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Disease and child growth in industrialising Japan: Critical windows and the growth pattern, 1917–39☆

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ABSTRACT

This paper assesses how the disease environment, proxied by infant mortality rates, influenced children's growth in interwar Japan. We use data drawn from government records from 1929 to 1939 which report the mean heights of boys and girls in school at each age (6–18) for each of Japan's 47 prefectures. We focus on two key questions: (1) how important was the disease environment in infancy in shaping the growth pattern of children? and (2) were shocks to child health more salient in the first thousand days of life, often held as a critical window to prevent stunting, or at later ages? We quantify the characteristics of the growth pattern of birth cohorts using the SITAR growth model and then relate the predicted SITAR parameters to infant mortality in the year of birth. In addition, we test for instantaneous effects of morbidity, proxied by infant mortality, on growth at ages 6–11. We find that infant mortality in early life did not have a strong influence on the growth pattern of children, but there were statistically significant and economically meaningful instantaneous effects of infant mortality on child height at ages 6–11 for both boys and girls. This suggests that interventions outside of the thousand-day critical window can be effective and that the secular increase in height in interwar Japan was more strongly influenced by cumulative responses to the health environment across child development rather than simply improvements in early life health.

Children's growth is an important measure of health and nutritional status that has been employed in the historical literature to track economic development and health (Harris, 1994; Hatton, 2011; Schneider, 2016, 2017a; Steckel, 1987). Since the late nineteenth century, countries all over the world have seen substantial increases in the mean stature of adults (Baten and Blum, 2014; NCD Risk Factor Collaboration, 2016). There is a vast and developed historical literature studying the factors that have led to changes in the mean height of adults over time. Recent studies have highlighted the importance of both improvements in nutrition and the disease environment in increasing adult stature (Baten and Blum, 2014; Hatton, 2014). However, there has been much less focus on how child growth has changed to make the secular increase in height possible (though c.f. Cameron, 1979; Cole, 2003; Steckel, 1987). In fact, there have been fundamental changes in the pattern of growth that children experience. Not only have children grown taller, but they experience an earlier pubertal growth spurt (and earlier maturation more generally) and faster velocities of growth during

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the growing years. Understanding how the growth pattern changed and the factors that led to changes in the growth pattern is useful because it helps us understand the secular increase in height better.

There is also considerable concern with child growth today because child stunting (the percentage of children below a certain height for age) is one of the most important indicators of malnutrition used to measure progress in the Millennium Development Goals and Sustainable Development Goals around the world (de Onis and Blössner, 2003). The causes of stunting are multidimensional and include poverty, inadequate nutrition and chronic illness from poor water supply, sanitation and hygiene (WASH) infrastructure and practices (Black et al., 2013; Headey et al., 2017b; Spears et al., 2013). These factors cause children to grow more slowly in the first two years of life, leading them to become progressively shorter than healthy children. After age two, stunted children tend to grow at rates similar to healthy children and therefore maintain their position relative to modern growth standards at least until the age of five. This pattern has led many development economists and epidemiologists to argue that there is a 1000-day critical window from conception to age 2 to intervene to prevent stunting (Victora et al., 2010).

A historical perspective can contribute to our understanding of stunting in two key ways (Schneider, 2018b). First, reductions in stunting rates in essence are driven by the same processes as the secular increase in height and change in the growth pattern discussed above (Cole and Mori, 2017). Thus, historical examples of how the growth pattern changed may reveal the kinds of policies and interventions that might help reduce stunting in the developing world today. Second, we can use historical data to test whether shocks to child health in the first thousand days are more salient for later growth outcomes than shocks occurring at later ages. There is some evidence that children can experience catch-up growth at later ages (c.f. Prentice et al., 2013; Steckel, 1987), but little is known about what factors allow for this kind of catch-up growth.

This paper uses interwar Japan as a case study to address two key questions: (1) how important was the disease environment in changing the growth pattern of children? and (2) were shocks to child health more salient for child growth if they occurred in the thousand-day critical window or at later ages? Japan is a useful environment to study these questions for several reasons. First, between the birth cohorts of the 1880s and 1980s, the average height of Japanese adult men increased by 13.9 cm (Baten and Blum, 2012), so the secular increase was strong in Japan. There was also a large decline in the stunting rate of children in Japan from over 65% in 1900 to around 6% today (Schneider, 2018b).[1] Thus, there was a radical change in the growth pattern in Japan across the twentieth century, making it an ideal case for studying changes in children's growth. Japan is also an ideal case because there is very rich historical data available. Drawing on government reports, we have constructed a panel dataset containing the average heights of boys and girls from age 6 to 21 for all 47 prefectures from 1929 to 1939. We have also collected data on a host of variables related to the disease environment and nutrition levels in each prefecture, including the infant mortality rate which we use as our primary proxy for the disease environment.

To test the influence of disease on the growth pattern, we use the SITAR growth model developed by Cole et al. (2008, 2010) to parameterise the growth pattern of historical populations. SITAR predicts parameters that measure the size, tempo (timing of the pubertal growth spurt) and velocity of each cohort growth curve in our dataset. We then take the predicted SITAR parameters for each prefecture-birth cohort and use these as dependent variables in fixed effects regressions. These regressions analyse how changes in infant mortality along with a number of other variables that capture the health conditions in the prefecture in the year of birth affected each SITAR parameter.

In addition, to address whether interventions outside the thousand-day critical window can influence growth, we estimate the effect of infant mortality and a few other covariates on the heights of children at ages 6–11. We use infant mortality to proxy the chronic disease load of children at later ages because the diseases that kill infants such as diarrhoea and respiratory diseases tend to make older children chronically ill without the same mortality consequences (Hatton, 2011; Sharpe, 2012). We use a bilateral-specific fixed effects model, which includes fixed effects for prefecture interacted with birth year, to control for any differences in initial birth conditions across prefectures and cohorts. There is greater potential for endogeneity in these instantaneous regressions, but by focusing on mortality of younger children, especially infant mortality, in the same year, we mitigate this potential bias.

Briefly, we find that health conditions in early life did not have a strong influence on the growth pattern of children in interwar Japan. However, we do find a statistically significant and economically meaningful effect of the infant mortality rate children were exposed to at ages 6–11 on child height at the same ages. Thus, the answers to our two questions are mixed. We find that the disease environment around birth did not strongly shape the growth pattern and that health shocks outside the thousand-day window were more important for child growth than those in early life. This suggests that the secular increase in height in interwar Japan was more strongly influenced by cumulative responses to the health environment at all ages across development rather than being simply the outcome of improving health conditions in early life.

1. Background

1.1. Child growth in Japan

Before getting into the specifics of our study, it will be helpful to discuss briefly the secular increase in height and changes in children's growth in Japan. Fig. 1 shows the mean male adult height of Japanese men compared with other East Asian countries and the United Kingdom. In the late nineteenth century, Japanese men were shorter than their other East Asian counterparts and 10 cm

[1] These rates are estimated from the mean national height-for-age Z-scores of children at age 6 assuming a normal distribution and constant standard deviation over time. These are rough estimates and should be taken as such.

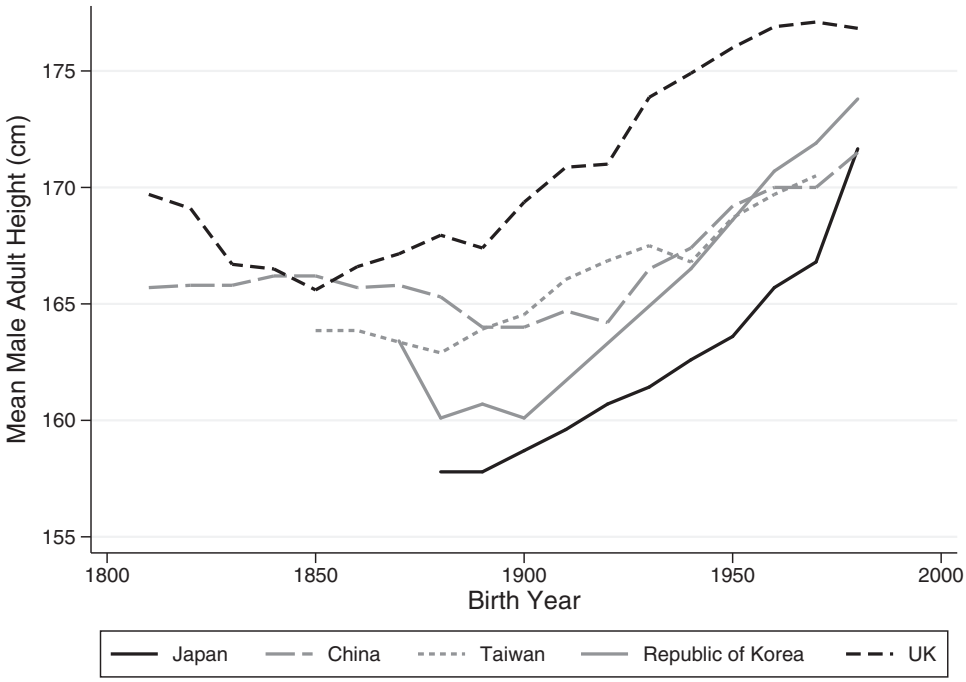

Fig. 1. Trends in mean adult male stature in Japan compared with other East Asian countries and the UK.
Source: Baten and Blum (2012).

shorter than men in the UK. However, there was a strong secular increase in height in Japan so that men born in 1980 were nearly 14 cm taller than those born in 1880. This led to considerable convergence with China and Taiwan although South Korea now has the tallest men in East Asia. This substantial increase in height is comparable in magnitude and rate with the secular increase in many countries (NCD Risk Factor Collaboration, 2016).

