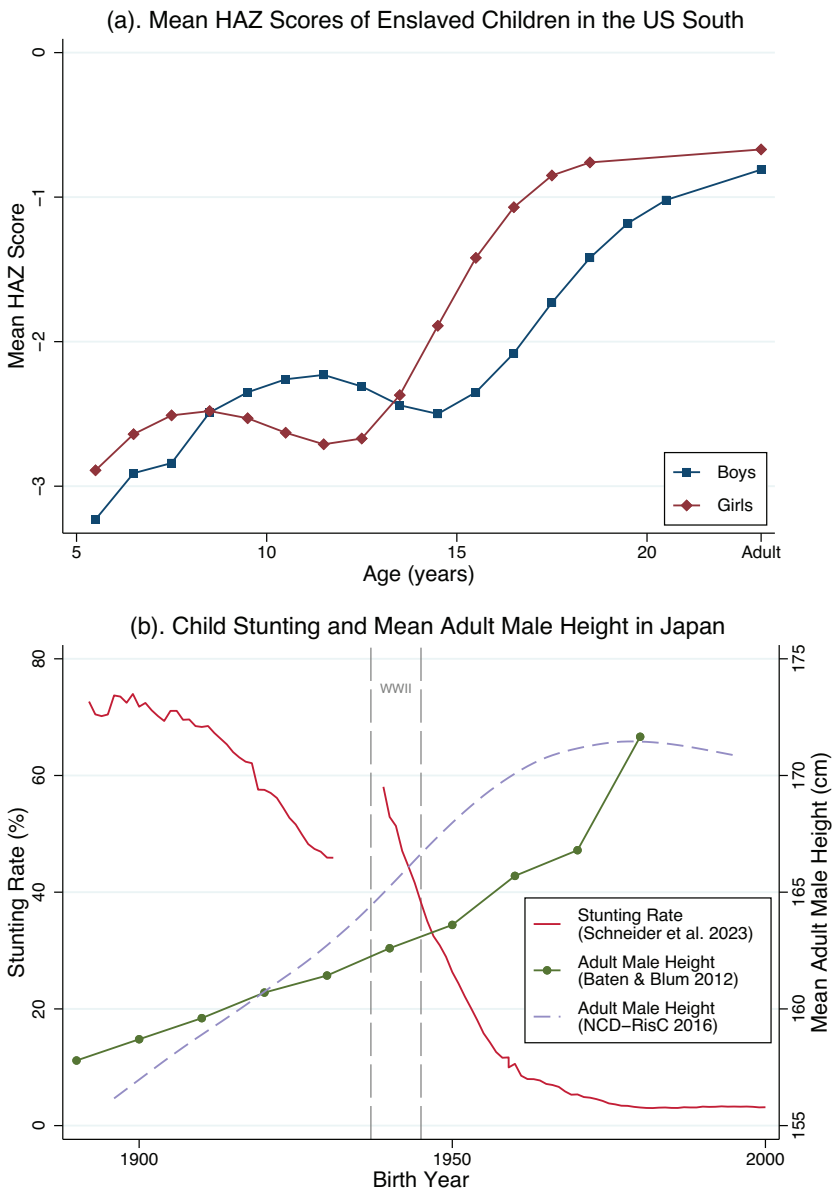
To conclude this section on determinants of change in the growth pattern, it is important to note that the factors discussed above may interact with one another such that some are preconditions necessary for others to become salient. For instance, poverty and poor general health conditions may be necessary for maternal mental health to have an effect on child growth. Likewise, household resource allocation may only matter for child growth when household resources are scarce. This highlights the importance of context, both historical and country-specific, in studying changes in the growth pattern. Differences in these interactions across contexts likely account for the heterogeneity in the results described above.

## 6 | CATCH-UP GROWTH: CRITICAL WINDOWS AND RECOVERY FROM HEALTH SHOCKS

The previous section has highlighted many of the factors that have influenced change in the growth pattern over time, but there is one final element that makes it difficult to precisely determine the factors influencing the growth pattern. Children's growth is strongly responsive to health conditions. While I have discussed the detrimental effects of poor conditions in early childhood that lead to growth faltering, the opposite is also possible. Children who have already fallen behind their growth potential are able to experience catch-up growth, faster than normal growth, when health conditions improve. The magnitude of this catch-up growth can be rather large and therefore mitigate the effect of an early life health shock if conditions later improved.

Figure 9 presents two historical examples of catch-up growth. Panel (a) shows the mean HAZ score for enslaved children in the US South before the Civil War. Enslaved children experienced very large levels of catch-up growth from HAZ scores around  $-3$  at age 5.5 to  $-0.75$  in adulthood. Enslaved children experienced growth faltering in early life because of terrible nutrition and sanitary environments and experienced catch-up growth later because once they entered the labor force on the plantation, they were given more plentiful rations and shoes that could protect them from hookworm (Coelho & McGuire, 2000; Schneider, 2017a; Steckel, 1986). Second, Panel (b) shows trends in the stunting rate and two series of mean male adult stature across the 20th century in Japan. Looking at the stunting rate, it is clear that the food shortages and collapse of the health system during the Second World War led to a substantial increase in the stunting rate for Japanese children. Schneider et al. (2021) also show that the war had a strong effect on the growth pattern, reducing adult height, delaying the pubertal growth spurt and slowing the speed of maturation compared to counterfactual cohorts who did not experience the war. However, the war shock to the growth pattern is strikingly absent from the mean adult male height series because children experienced catch-up growth after the end of the war when economic and health conditions improved dramatically. These gains were even possible for children who experienced the harshest conditions of the war in the critical first thousand days of life (Schneider et al., 2021).

These findings are in accordance with recent research on current LMICs as well. Prentice et al. (2013) showed that children in the Gambia experienced catch-up growth during adolescence that left them less stunted in adulthood than they had been in early childhood. Two papers have challenged their findings arguing that although the HAZ score improved with age in adolescence, the difference in mean height between the reference and the Gambian children did not: that is,



**FIGURE 9** Evidence of catch-up growth following early life shocks. *Sources:* (a) Figures from Steckel (1987) and computed into HAZ scores in Schneider (2017a), (b) stunting rates are computed from Ministry of Education data (Schneider et al., 2023). The break in the stunting rate series reflects missing data. Mean adult male height comes from two sources: the Baten-Blum dataset, which is based on the heights of conscripts (Baten & Blum, 2012) and the NCD-RisC estimation of mean height, which is a smoothed measure produced through their Bayesian Hierarchical Modeling exercise (Bentham et al., 2016). [Colour figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com)]

the HAZ scores were increasing because the standard deviation of the reference was growing over age rather than the Gambian children moving closer to the mean of the reference (Leroy et al., 2015; Lundeen et al., 2014). However, the striking historical findings presented above cannot be explained as an artifact of the reference. The catch-up growth for enslaved people was large enough to close the gap between the mean heights of enslaved children and the reference, and Schneider et al. (2021) measured catch-up growth of cohorts of Japanese children in relation to one another rather than relative to the WHO reference. Thus, it is clear that catch-up growth from poor health in early life is possible.

However, catch-up growth does raise questions about which indicators should be used to measure the effects of health shocks. As is clear from the Japanese case, health shocks that had a significant influence on the growth pattern can end up having little noticeable impact on adult height. Thus, child stunting and the growth pattern more generally may be more sensitive indicators of health shocks than adult height. Likewise, adult height may be more sensitive to shocks in adolescence than in early life since there is less time for adolescents to experience catch-up growth (Depauw & Oxley, 2019; Thompson et al., 2019; van den Berg et al., 2014). Recent studies show that the growth path also reveals deficiencies even if someone on a relatively unhealthy growth path ends up at a normal adult height (Thompson et al., 2020; Wells, 2017). It is not clear that reaching a “healthy” adult height per se means that an individual had a healthy growth pattern.<sup>22</sup>

This subsection has highlighted how dynamic the process of growth is. Children are particularly sensitive to health shocks and interventions in particular critical windows such as the first 1000 days and adolescence, but changes in health conditions can lead to either growth faltering or catch-up growth, complicating the process of interpreting a child’s health from a height measurement at any particular age. Researchers should strive to understand the growth pattern more holistically and use longitudinal data to study this dynamic process directly.