The Japanese secular increase in height also coincided with changes in the growth pattern of children. Fig. 2 presents a typical height curve for a boy, based on the 2007 WHO growth reference, on the left with its corresponding growth velocity curve on the right. The growth pattern in childhood and adolescence, then, is defined by three key characteristics: (1) the final adult height; (2) the timing of the pubertal growth spurt or the age at which a child reaches peak growth velocity in puberty; and (3) the level of velocity at the peak and surrounding the peak. Fig. 3 shows average growth and velocity curves for boys measured cross-sectionally from 1900 to 2016 based on national data collected by the Japanese government. These curves show a definitive change in the growth pattern of Japanese children. Not only did the heights of children increase over time, but the age at peak velocity shifted to earlier ages from age 14 in 1900 to age 12 in 2016. The peak velocity during the growth spurt also increased over time. These changes in the three characteristics of the growth pattern are similar to those observed in other countries (Cameron, 1979). Previous historical studies have almost exclusively focused on only one characteristic of the growth pattern, final adult height, so one of the goals of this paper is to understand how the disease environment influenced all three characteristics of the growth pattern.

We are not the first to study the anthropometric history of Japan (c.f. Mosk, 1996; Saito, 2004; Bassino, 2006) or use the Ministry of Education data on children's heights. Saito (1989) first studied these records finding a significant correlation between changes in the infant mortality rate and children's heights at various ages in 1938. Later, he conducted a more specific study of Yamanashi prefecture in 1902–1906 to look at the differences in children's growth in rural and urban areas (Saito, 2003). Bassino and Kato (2010) have analysed prefecture-level data on children's growth from the post-war period in an attempt to understand why the secular increase in height stopped in Japan in the 1980s. Bassino's (2006) article is also relevant as it shows the importance of income and access to health services for final adult heights in Japan. Finally, Ogasawara (2017) recently showed that children born during the 1918–20 flu pandemic experienced a long-run height penalty relative to cohorts born shortly before or after the pandemic.

1.2. The disease environment and child growth

Child growth and final adult height are influenced by a number of factors related to children's health during the growing years. The quantity and quality of the diet that children receive influences their growth, and many studies have found that protein rich foods such as milk and eggs are especially important to promoting more rapid growth in childhood and increasing adult height (Baten and Blum, 2014; Headey et al., 2017a). However, more recently the literature has begun to recognize that the disease environment that children are exposed to can also have a profound influence on their growth. In both contemporary and historical settings, children exposed to cramped living conditions, poor sanitation and unclean water and airborne pollution are shorter than their counterparts

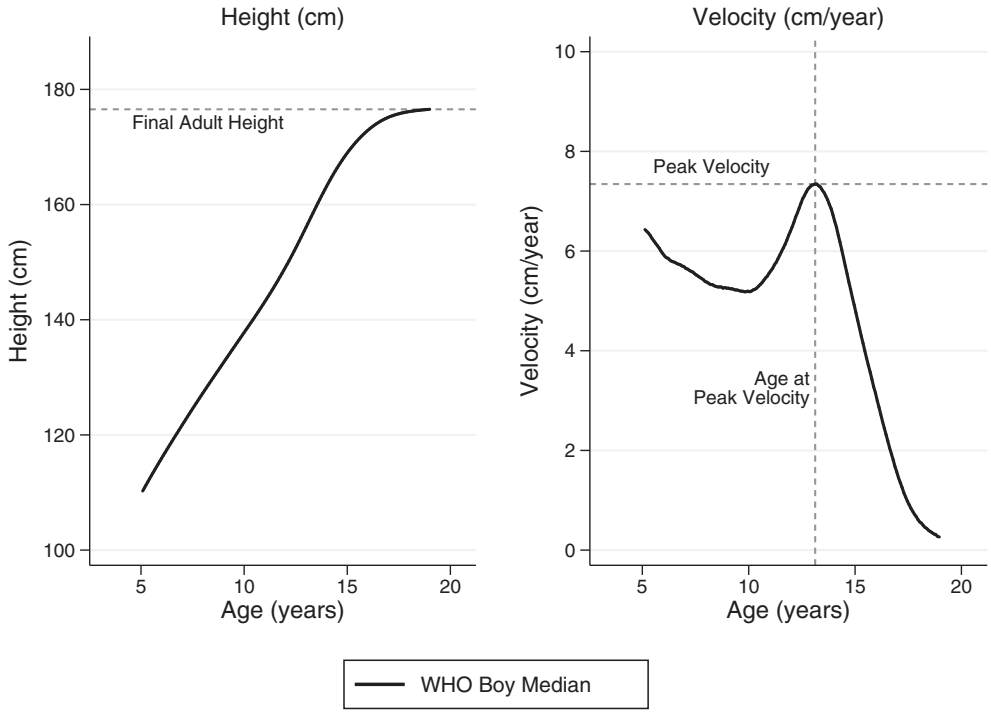

Fig. 2. Characteristics of a typical growth curve for boys.
Source: WHO Child Growth Standards—data drawn from http://www.who.int/growthref/en/.

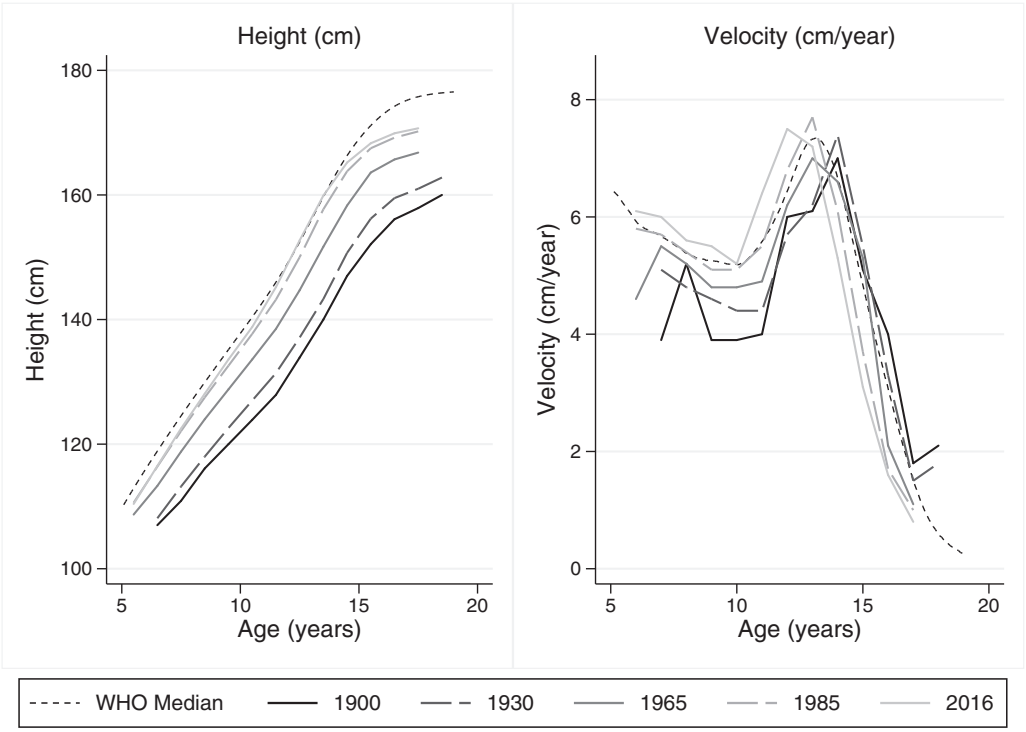

Fig. 3. Growth pattern of boys measured in Japan at the following years.
Source: WHO Child Growth Standards—data drawn from http://www.who.int/growthref/en/; Japan—see Web Appendix B.

living in healthier conditions (Bailey et al., 2016a,b; Headey et al., 2017b; Nabwhera et al., 2017; Spears et al., 2013). This paper attempts to measure the influence of the disease environment in infancy and at later ages on growth while also controlling for important nutritional variables.