## 7 | DISCUSSION

Having presented how the growth pattern has changed over time and reviewed literature on the factors affecting the growth pattern in current LMICs and historical HICs, this section attempts to draw some general conclusions about what these two groups of researchers can learn from each other.

### 7.1 | Lessons from current LMICs for historical research

The literature on child stunting in current LMICs presents a number of suggestions for future historical research. First, the focus of modern development literature on children is important for historical researchers. While sources of adult stature are far more numerous historically than records of children’s growth, to truly understand the secular change in the growth pattern, we need to research all aspects of the growth pattern rather than simply focussing on one characteristic. The discussion of catch-up growth also highlights that some health shocks may be attenuated in adult stature if health conditions improved afterwards. There is much to learn from the growth trajectory that children took to their adult height.

In addition, the emphasis in the modern development literature on reinforcing and compensating parental investments is something that economic historians could study more carefully (Almond & Mazumder, 2013). Before the health transition, higher prevalence of infectious

disease and the ineffectiveness of most medical treatment meant that there were substantial and observable differences in health endowments across children in a household. Whether parents compensated for or reinforced these endowments would have had important impacts on intra-household inequality and gender disparities in health and growth. More historical research is needed to disentangle these effects, though the challenge of sources is particularly acute here. Historical studies also need to work harder to include girls or women in studies of parental investments. Sources for female stature are rarer than those for male stature, and it is difficult to link women in censuses or other administrative records, but capturing the full household is crucial for understanding how parental responses shaped the growth pattern across the health transition.

The comparison between maternal health in India and historical HICs also suggests that maternal health may have been substantially better at the outset of the secular change in the growth pattern in historical HICs than in current LMICs. However, more research is needed on maternal health in history to confirm this pattern. This research should take two forms. First, historical researchers need to collect information on maternal health, such as height and BMI, for additional countries to confirm the tentative patterns laid out above for Britain and the United States and to extend our understanding of maternal health to other current HICs like Japan and South Korea that had very high stunting rates in the past. Were high BMIs and low rates of underweight the norm in the past? Did early health interventions such as the end of open defecation improve women's health before the start of the secular change in the growth pattern? How did inequality in women's BMI change over time? Second, researchers need to expand the set of indicators for maternal and prenatal health in order to capture the multiple dimensions of health and how these have changed. Researchers have already begun collecting information about placental weight as another useful indicator (Butie et al., 2020; Galofré-Vilà & Harris, 2021), but other maternal health outcomes such as gestational diabetes, anemia, and nutrition will also help to understand how maternal health changed over time.

Another important insight from research on current LMICs is that atmospheric pollution may be a major driver of child stunting. While initial historical work has shown that coal pollution affected adult height in Britain (Bailey et al., 2018), extending these cross-sectional findings to the secular change in the growth pattern is more difficult. Respiratory disease deaths were a large contributor to infant mortality, so it is possible that part of the effect of declining infant mortality on increasing heights in Europe (Hatton, 2011, 2014) could be driven by reductions in coal pollution. However, the trends in levels of atmospheric pollution are unclear. While some have suggested that pollution (at least in London) improved before the First World War (Clay & Troesken, 2011), levels of ambient pollution were still extremely high in the 1950s when instrumental measures of pollution started (Hanlon, 2022). This could mean that the changes in the growth pattern and reductions in mortality from respiratory diseases had occurred despite continued high levels of ambient pollution. However, there had been considerable change in household pollution as people shifted from burning coal for heating and cooking to using gas (Clay & Troesken, 2011). Thus, more research is needed to understand how the changing sources and levels of atmospheric pollution affected changes in health and the growth pattern over time.

Finally, there are lessons for anthropometric historians from the current paradox that WASH, and especially open defecation, appears to be a very important cause of child stunting (Spears, 2020) but that WASH interventions are difficult to implement and can be ineffective (Pickering et al., 2019). Anthropometric historians have not measured WASH interventions with the same precision as researchers studying historical mortality decline. Assessing the effects of the construction of clean water and sewerage systems would be a good place to start, but further research is needed to capture the effects of household-level sanitation behavior as well. In addition,



contemporary research suggests that the shift from open defecation to latrines may have been an important early health intervention that would be worth exploring.

## 7.2 | Historical lessons for sustainable development

A historical perspective provides interesting insights for researchers studying LMICs today as well. First, it is very striking that birth weights have not increased in the past 150 years in Western Europe, North America, Australia, and New Zealand (Schneider, 2017b). When comparing maternal BMIs in historical HICs with current LMICs, it is also clear that maternal health may have been better at the outset of the secular change in the growth pattern in historical HICs than it was in current LMICs, and especially India. Thus, this is a key difference between the secular change in the growth pattern between Western countries and some LMICs. This yields two important realizations. First, it suggests that the kinds of interventions that prompted the secular change in the growth pattern (and elimination of child stunting) in current HICs may not be enough to eliminate stunting where maternal health conditions continue to be so poor. Poor maternal health can significantly attenuate the improvements that other factors might have on shifting the growth pattern since women give birth to all children and therefore pass on their health status to the next generation (Bhalotra & Rawlings, 2013; Osmani & Sen, 2003). Second, it emphasizes the need to understand why maternal health is so much worse in current LMICs. Is maternal health bad because all men and women are underweight, or is gender inequality contributing to poor maternal health?

On both points, the historical experience of Japan may be instructive. Across the first three-quarters of the 20th century, Japanese mean birth weights increased by perhaps 250 g from a very low level of 2955 g (Kato et al., 2021; Misawa, 1909). This was the same period in which stunting was eradicated (Schneider et al., 2023), suggesting that improvements in maternal health contributed to the eradication of stunting in Japan from a very high level. However, since the 1970s, birth weights in Japan have fallen 200 g to a mean of 3000 g. This large decrease cannot be explained by changes in gestational age, parity, or paternal age, though it is associated with an increase in the share of women aged 20–39 underweight from c. 10% to 20% (Kato et al., 2021). Since the 1970s, Japanese adults have stopped growing taller, but there has been no increase in child stunting. Japan's experience suggests that increases in birth weight may be necessary to eradicate stunting (Aiyar & Cummins, 2021; Coffey, 2015; Grafenstein et al., 2023), but once a population has reached a certain baseline level of health, stunting can be avoided through postnatal interventions.<sup>23</sup>

Second, reducing child stunting is essentially manufacturing a change in the growth pattern of children. Given the evidence that we have about changes in the growth pattern, it is clear that this is a long-term process taking many decades. Thus, historical analysis of trends in child stunting over time may help in setting targets for the elimination of child stunting in LMICs in the future.

Third, many current HICs had high levels of child stunting in the past. This confirms that reductions in child stunting have been a general part of the health transition around the world. Figure 8 also demonstrates clearly that geography and income are not fate. Subtropical and tropical countries are not doomed to high levels of child stunting because Cuba and Jamaica had reached low levels of stunting by the mid 20th century and most Latin American countries have reached low levels of stunting today. Likewise, GDP per capita is only loosely related to historical stunting: stunting rates were far lower in Cuba and Jamaica in the 1940s than they were in Japan despite Japan being well on its way to becoming a HIC. Plotted together in Figure 8, it is interesting to see how much child stunting has fallen across all types of countries (Schneider et al., 2023).

Fourth, the historical examples presented in Figure 9 show that it is possible for children who face adverse health conditions in early life to experience catch-up growth later in life. These findings stand in contradiction to the strong consensus in the development economics and nutrition literatures that children who experienced health shocks and growth faltering during the first 1000 days cannot recover at later ages (Alderman et al., 2006; Almond et al., 2018; Hoddinott & Kinsey, 2001; Proos, 2009; Victora et al., 2010; Wells, 2017). However, these examples concur with a growing range of other studies that suggest that it may be possible for children to recover in the long run from health shocks at earlier ages and that late childhood and adolescence might also be a critical period for development (Akresh et al., 2021; Aurino et al., 2023; Depauw & Oxley, 2019; Prentice et al., 2013; Schneider & Ogasawara, 2018; Singh et al., 2014; van den Berg et al., 2014).