When analysing the influence of improvements in the disease environment on subsequent health outcomes, it is important to remember that the observed relationship will be the balance of two sets of factors (Bozzoli et al., 2009; Deaton, 2007; Hatton, 2011). Reductions in child morbidity would decrease the physiological penalty of chronic morbidity on growth making the surviving children taller than children in a high morbidity environment, i.e. before the infant mortality decline. This is a reduction in the scarring effect of the disease environment on later health outcomes. However, improvements in the disease environment could also affect the survival of children in a given cohort. If the survival of children was positively related to their height, this could mean that in poor disease environments, short children were more likely to die than tall children. Thus, an improvement in the disease environment could lead to more short children surviving and therefore reduce the average height of survivors after the improvement. This is commonly known as the selection or culling effect.

Many researchers have been unable to separate the scarring and selection effects and report their results as the balance of the two (Bozzoli et al., 2009; Deaton, 2007). However, there have been two strategies to isolate the scarring and selection effects. The first is to focus on diseases that cause high rates of chronic morbidity but do not affect mortality. Bleakley (2007) uses the eradication of hookworm in the US South as an example of this type of morbidity since hookworm affected a large number of children but did not increase their mortality risk. The second strategy uses the difference in the causes of morbidity and mortality at different ages in childhood. Hatton (2014) argues that diseases that tended to kill infants like diarrhoea, bronchitis and pneumonia were the kinds of diseases that made young children chronically ill and that these diseases had much lower fatality rates in early childhood than during infancy (Sharpe, 2012). Thus, Hatton (2011) uses the infant mortality rate that children were exposed to at ages 2–4 to measure the scarring effect of child morbidity on subsequent growth. Any selection is mitigated by the fact that infant mortality rates at ages 2–4 do not affect the cohort being observed later and the diseases most strongly linked to infant mortality do not cause high mortality in childhood.

We follow Hatton's approach in this paper, and his arguments about child morbidity seem to hold for interwar Japan. Although infant and child mortality (exact ages 1–5) are positively correlated between 1929 and 1939, the correlation is not particularly strong at only 0.397. It seems infant and child mortality capture different aspects of the disease environment. In addition, on average the infant mortality rate was 6.0 times higher than the child mortality rate, making any potential selection effects much weaker in childhood. Web Appendix D also shows that the levels, rates of decline and causes of infant mortality in Japan were not substantially different from those in England. Thus, we think results in Japan will be comparable with the existing literature mainly focused on Europe.

The results in the historical literature for the influence of the disease environment, proxied by the infant mortality rate, on height are somewhat mixed so far. Hatton (2011) and Bailey et al. (2016a) find strong scarring effects in late-nineteenth and twentieth-century Britain. Hatton (2014) and Baten and Blum (2014) again find strong scarring effects when examining adult heights in the nineteenth and twentieth centuries in European and global panels respectively. However, Stolz et al. (2013) do not find a significant effect of infant mortality on adult male stature in a European cross-country panel covering the period 1720–1910, suggesting that declines in infant mortality in the twentieth century may be most important in driving the other findings. Likewise, Öberg (2015) finds only very small scarring and selection effects on adult height in Sweden between 1814 and 1948. On the other hand, Deaton (2007) and Bozzoli et al. (2009) find that the selection effect was stronger than scarring in developing countries in the late twentieth century. Thus, this paper seeks to expand earlier analysis of the disease environment on child growth in two ways: first, we extend the historical analysis to a non-Western country; and second, we measure the influence of the disease environment on the growth pattern rather than children's heights at a specific age or adult height.

2. Data

The main sources of data for this paper are the Statistics of School Physical Examination published by the Physical Bureau of the Ministry of Education between 1931 and 1943. These reports record the average heights of boys and girls aged 6–21 for each of Japan's 47 prefectures measured annually between 1929 and 1939. These average figures were compiled from physical examinations that occurred in all primary schools (*shōgakkō*), higher primary schools (*kōtō shōgakkō*), secondary schools (*chūgakkō*), and girls' high schools (*kōtō jyogakkō*). The averages reported therefore reflect the children in school at each age. Unfortunately, the individual-level data originally collected has not survived. The data list the heights of primary school and secondary school children separately, and the two school types overlapped between ages 11 and 15. Thus, we have a 47-prefecture panel of children's heights at a large range of ages measured each year between 1929 and 1939. Appendix Fig. A1 presents maps that display the large variation in the average height of children at age six across the prefectures. Okinawa is clearly an outlier with especially short children.

Given the recent debate over the importance of selection bias in anthropometric sources (Bodenhorn et al., 2017; Schneider, 2018a), it is worth discussing potential selection biases in the data presented here. As mentioned above, the Statistics of School Physical Examination report the average heights of children in school. Thus, changes in the percentage of the population enrolled in school could lead to selection bias in our sample. Fig. 4 shows the mean height of boys in Japan measured in 1936 separating primary and secondary schools. On the right-hand axis, it also shows the percentage of boys enrolled in 1938 at each age in primary and secondary school. For boys (and girls which are not reported but very similar) the share of children in primary school hovered around 95% of the population from ages 6 to 11 declining to 60% by age 13. The share in primary school then dropped to very low shares of 2.8 and 0.1% at ages 14 and 15 respectively. Secondary schools, on the other hand, were the reserve of a privileged few in

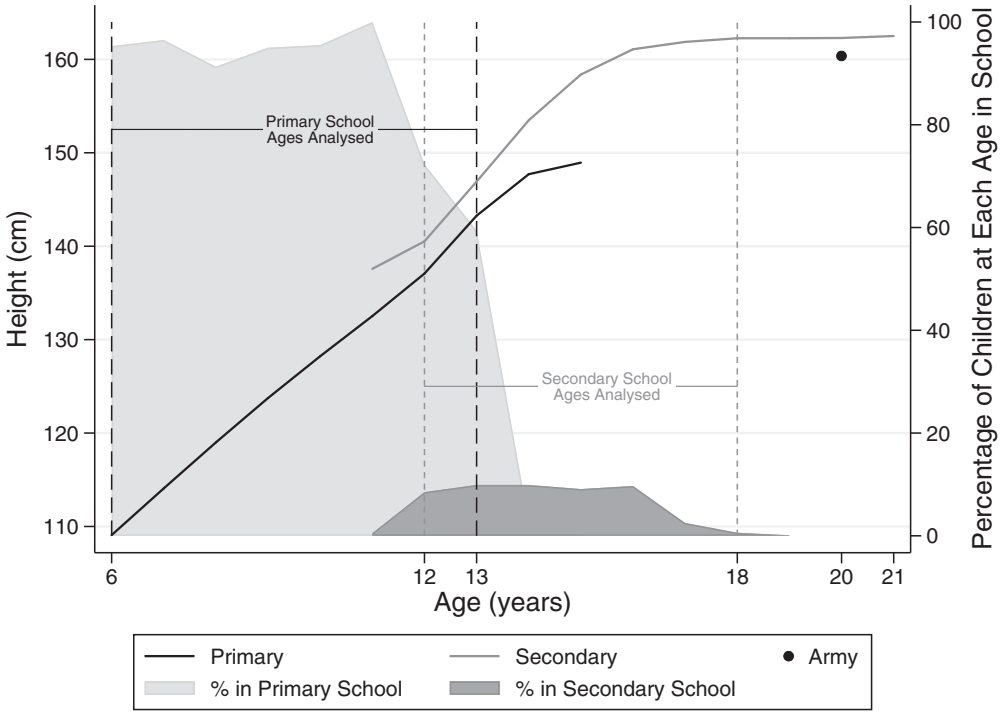

Fig. 4. Weighted mean height of boys in primary and secondary schools measured in 1936 across the 47 prefectures compared with the heights of conscripts.
Source: See Web Appendix B.

the interwar period with only around 9% of the population enrolled between the ages of 12 and 16 and lower share at other ages. The elite, selected nature of secondary schools shows up in the data with secondary students around 3.4 cm taller than primary students at ages 12 and 13 when the data are reliable for both. We can also see this positive selection when comparing secondary school children at age 20 with army conscripts representing again 95% of the population: the secondary school children were 1.3 cm taller than army conscripts. Thus, given the selection of secondary school students, we conduct the bulk of our analysis on the primary school children, only using the secondary school children to complete the growth curve.