Whether recovery in height translates into recovery in other dimensions such as cognitive development and health is more difficult to answer. There is clear evidence that stunted children have poorer human capital outcomes later in life (Hoddinott et al., 2013; Perkins et al., 2017). However, there is a small but growing literature on “double shocks” that explicitly tests whether children who faced a nutritional shock in early life can recover in the cognitive domain when targeted by an intervention (Almond et al., 2018). Using prospective cohort data for Ethiopia, Peru, India, and Vietnam, Crookston et al. (2013) found that children who recovered from stunting in early life had improved cognitive outcomes relative to those who remained stunted. In Peru, children who recovered from early life stunting had similar cognitive outcomes to never stunted children in mid childhood (Crookston et al., 2010). However, recovery from stunting was not randomized in these studies, which could mean that recovery was correlated with unobserved confounding factors. Two papers in economics try to address this potential bias more directly. Akresh et al. (2021) test whether individuals exposed to adverse health conditions in early life caused by the Biafran Civil War in Nigeria (1967–70) could recover when exposed to a primary education campaign years later, and Adhvaryu et al. (2023) test whether Mexican children who experienced rainfall shocks in early life could recover after being randomized into the PROGRESA conditional cash transfer program. In both cases, the positive interventions substantially mitigated the initial shocks when looking at cognitive and human capital outcomes for exposed children at later ages. Thus, although the literature is relatively underdeveloped, it does seem that catch-up growth can produce gains in cognitive development as well.

To be clear, the vast majority of stunted children do not experience interventions that would allow them to recover from growth faltering in early life (Benjamin-Chung et al., 2023). Thus, policymakers should continue to focus on protecting children in the first 1000 days. However, the fact that recovery in both growth and cognitive outcomes may be possible should encourage researchers to test interventions that could mitigate the damage of being stunted for the 148.1 million children who are already stunted in the world today (Unicef et al., 2023).

## 8 | CONCLUSION

Taking a global and long-run perspective, this review has reconstructed changes in the growth pattern around the world and surveyed literature on the causes of change in the growth pattern. Stunting is caused by a wide range of factors that vary across different contexts (Headey et al., 2017; Nisbett et al., 2023). Both the contemporary and historical literature often do a better job of identifying the causes of child stunting than testing direct interventions that could help to reduce stunting in the future. This is particularly true of economic historians since we are not able to run field experiments to test specific policies. Unfortunately, it is easier to rule out certain factors than

to highlight specific interventions to implement moving forward. It seems that genetic differences, selective mortality, and improvements in nutrition have only very small effects on explaining changes in child stunting. While household allocation in favor of first-born sons may explain part of the Indian Enigma, it seems unlikely that within household allocation could be responsible for the secular changes in the growth pattern at the population level: creating parity among Indian siblings would not eliminate stunting. Atmospheric pollution is relatively untested compared to other factors, but what little evidence there is suggests it may have a relatively small effect. Economic growth seems to have mattered more in historical Europe than for current LMICs. On the contrary, poor maternal health seems likely to be more important in explaining child stunting in India and many other LMICs but cannot explain the secular change in the growth pattern in Western countries since maternal health (proxied by BMI) had reached a fairly high level before the secular change in growth began. WASH seems the best explanator of high levels of child stunting, but the failure of WASH interventions today and the limited effects of the introduction of clean water and sanitation in reducing historical diarrhoeal mortality suggests that WASH interventions may not have been most critical to reductions in stunting.

The question, then, is what factors remain having ruled out most of those discussed? To me there are two factors missing. The first factor is people's attitudes toward health, hygiene, and sanitation. WASH interventions require people to fundamentally change their most closely held habits and health behaviors. This change in perspective took place in countries that have eliminated stunting. The second factor is generalized development where over time people have more resources and human and social capital to expend on cleaning their living space, learning about and adopting new hygiene technologies, moving to less crowded and more hygienic homes and neighborhoods and demanding better public health provision from governments. The interconnections between income, housing quality, neighborhood amenities, and governance make it extremely difficult to separate these factors econometrically, but researchers should not lose sight of the fact that these less tangible factors likely mattered a great deal. Future research may want to explore these factors further.

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## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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## ENDNOTES

<sup>1</sup>This review focusses on height rather than weight or body mass index (BMI) in order to make the discussion manageable. Weight and BMI are caused by different biological and social processes, which would be difficult to cover in detail here.

<sup>2</sup>The heritability of height changes across the growing years with heritability at its lowest in infancy and early childhood, increasing to mid-adolescence and falling slightly afterwards to adulthood (Jelenkovic et al., 2016).

<sup>3</sup>Online Appendix B provides a brief history of growth standards and references.

<sup>4</sup>See Figure 9a for an example.

- <sup>5</sup>I use the Northeastern region of Brazil as a case study to show that growth faltering was common in history and to show a more severe form of growth faltering: see Figure 5 for more examples.
- <sup>6</sup>Although children can achieve catch-up growth from early life shocks. See Section 6 for more detail.
- <sup>7</sup>Note that the dispersion of the HAZ distribution does not affect the stunting rate when the mean HAZ score is  $-2$  because the stunting threshold is equal to the mean of the distribution so the stunting rate will be 50% no matter what the dispersion in HAZ.
- <sup>8</sup>Online Appendix C describes and critiques the historical and recent data sources used to reconstruct the growth pattern over time.
- <sup>9</sup>The share of children born weighing 2500 g or less.
- <sup>10</sup>Although birth length is recorded in some historical sources in Europe and North America, the substantial error in measuring infant length makes long-run comparisons difficult.
- <sup>11</sup>Note that they do not have data for the 1895 birth cohort for all countries, but instead impute adult heights for many countries using a Bayesian hierarchical model.
- <sup>12</sup>Unfortunately, I could not find a study from the early 2000s covering representative Indian children over age 5, so India can only be studied in reference to children under the age of 5.
- <sup>13</sup>Evidence on children's growth at later ages suggests that growth faltering would have been greater in the early 20th century (Schneider et al., 2023).
- <sup>14</sup>The fact that individuals grew well into their 20s also cautions against using conscription records of 18 year olds as a straightforward measure of final adult height.
- <sup>15</sup>Data on the heights of enslaved children in Trinidad and Tobago in 1813 suggest a stunting rate of 78%, so it seems unlikely that stunting rates were always low in the Caribbean (Higman, 1979).
- <sup>16</sup>I will primarily focus on research published since the last major survey of the anthropometric history literature (Steckel, 2009). This is not an exhaustive literature review of anthropometric history since 2009 but instead focuses on research related to the secular change in the growth pattern.
- <sup>17</sup>Bodenhorn et al. (2017) argue that sample-selection bias drove the declines in mean adult stature during the 19th century in Britain and the United States. Their critique has sparked substantial research on sample-selection bias in historical sources (Inwood & Maxwell-Stewart, 2020; Komlos & A'Hearn, 2019; Zimran, 2019), but the general consensus seems to be that these declines were not simply figments of selection bias.
- <sup>18</sup>See the forthcoming review paper by Gallardo-Albarrán for more detail.
- <sup>19</sup>This is a form of collider bias that arises from implicitly adjusting for a descendant of a mediator (Schneider, 2020a).
- <sup>20</sup>Maternal health capital is a combination of the mother's health status at the time of the pregnancy and her own experience of health shocks during development (Wells, 2017).
- <sup>21</sup>These theories are discussed in more detail in Online Appendix A.
- <sup>22</sup>Another caveat is that catch-up growth in height may not compensate for other cognitive and developmental losses (Hoddinott et al., 2013): see Section 7.2 below for further discussion.
- <sup>23</sup>This also makes one wonder whether the secular increase in adult stature would have continued in Japan if this decline in birth weights had not occurred.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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