In addition to these selection issues across school types, there are also selection problems across ages during the transition from primary to secondary school. Children who enter secondary school at age 11 were slightly taller than expected based on secondary children's heights at later ages because only the very elite entered secondary school at such an early age. Likewise, primary school children at ages 14 and 15 became more and more negatively selected as the proportion of all children in primary school at these ages fell. After the age of 18, there was also a huge amount of measurement error in the average heights of secondary school children because of small sample sizes. The error in the estimates at these ages is even more pronounced when looking at the individual cohort growth curves (Appendix Fig. A2). Thus, we only include primary school children aged 6–13 and secondary school children aged 12–18 in our analysis below (see Fig. 4).

Another feature of our panel is that we can express the time dimension in two ways: the year in which the children were measured (period), 1929–39, or the year in which the children were born (cohort), 1911–1933. Thus, we can relate the heights of children either to conditions they were instantaneously facing in a given year (period effects) or to the conditions they faced in early life (cohort effects). Accordingly, we have constructed a prefecture panel of control variables that capture socioeconomic and environmental conditions in each prefecture from 1911 to 1939. These controls and their sources and method of construction are discussed at length in Web Appendix B. Descriptive statistics for all covariates are also included in Appendix Tables A1 and A2. However, it is worth briefly considering the infant mortality rate since this is the most important independent variable in our analysis. Fig. 5 presents the infant mortality rate in Japan as a whole, in major cities with more than 100,000 inhabitants and in other areas aside from the major cities. Clearly, there was a major decline in the infant mortality rate across our period from over 180 infant deaths per 1000 births to less than 100 with an annual average rate of decline of 3.17%. Thus, if the changing disease environment, proxied by infant mortality, had a strong influence on child growth, we would expect to find it in our Japanese case study.

3. Methodology

Having explained the data, we can now discuss our empirical strategy. We use observational data and do not employ any instrumental variables or natural experiments to precisely identify causal channels. While there are exogenous shocks in our period which

Fig. 5. Infant mortality rates in Japan, 1918–40.
Notes: Major cities have populations larger than 100,000 inhabitants, other areas are the rest.
Source: Statistics Bureau of the Cabinet (1924).

might provide helpful internal validity, for instance the 1918–20 flu pandemic and 1923 Kanto earthquake (c.f. Ogasawara, 2017, 2018), flu pandemics and earthquakes did not happen every year, and therefore the external validity of these natural experiments may be low in helping us understand a long-term process like the secular change in growth (Deaton and Cartwright, 2018). Likewise, we have not found policy shocks that could be employed to sort out the causation.[2] Thus, while we believe our empirical design is an improvement over the existing literature, our estimates cannot be interpreted as fully causal.

3.1. Early life health conditions and growth

We first attempt to answer question one above: how does the disease environment *in utero* and the first year of life influence children's growth pattern in childhood and adolescence? Generally, in order to study the growth pattern, one needs longitudinal data so that individuals' growth can be tracked across various ages. Unfortunately, we do not have individual-level data, only mean heights of children at various ages for each prefecture. However, we can create pseudo-longitudinal growth curves for each prefecture-birth cohort by grouping the heights of children born in the same year but measured at different ages (and in different years) later in life. For example, we observe the heights of children born in Tokyo prefecture in 1923 at age six in 1929, at age seven in 1930, at age eight in 1931, and so on. Therefore, we can create a cohort growth curve from which to analyse the growth pattern of children (see Appendix Fig. A3).

In order to understand the influence of early life health conditions, we have to quantify the characteristics of the growth pattern. We do this using the SITAR (SuperImposition by Translation and Rotation) growth model developed by Tim Cole and co-authors (Cole et al., 2008; Cole et al. 2010). SITAR fits a non-parametric, random effects model to longitudinal growth curves in an attempt to simplify a vast array of individual-specific variation into a single mean growth curve (R Core Team, 2016; Cole, 2016). It is defined by the following equation:

$$y_{i,a} = \alpha_i + h\left(\frac{a - \beta_i}{\exp(-\gamma_i)}\right) \tag{1}$$

[2] Saito (2008a,b) analyses the effect of the Aiiku-son project in 1936, which was a government project that trained midwives in rural areas to improve sanitary conditions and reduce infant mortality rates. We do not use this project to investigate children's growth because the number of designated villages was quite small at the beginning (for example, only 5 villages in 1936), and the project only became widespread from 1939, which is out of the period when data is available.

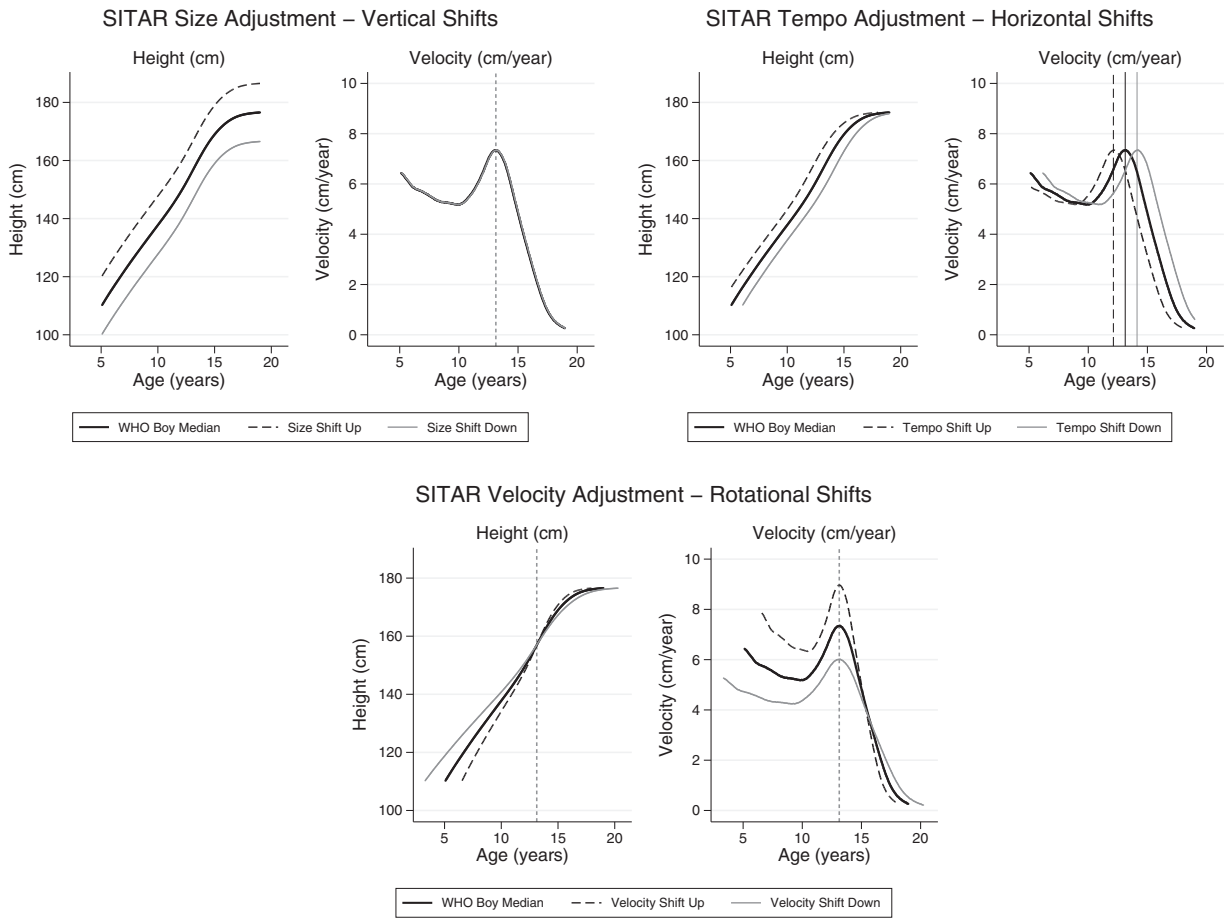

Fig. 6. Height and velocity charts showing the various SITAR parameter adjustments to the growth curve.
Notes: In these figures, the median male growth curve from the WHO school-aged and adolescent growth reference (de Onis et al., 2007) is adjusted to show how each SITAR parameter influences the growth pattern of children.
Source: Sources: WHO Child Growth Standards—data drawn from http://www.who.int/growthref/en/.

where $y_{i,a}$ is the height of prefecture-cohort i at age a, $h(a)$ is a natural cubic spline of height versus age and α_i, β_i, and γ_i are prefecture-cohort-specific random effects. These random effects will be referred to in the rest of the paper as the SITAR parameters. α_i adjusts for differences in mean height between the various growth curves and will be referred to as the size parameter. β_i adjusts for differences in the timing of the pubertal growth spurt between the different growth curves and will be referred to as the tempo parameter. Finally, γ_i alters the duration and velocity of growth during the pubertal growth spurt and will be referred to as the velocity parameter. More technically, this parameter stretches or shrinks the age scale adjusting for differences in developmental age and calendar age across individuals. Thus, SITAR attempts to place each individual growth curve onto the median curve predicted by the spline by adjusting the three parameters.

We present the three parameters and the way they influence the height and velocity curves in the panels of Fig. 6. The size parameter shifts the height curve up or down and does not influence the velocity curve. The tempo parameter shifts the height curve left or right and also shifts the velocity curve left or right but does not change the level or shape of the curve. The velocity parameter stretches or shrinks the age scale and thus changes the velocity of growth and the peak velocity during the pubertal growth spurt. Thus, using the SITAR growth model, we are able to estimate size, tempo and velocity parameters for each prefecture-birth cohort in our data.

However, estimating the SITAR parameters is more complicated than it would first appear because the secondary school children in our data were positively selected. To deal with this, we treat the primary and secondary prefecture-birth cohorts as separate individuals in the SITAR specifications. Thus, the growth curve for primary school students born in Tokyo prefecture in 1922 is entered separately from the growth curve for secondary school students born in Tokyo prefecture in 1922. This allows us to use the secondary data to draw the spline but estimate the SITAR parameters for the representative primary school data and biased secondary

school data separately.[3] We exclude the SITAR parameters produced from the secondary school data in further analysis since any pattern that we might find would be confounded by the changing selection into secondary schools across prefectures and over time. We estimate the parameters for boys and girls separately at all times.

Having estimated the SITAR parameters, we then use them as dependent variables in a series of fixed effects regressions that try to explain variation in the growth pattern using covariates from the birth year. We estimate the following equation:

$$SITAR_{i,t} = \alpha + \beta_1 d_{i,t} + \boldsymbol{x'}_{i,t}\boldsymbol{\gamma} + \nu_i + t\lambda_i + \varepsilon_{i,t} \tag{2}$$

where $SITAR_{i,\,t}$ is the value of each SITAR parameter (size, tempo and velocity estimated separately) for primary school children in prefecture i and birth year t, $d_{i,\,t}$ is the infant mortality rate and $\boldsymbol{x'}_{i,t}$ is a vector of prefecture and birth year variant controls. Controls include those accounting for the environment (annual average temperature), urbanisation, health infrastructure (density of doctors, midwives and hospitals) and income and nutrition (rice yields, soy yields and milk production per capita). It is fairly straightforward to understand why the health infrastructure and income/nutrition variables would affect growth. We include temperature to account for the fact that hotter years typically had worse disease environments and urbanisation to account for disamenities to health from overcrowding in growing cities. We also include prefecture fixed effects (ν_i) and prefecture-specific trends ($t\lambda_i$). The prefecture-specific trends capture unobserved improvements in hygiene (handwashing and general cleanliness) and nutrition quality and diversity that we cannot capture with the other controls (Hatton, 2014; Ogasawara and Kobayashi, 2015; Ogasawara et al., 2018). We are particularly interested in the effects of infant mortality and temperature because these variables reflect the disease environment of the children in early life, which theoretically can have strong effects on the growth pattern (Schneider, 2017a). Statistically significant and economically meaningful results would suggest that the disease environment in early life had a strong influence on the growth pattern of an entire cohort.

In addition to our baseline specification above, we also conduct further analysis to separate the influence of scarring and selection of infants on the growth pattern. We follow Hatton (2011) as discussed above by lagging and leading the infant mortality rate in the regressions. Thus, we use the infant mortality rate as a proxy for child morbidity to test whether disease exposure at later ages affected the growth pattern. We estimate the following equation:

$$SITAR_{i,t} = \alpha + \beta_1 d_{i,t+n} + \boldsymbol{x'}_{i,t}\boldsymbol{\gamma} + \nu_i + t\lambda_i + \varepsilon_{i,t} \tag{3}$$

which is identical to Eq. 2 except that we now allow for lags and leads in the infant mortality rate, $d_{i,\,t+n}$, allowing n to vary from −2 to 8. We do not include multiple lags or leads in the same specification because of very high collinearity. We include a lag of one year before the birth year to capture potential effects *in utero* and a two-year lag as a placebo test since this should not influence the growth pattern. Infant mortality in the year of birth will show the net effect of selection and scarring, but infant mortality in subsequent years ($n > 0$) will reflect the scarring mechanism. Testing for the scarring mechanism is especially important given the focus in the current paediatric and development literature on preventing stunting in the first thousand days of a child's life (Victora et al., 2010). These leads allow us to test how important the early years were to children's growth pattern in interwar Japan and compare with other historical studies (Hatton, 2011).

Although these equations are estimated on observational data, our empirical design substantially improves on previous designs in the literature. Reverse causality problems are limited because of the implicit lags in the data. We regress parameters reflecting a cohort's growth pattern measured at ages 6–18 on conditions in the cohort's birth year. We also have a much wider range of control variables than earlier studies (Hatton, 2011), and we have data for annual birth cohorts rather than data for five or ten-year birth cohorts (Bailey et al., 2016a; Baten and Blum, 2014; Hatton, 2014; Stolz et al., 2013). These precise specifications drastically reduce the potential for omitted variable bias in our regressions relative to earlier work. However, there are still several potential threats to identification. Because we do not include year fixed effects in our baseline, omitted variable bias could arise from any national-level unobservable confounder or shock to health that is correlated with infant mortality rate and causes deviations from prefecture trends in the SITAR parameters. In addition, omitted variable bias could appear if prefecture-specific, time-variant shocks to health that are correlated with infant mortality rates shift the SITAR parameters from their prefecture trends.

3.2. Instantaneous influences on growth

In addition to studying how birth conditions affect the growth pattern of children, we also analyse how the child morbidity during the growing years influenced the children's instantaneous growth controlling for conditions that might have affected each cohort at birth. This allows us to test whether health shocks outside the thousand-day critical window matter for children's growth. We focus our analysis on the heights of primary-school children ages 6–11 where the mean heights of children in our dataset cover over 90% of the population. Fig. 7 presents a lexis diagram that explains the data included in the analysis. Prefecture-birth cohorts are treated as the group variable in the panel with age providing the time variable. Thus, to have a balanced panel, we can only include birth cohorts with the complete set of ages (6–11), i.e. children born from 1923 to 1928. The variation that we analyse then is how health

[3] Appendix C discusses some other robustness checks that we performed to test these assumptions. Unfortunately, we cannot simply exclude the selected secondary school data. The growth curve is nearly linear during the primary school ages (6–13), making it difficult for the model to distinguish between vertical (size) and horizontal (tempo) shifts in the height curve. Thus, we need the curve of the pubertal growth spurt provided by the secondary school data to fit the SITAR model.

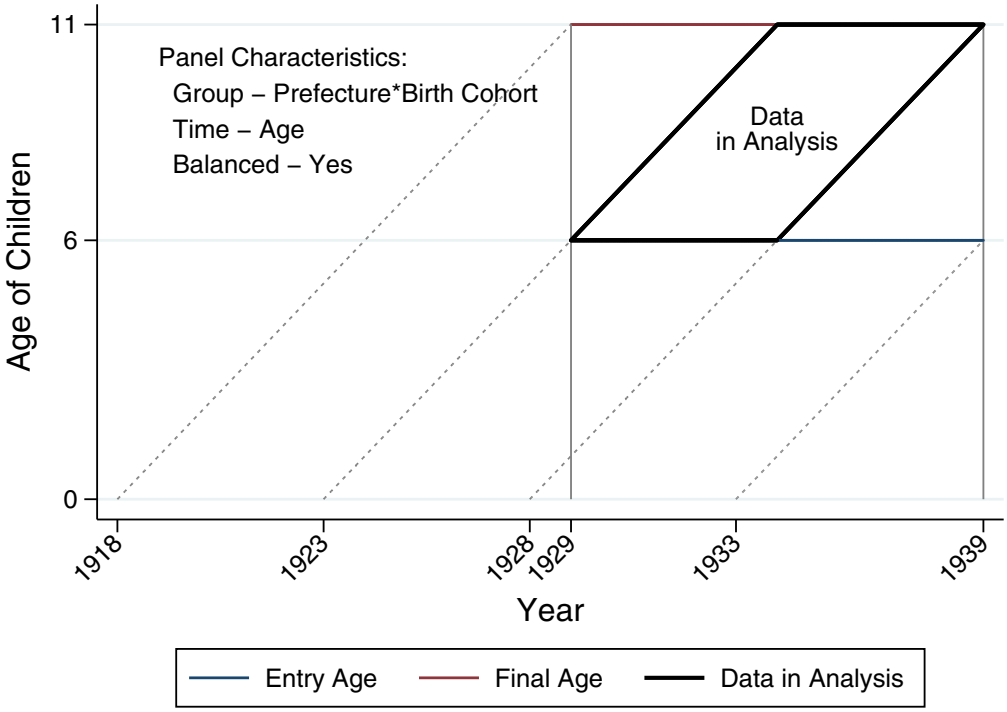

Fig. 7. Lexis diagram showing data used in the instantaneous growth regressions.

conditions in the years in which the children were measured (period years from 1929 to 1939) influenced their growth. To do this, we use the bilateral-specific fixed effects model presented below:

$$h_{i,t,a} = \alpha + \beta_1 d_{i,t} + \beta_2 cd_{i,t} + \boldsymbol{x}'_{i,t}\boldsymbol{\gamma} + \nu_{i,t-a} + \phi_a + \varepsilon_{i,t,a} \tag{4}$$

where $h_{i,\,t,\,a}$ is the mean height of primary school children in prefecture i measured in year t at age a. Our primary variables of interest are the infant mortality rate ($d_{i,\,t}$) and the child mortality rate between ages one and five ($cd_{i,\,t}$). $\boldsymbol{x}'_{i,t}$ is a vector of prefecture and measured year variant controls including those accounting for environment (annual average temperature), family size (crude birth rate), health infrastructure (number of water taps per 100 people, density of doctors, midwives and hospitals) and income and nutrition (rice yields, soy yields and milk production per capita). To control for conditions in the first year of life that would have been the same for all children born in a given year and prefecture, we include a fixed effect for each prefecture-birth cohort ($\nu_{i,\,t-a}$). We also include age fixed effects (ϕ_a) to capture the differences in height at various ages.[4]

The intuition behind Eq. 4 is that there is a typical national growth curve between ages six and eleven captured by the age fixed effects. This national growth curve is shifted up or down by the prefecture-birth cohort fixed effects controlling for conditions the cohort faced between conception and age eleven. We identify off deviations in each prefecture-birth cohort growth curve (adjusted up or down) from the national growth curve. Thus, these regressions capture how changes in the health environment shift a prefecture-birth cohort off the national trajectory. Note that there are no selection issues related to the mortality variables because the deaths do not relate to children in the same cohort as the height data. For instance, we are looking at the effect of the infant mortality rate of infants born in 1929 on the heights of children age 6 in 1929 who were born in 1923. The same is true for the child mortality rate. Thus, these variables are a proxy for the level of child morbidity that could shape children's immediate growth. There is greater potential for endogeneity in these regressions since we are looking at variables measured in the same year. However, we focus principally on the instantaneous influence of infant and child mortality. As mentioned above, our specifications are more precise and include more controls than the existing literature, limiting omitted variable bias. However, there would be potential for omitted variable bias if an unobservable confounder trending with respect to age simultaneously caused infant mortality to decline and height to increase.

4. Results

4.1. Results of SITAR models

Before presenting results from the regressions, we must first discuss the SITAR models and how well they performed in summarising the growth pattern of the prefecture cohorts. In general, the models performed well. Table 1 presents the standard deviations for each

[4] We could include the water tap and child mortality variables in these instantaneous height regressions and not in the birth conditions regressions presented above because these data were only available from the mid 1920s onward.

Table 1
Summaries of SITAR models for boys and girls.

	Boys			Girls		
		Correlations			Correlations	
	Standard deviations	Size	Tempo	Standard deviationsK	Size	Tempo
Size (cm)	0.828			0.822		
Tempo (years)	0.235	−0.117		0.318	−0.099	
Velocity (fractional)	0.031	0.245	−0.990	0.035	0.244	−0.917
Residual	0.790			0.713		
Degrees of freedom	5			4		
BIC of model	22782.74			19883.53		

Notes: SITAR models were estimated with varying degrees of freedom. We used the degrees of freedom that provided the lowest BIC score in line with Cole et al. (2010) as our baseline reported here.
Sources: See Web Appendix B and Schneider and Ogasawara (2018). Statistical citations: R Core Team (2016) and Cole (2016).

of the three SITAR parameters along with standard deviation of the residuals. The standard deviation of the residuals can be taken as a measure of the quality of fit of the model. At between 7 and 8 mm, the residual standard deviations are similar to other height datasets analysed using SITAR suggesting a good fit (Cole et al., 2010). The standard deviations of the three SITAR parameters are substantially lower than those estimated for individual-level datasets because our cohort growth curves are missing individual-level variation. We see that size and tempo are negatively correlated and size and velocity are positively correlated, though these correlations are fairly low. There is a strong negative correlation between tempo and velocity, which suggests that these two parameters were highly related in the model and should not be taken as entirely independent of one another. The models were estimated with varying levels of degrees of freedom taking the estimations with the lowest BIC as the best fit. Thus, the SITAR models did a good job of describing the growth of Japanese children, and therefore parameters predicted from the model can be used in subsequent regressions to describe the growth pattern of the children.

The predicted SITAR parameters for primary school boys and girls in each prefecture and birth year are presented in Appendix Fig. A4. We have excluded the SITAR parameters for the first and last birth cohort, 1916 and 1933, because the parameters are estimated from only one height at one age making them unreliable. The size and velocity parameters tended to increase over time whereas the tempo parameter fell in line with our expectations of the change in the growth pattern during the secular increase in height. However, in general it is clear that there is enough annual variation across prefectures to be able to test the influence of the health environment at birth on the growth pattern of children.

4.2. Results of early life health conditions on the growth pattern

Table 2 presents the results for the estimation of Eq. (2) above showing the influence of disease environment at birth on the growth pattern measured through the SITAR parameters. Columns 1–3 show each SITAR parameter, size, tempo and velocity, for boys and 4–6 the same for girls. We do find some statistically significant effects, especially for boys, and interestingly these follow a slightly counterintuitive but consistent pattern. Remember from Fig. 3 that we know the growth pattern of children has changed in three ways across the twentieth century: height has increased (size); the timing of the pubertal growth spurt has occurred at earlier ages (tempo); and the velocity of growth has increased at most ages and especially in puberty. Thus, in the long run improving health conditions led size and velocity to increase and tempo to decrease. However, for the significant coefficients in Table 2 taking infant mortality as an indicative example, we see that decline in infant mortality increases the size component of the growth pattern as expected but unexpectedly leads to a later pubertal growth spurt and lower velocity going against the long run trends observed across the century. This relationship holds for infant mortality, average temperature, urbanization and soy yields. Although this pattern does not match the long run trends, it does match the expected response that an individual would have to poor conditions *in utero* and in early life under the predictive adaptive responses theory of human development (Gluckman and Hanson, 2006; Schneider, 2017a). Natural selection would lead children exposed to poor conditions *in utero* to be shorter to reduce the physiological costs of being alive, but it would also lead to faster maturation with earlier puberty and faster growth so that the child could reproduce as quickly as possible. Thus, we take our results as tentative support for this theory.

However, one must be careful not to over-interpret these results, which are not all stable across different specifications or precisely identified (see Appendix Tables A3 and A4). Most of these effects seem to be driven by annual shocks because all but the effects for urbanization and soy yields for boys disappear when including birth year fixed effects. The magnitudes of the coefficients are also quite small when looking at a standard deviation change in each variable. For instance, a one standard deviation decrease in the infant mortality rate, a decrease of 33.67, leads to a 0.06 cm increase in the SITAR size parameter. This is small relative to estimates in other articles and compared to results presented later in this paper. This lack of strong effects holds if we replace the SITAR size parameter with simply the heights of children at age 6 or 10 and run the same specification (see Appendix Table A5). Thus, we do not believe that our weak results stem from problems relating to the fitted SITAR models. On the whole, Table 2 suggests one of two possibilities: either conditions *in utero* and in infancy did not have a strong influence on the growth pattern of children in interwar Japan or the selection and scarring effects balanced each other out leaving a null effect on the growth pattern of survivors.

Table 2
Regression of birth health conditions on the growth pattern (SITAR parameters).

	(1)	(2)	(3)	(4)	(5)	(6)
Sex	Boys	Boys	Boys	Girls	Girls	Girls
Dep. var.K	Size	Tempo	Velocity	Size	Tempo	Velocity
Infant mortality rate	−0.002**	−0.001*	0.008*	−0.001	−0.000	0.005
	(0.001)	(0.000)	(0.005)	(0.001)	(0.000)	(0.005)
Average temperature	−0.080***	−0.012	0.119	−0.063***	−0.035***	0.541***
	(0.024)	(0.009)	(0.116)	(0.017)	(0.009)	(0.108)
Urbanization	−0.008*	−0.003**	0.033**	−0.000	0.002	−0.016
	(0.004)	(0.001)	(0.014)	(0.002)	(0.001)	(0.016)
Rice yield	−0.001	−0.002	0.023	0.003**	−0.000	0.001
	(0.004)	(0.002)	(0.018)	(0.001)	(0.001)	(0.009)
Soy yield	0.013*	0.007*	−0.080*	−0.003	0.002	−0.023
	(0.007)	(0.003)	(0.043)	(0.007)	(0.003)	(0.042)
Milk production per capita	−0.021	0.001	−0.027	0.008	−0.005	0.058
	(0.018)	(0.012)	(0.150)	(0.016)	(0.009)	(0.116)
Doctors per 100 people	−1.040	−0.083	0.529	2.452	1.201	−11.614
	(2.225)	(1.019)	(12.443)	(1.793)	(0.993)	(13.275)
Midwives per 100 people	−1.421	0.116	−2.170	−2.250	0.390	−3.683
	(2.497)	(1.110)	(13.597)	(1.749)	(0.993)	(12.485)
Hospitals per 100 people	−10.496	−5.284	62.993	24.417	−8.612*	83.020
	(19.166)	(4.464)	(50.376)	(14.587)	(4.324)	(67.644)
Prefecture fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Prefecture-specific time trend	Yes	Yes	Yes	Yes	Yes	Yes
N	751	751	751	751	751	751
Adj. R-sq	0.8160	0.6913	0.7554	0.8536	0.8061	0.7330

Notes: Standard errors in parentheses clustered at the prefecture level.
* denotes significance at the 10% level.
** denotes significance at the 5% level.
*** denotes significance at the 1% level.
Sources: See Web Appendix B and Schneider and Ogasawara (2018).

We extend the analysis in Table 2 by estimating Eq. (3) above, which allows the infant mortality rate variable to shift from two years before birth to eight years after birth while holding all other variables at the birth year. Again, here we take the infant mortality rate as a good proxy for diseases that are likely to lead to scarring from chronic illness in young children and will lead to minimal selection. Appendix Table A6 presents the coefficients on the infant mortality variables in these various specifications. The infant mortality rate never had an economically meaningful influence on any aspect of the growth pattern in any of the lags or leads. In addition, the coefficients did not have a consistent sign in the years following the birth year, suggesting that taking an average of multiple years would be unlikely to produce a significant result. This result is incongruent with Hatton's earlier work that showed that the infant mortality rate between ages 2 and 4 strongly and significantly reduced children's heights at later ages (Hatton, 2011). Again, to test whether our results are being driven by potential problems with the SITAR models, we ran the same specification using the heights of children at age 6 and age 10 as the dependent variable in separate specifications. These specifications more accurately reproduce Hatton's estimation strategy for Britain. The results are reported in Appendix Table A7 and again show no significant relationship between the infant mortality rate in the years surrounding birth and heights at later ages for either boys or girls. We also include the average infant mortality from ages 2 to 4, Hatton's preferred variable, in our regressions and do not find a significant effect. Thus, it appears that the scarring effects of exposure to a poor disease environment in early childhood, proxied by infant mortality, that Hatton (2011) and Bailey et al. (2016a) found to be so prominent in late nineteenth and early twentieth century Britain were not as important in interwar Japan. Our results more closely align with the small effects found in Sweden by Öberg (2015).

4.3. Results of instantaneous influences on growth

If health conditions in the birth year do not strongly influence the growth pattern, the opposite is true of the instantaneous effects of childhood morbidity, proxied by infant mortality, on children's heights aged 6–11. Table 3 presents the baseline results estimated from Eq. 4. The infant mortality rate had a strong, negative effect on the heights of children with a one standard deviation increase in infant mortality (23.9 deaths per thousand live births) leading to a decrease in height of 0.12 and 0.22 cm for boys and girls respectively. This may seem like a small amount, but these effects would accumulate as children aged, leading to sharp reductions in height. These coefficients are also 2.5–9 times as big as those found on infant mortality in the year of birth. On the other hand, we do not find a significant instantaneous effect of the child mortality rate aged 1–5 on children's heights. The child mortality rate is never statistically significant in any of our specifications and the size of its effect on height is also an order of magnitude lower than that of the infant mortality rate. This result seems to confirm that the infant mortality rate is a better proxy for chronic morbidity in childhood than the child mortality rate. Finally, we find a negative relationship between the average temperature in a year and the

Table 3
Baseline estimation of instantaneous influences on child height ages 6–11 measured 1929–38.

	(1) Boys	(2) Girls	(3) Boys	(4) Girls
Infant mortality rate	−0.005**	−0.009***	−0.007**	−0.016***
	(0.002)	(0.002)	(0.003)	(0.003)
Child mortality rate	0.002	−0.007	0.006	0.007
	(0.006)	(0.007)	(0.006)	(0.007)
Average temperature	−0.087**	−0.112**	−0.090**	−0.129**
	(0.042)	(0.051)	(0.045)	(0.051)
Crude birth rate			−0.013	−0.052***
			(0.017)	(0.016)
Milk production per capita			0.035	0.065***
			(0.031)	(0.023)
Coverage of tap water			0.077***	0.066**
			(0.029)	(0.029)
Doctors per 100 people			7.799***	6.615**
			(2.990)	(3.026)
Additional controls	No	No	Yes	Yes
Age fixed effects	Yes	Yes	Yes	Yes
Prefecture-birth year FE	Yes	Yes	Yes	Yes
N	1645	1645	1645	1645
Adj. R-sq	0.9966	0.9955	0.9966	0.9956
F-statistic	2.599*	11.198***	3.076***	7.042***

Notes: Standard errors (in parentheses) are clustered at the prefecture-birth year level.
* denotes significance at the 10% level.
** denotes significance at the 5% level.
*** denotes significance at the 1% level.
Additional controls include rice yield, soy yield, coverage of midwives, and coverage of hospitals.
Sources: See Web Appendix B and Schneider and Ogasawara (2018).

average height with a one standard deviation increase in average temperature reducing height by 0.20 and 0.26 cm for boys and girls respectively. Interestingly, girls are more responsive to both of these variables. One might be concerned about overfitting in these regressions since the adjusted R-squares are so high. However, the source of the high R-squares is not the prefecture-birth cohort fixed effects but rather the age fixed effects because age is such a powerful predictor of height.

Specifications 3 and 4 of Table 3 introduce additional controls but the coefficients on infant mortality rates and average temperature remain largely unchanged. The controls also provide some interesting but tentative secondary results. Family size is associated with girls' heights with more births leading to shorter girls. Girls also benefited in regions with higher milk production per capita. Both boys' and girls' heights were positively associated with the coverage of tap water in their prefecture and the number of doctors per hundred people, suggesting that health infrastructure could help improve children's heights as well. To test the gender differences in the effects, we also ran a regression pooling both boys and girls and interacting all variables with a girl dummy. The results suggest that gender differences in the coefficients are only statistically significant for the infant mortality rate (not reported).

All of these results are also robust to a host of checks to account for internal migration and wage differences. Internal migration in our period was not large relative to internal migration in later periods in Japan or in other countries (Nakagawa, 2001, pp. 38–45). Only Tokyo and Osaka had net migration rates greater than 2% per annum (Fukao et al., 2015, pp. 113–15). However, we test the potential importance of these net migration effects with two tests: we excluded Tokyo and Osaka from our analysis, and we clustered the standard errors around 16 larger macro-regions to capture urban-rural migration within regions. These checks did not alter our main results (not reported). In addition, although we have many proxies for income included as controls, we included the regional wage data produced by Saito (2005, 2006) to see whether adding this income measure influenced our results. Unfortunately, Saito's data is not reported at the prefecture level, so we could only include wages for seven regions. When including these wages in the instantaneous growth regressions, the coefficients on wages were highly insignificant and equal to zero (not reported).

In addition, we added a number of other types of mortality to these instantaneous regressions to see whether the infant mortality variable was merely capturing a sum of mortality from other causes. Unfortunately, these cause-specific mortality rates contain deaths at all ages, so we could not specifically look at infant or child mortality by cause. The diseases we included were the fetal death rate and deaths per thousand population from congenital infirmity, typhoid, diarrhoea, tuberculosis, beri beri, measles and whooping cough. The results are presented in Appendix Table A8. Including the additional mortality variables only strengthens the effect of infant mortality rather than diminishing it. All of the other mortality variables were statistically insignificant. These regressions confirm that the chronic morbidity proxied by the infant mortality rate is not highly correlated with the mortality rates from infectious diseases or deaths from these chronic diseases, i.e. the diarrhoeal death rate. Thus, chronic diseases seem to be more important in altering children's growth than epidemic diseases.

5. Discussion

The most puzzling finding in our results was that conditions in the year of birth did not seem to be as important in determining growth and the growth pattern at later ages as instantaneous conditions. It is incongruent with a large part of the existing historical literature (c.f. Baten and Murray, 2000), it goes against the emphasis on health conditions in the first thousand days in the development and paediatrics literature (Victora et al., 2010), and it also contradicts Ogasawara (2017)'s finding that children *in utero* and born during the 1918–20 flu pandemic were shorter later in childhood than cohorts born before or after the pandemic. Thus, it is worth considering the robustness of our results, why our results differ from other historical studies and finally their external validity to be applied in other contexts. We will deal with each of these in turn.

To begin, our result that the disease environment in the thousand-day critical window had only a very small influence on subsequent growth seems fairly robust. We tried a number of specifications with various controls, fixed effects, clustering, etc., and we never produced a specification that produced significant and economically meaningful coefficients. Even if we happened upon one, the instability of the results would be very troubling. Prefecture-specific time trends also do not drive our results because the effects are similar if we only include a national time trend (not reported). As mentioned above, we tried using the heights of children at various ages rather than the SITAR parameters to ensure that error in the SITAR parameters was not driving our results. We also included various leads and lags on the infant mortality rate to see if it mattered more in years after the birth year than in the birth year itself. Thus, our null result appears to be stable.

Our results could differ from the existing historical literature for a number of reasons. First, our regression specifications are far more precise than many of the existing studies. We use prefecture-level data and relate children's growth to the annual infant mortality rate in the year of their birth rather than using five- or ten-year averages as Hatton (2014), Baten and Blum (2014) and Bailey et al. (2016a) do. It is possible that in these less precise specifications, the infant mortality rate incorporates aspects of a locality other than the exact conditions that are leading infant mortality to rise or fall. Or it may be that the infant mortality rate over a five- or ten-year birth cohort is actually capturing the cumulative effects of the instantaneous influence of childhood morbidity on growth rather than an early life effect, though this would not be true of Hatton (2011) which uses annual data. The strong effect of infant mortality on child height that Hatton (2011) finds in 40 cities across Britain may also be stronger because of the urban focus which may not represent the effect in the surrounding countryside. None of these issues invalidate these earlier studies, but these slight differences in study design may influence the results.

Our own study has some limitations as well. Our prefecture aggregate data hide a tremendous amount of individual-level and sub-prefecture-level variation in health conditions and growth. There were also small differences in the enrolment rate of primary school children in different prefectures over time, though these enrolment rates were always above 90% between 1929 and 1939 and including the enrolment rate as a control in the regressions above did not influence our results (not reported). We also lack an identification strategy that would allow us to fully mitigate concerns of omitted variable bias. These potential flaws are not unique to our data or empirical strategy though. None of the earlier studies in this literature fully accounted for endogeneity concerns, and Hatton (2011) and (2014) also use aggregated data with representativeness issues and find strong results, so the weaknesses with our study cannot solely explain our results.

Finally, it is important to consider how relevant our findings for Japan are to historical comparisons with other parts of the world and to a wider discussion of influences on children's growth today. One important threat to external validity would be if Japanese children's growth was different from children's growth in other parts of the world. Eveleth and Tanner (1976, 1990) analysed the growth patterns of children in various parts of the world and found that East Asian children, including the Japanese, had a slightly different growth pattern than Africans and Europeans, experiencing earlier maturation, shorter adult stature and shorter leg lengths. This is born out in Fig. 3 above, which shows that the secular increase in stature has largely stopped since the 1980s despite other aspects of health continuing to improve (Bassino and Kato, 2010). However, despite these minor differences, Japanese children experienced similar changes in the growth pattern over the twentieth century as European children including a comparable or larger increase in mean adult stature. Thus, it is difficult to argue that our findings are purely driven by relatively minor differences in the growth pattern of Asian children, especially since the instantaneous effects are so strong. In addition, as mentioned above, there were no substantial differences in the level, rate of decline or causes of infant death between Britain and Japan (see Web Appendix D).

In conclusion, we cannot easily dismiss our finding that although infant mortality mattered for instantaneous growth, it did not have strong scarring effects in early life as found in other countries. Our estimation strategy is more precise than others employed in the literature. In addition, differences in the growth pattern and disease environment between Japan and other countries cannot explain away our results. In the end, it seems that in Japan year-to-year conditions even outside of the critical window of the first thousand days played a more important role in shaping children's growth than their initial conditions at birth.

6. Conclusion

This paper has tested the influence of childhood morbidity, proxied through the infant mortality rate, on the growth of children in interwar Japan. It is the first paper to use the SITAR growth model to parameterise the growth pattern of historical children and attempt to explain the parameters. We found that in Japan the infant mortality rate and other health conditions around the time of birth did not have a strong influence on the growth pattern of children from age six onwards. This is a striking contrast to recent literature that has highlighted the importance of conditions in the first thousand days of life (Victora et al., 2010) and found strong scarring effects of exposure to higher childhood morbidity in early life on later growth in historical Britain and Europe (Hatton, 2011; Hatton, 2014; Bailey et al., 2016a). However, infant mortality did have an important instantaneous effect on children's growth in

Japan, suggesting that annual variation in health conditions across the growing years may have been more important in shaping the growth pattern than early-life scarring contrary to assumptions about the thousand-day critical window.

These results are important in two respects. First, they help provide context to explain the secular increase in height in Japan and elsewhere. The results suggest that reductions in child morbidity were important for increasing stature during the interwar period in Japan. We also have tentative evidence that the expansion of clean water and other health infrastructure could improve child health; there is substantial evidence that the rollout of sanitation and clean water and the introduction of social workers who gave poorer people access to medical treatment reduced mortality in interwar Tokyo (Ogasawara and Kobayashi, 2015; Ogasawara et al., 2018). However, most of the income and nutrition related variables were insignificant, suggesting that the disease environment may have been more important for the secular increase in height in interwar Japan than nutrition. This balance of disease versus nutrition may have been different after the Second World War, though, when economic growth increased and milk consumption expanded (Takahashi, 1984). Our results also suggest that health conditions in early life were not good predictors of the growth pattern of children or simply their heights at later ages. Thus, we find that children's growth was plastic across the growing years with children catching up or falling behind relative to modern standards as they developed and grew based on the conditions they faced each year. The observed cohort growth pattern, then, was the cumulative effect of these annual fluctuations in health conditions rather than a predetermined pattern set *in utero* and in early life. This view of the secular increase in height places a greater emphasis on growth in childhood and adolescence. Essentially, we find that the period effects on growth are stronger than the cohort ones.

Second, the strong instantaneous effects of infant mortality on children's heights at ages 6–11 present challenges to the growing orthodoxy in the modern development literature that places enormous importance on growth in the first thousand days of life (Victora et al., 2010). Our evidence from interwar Japan shows that conditions outside of this early life critical window can influence children's growth and may be more important for their completed growth pattern. This concurs with evidence from the Consortium of Health Oriented Research in Transitioning Societies (COHORTS) longitudinal studies that showed that catch-up growth was possible between 24 months and mid-childhood and between mid-childhood and adulthood (Prentice et al., 2013). This matters for the current development debate on child stunting for two reasons. First, it highlights that, at least as far as growth is concerned, interventions outside the thousand-day critical window can influence children's growth pattern. Thus, it may be possible to design interventions to help the 155 million children currently stunted in the world today. However, we are cautious about this finding since studies have found that growth faltering can have detrimental cognitive effects for children, and we cannot measure the cognitive effects of interventions at later ages (Hoddinott et al., 2013; Schneider, 2018b). Second, if one takes a long-run perspective on reducing child stunting, the goal is in effect to create the conditions that led to the historical secular increase in height. Recent evidence on birth weights (Schneider, 2017b) suggests that birth weights have not changed very much over the past one hundred years in Western Europe and North America. Thus, the secular increase was likely created by a combination of reducing growth faltering early in life and by strengthening positive interventions later in childhood and adolescence that could make up for slower growth at earlier ages.

Finally, our paper raises several important issues for future research. It highlights the importance of child morbidity for child growth but unfortunately was not able to test precisely how exposure to any given type of illness influenced children's growth. Infant mortality represents a fairly wide range of potential exposures so more detailed research on particular diseases would be helpful. In addition, more studies are needed that focus more holistically on the growth pattern rather than looking at height in very limited windows during childhood or at adulthood. It is possible that the growth faltering that we observe at early ages historically was simply the product of a growth pattern with a lower velocity and later pubertal growth spurt than that experienced by modern children. The only way to check this definitively is to try to understand how the growth pattern from early childhood to adulthood has changed over time. This will involve returning to archives and searching for new sources of data that cover children's growth over a wider range of ages since most of the existing sources on child growth are too limited to capture the full growth pattern. However, understanding how the growth pattern of children shifted over time is fundamental to understanding the causes of the secular increase in height.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.eeh.2018.05.001.

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