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HEALTH-LED GROWTH SINCE 1800

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This paper argues that the marked reduction in the burden of parasitic and infectious diseases (PID) together with improved nutrition over the past two centuries in today's advanced countries has resulted in markedly improved physiological capital and cognitive skills and, consequently, in productivity advances. Using a unique annual dataset covering the period 1800–2011 for 21 OECD (Organisation for Economic Co-operation and Development) countries, it is found that health improvements can account for approximately a third of the productivity advances in the OECD countries since 1865, and that these improvements have been influential for enhancement in education, savings, innovations, life expectancy, and democracy.

Keywords: Great Demographic Transition, Physiological Capital, IQ, Productivity Growth

1. INTRODUCTION

Empirically, the nexus between health and productivity growth has been highly controversial. Using life expectancy at birth as an indicator of health, Knowles and Owen (1997), Arora (2001), and Soares (2005) find a positive time-series relationship between growth and life expectancy. By contrast, Acemoglu and Johnson (2007) fail to find any positive relationship between increased life expectancy and per capita growth using cross-sectional changes in life expectancy over the period 1940–1980, and they suggest that increased life expectancy may even reduce growth due to the capital dilution effects induced by population growth. Using the same sample as Acemoglu and Johnson (2007), Aghion et al. (2011) and Bloom et al. (2014) find that the result of Acemoglu and Johnson (2007) is reversed once life expectancy at the start of the estimation period (1940) is controlled for in the regressions. They argue that initial life expectancy is an important regressor because life expectancy has been converging across countries over the period 1940–1980. Controlling for country-specific effects, Hansen and Lønstrup (2015) show that the original results of Acemoglu and Johnson (2007) hold, even when

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initial life expectancy and per capita income are controlled for. Similarly, using a cross-state variation in infectious causes of death and a time-variation in medical innovations for the US states, Hansen (2014) finds that life expectancy has no effect on per capita GDP, even when initial life expectancy is controlled for.

This paper examines the growth effects of health following two often neglected channels through which health can potentially influence income growth. First, parasitic and infectious diseases (PIDs) and lack of micronutrients in early childhood or while the child is in utero may permanently impair the child's cognitive development and, consequently, have potential adverse productivity effects through the channels of education, ideas production, savings, and work efficiency. Recent medical research shows that health in utero and in early childhood is decisive for cognitive ability during adulthood [see, e.g., Jardim-Botelho et al. (2008)], which, in turn, is important for productivity. Since most of the brain's development occurs from half way through pregnancy until the child reaches the age of 2, this period is by far the most important for cognitive development, in particular, and health capital, in general [Niehaus et al. (2002)]. Adequate development of the brain during infancy require sufficient supply of energy, essential micronutrition, and oxygen, noting that the brain of a newborn baby claims at least 87% of the body's energy budget [Holliday (1986)].

Second, Robert Fogel has long argued that protein-energy malnutrition in the centuries up to the mid-20th century in today's advanced countries significantly reduced per capita income growth, and he calculates that improved gross nutrition has been responsible for 30% of the per capita income growth in Britain over the period 1790–1980 [Fogel (2004)]. What is often forgotten nowadays is that a large fraction of the population in today's advanced countries suffered severely from malnutrition during the 19th century and that malnutrition prevailed for a significant proportion of the population all the way up to the mid-20th century [Fogel (2004), Harris et al. (2014)].

The contribution of this paper to the literature is three-fold. First, it examines the influence of cohort-health-induced cognitive skills and physiological capital on productivity growth and the extent to which these can help explain the transition from the low-growth post-Malthusian era during the 19th century to the modern growth regime that gained momentum during the 20th century. Thus far, little, if any, empirical investigation has been undertaken to identify the influence of malnutrition cohort-health-induced cognitive skills on economic growth. Second, this paper investigates savings, innovations, democracy, education, working-age mortality, and fertility as potential channels through which health during the growing years of life influences productivity during adulthood.

Third, annual mortality data and several other variables are constructed for 21 OECD (Organisation for Economic Co-operation and Development) countries over the period 1800–2011. Cohort infant mortality and cohort height indicators of the working-age population are constructed as proxies for development in cognitive and physiological capital from fetal life up to maturity and real food prices, the fraction of GDP that is spent on public infrastructure and significant

medical innovations in the world are used as instruments for these two cohort variables. In Section 4, it is argued that cohort infant mortality is an excellent indicator of the burden of PIDs, protein-energy malnutrition, lead poisoning, and mineral deficiency in utero and in early childhood, and that cohort height is a good indicator of physiological development, where cohort infant mortality is computed as the infant mortality experienced by the working-age population at infanthood, and cohort height is the weighted average height of the working-age population.

The regression results in this paper indicate that improved cohort health has been a significant determinant of growth in today's advanced countries and can explain more than a third of the productivity advances in the OECD countries over the period 1870–2011. This paper proceeds as follows. The next section briefly surveys the literature and, in Section 3, it is argued that a high burden of PIDs, malnutrition, iron and iodine deficiencies, and lead poisoning among infants and in utero potentially impair cognitive ability and the physiological capital significantly. Productivity growth regressions are carried out in Sections 4 and 5, and Section 6 tests for potential channels through which health insults during early life until maturity are transmitted to growth. Section 7 discusses the implications of the findings and concludes this paper.

2. RELATED LITERATURE

The economic consequences of marked improvements in life expectancy and the associated health improvements since the mid-19th century have long been debated in the literature. The literature has focused on (1) the growth effects of the increasing life expectancy as a summary measure of the health status, as briefly discussed in the Introduction, and (2) the potential channels through which health may influence productivity. Studies show that improved health can have potentially positive growth effects through the following channels: (1) increasing the present value of schooling and, as such, inducing young people to invest more in human capital [Chakraborty (2004), Zhang and Zhang (2005)]; (2) higher school and work attendance rates due to fewer sick days [Weil (2007)]; (3) improving the alertness and work intensity of students at school and workers in the workforce [Fogel (1994, 2004), Weil (2007)]; (4) reduced fertility rates [Kalemli-Ozcan (2002), Iyigun (2005), Soares (2005), Zhang and Zhang (2005), Lorentzen et al. (2008), Aksan and Chakraborty (2013, 2014)]; (5) enhanced savings [Chakraborty (2004), Zhang and Zhang (2005)]; (6) increasing returns to investment in human capital [Chakraborty (2004), Zhang and Zhang (2005), Lorentzen et al. (2008), Aksan and Chakraborty (2013, 2014)]; and (7) improved quality of learning [Madsen (2016b)].

This paper is most closely related to Madsen (2016b) in which it is argued that, during the school years and in adulthood, disease impairs the quality of learning and the ability to create new ideas. Specifically, Madsen (2016b) argues that, during the ages at which pupils take their education, health is influential for the quality of education because illness may lead to cognitive impairment, stigma, and

reduced concentration in the classroom, not to mention the fact that illness leads to increased rates of absenteeism and lower rates of school enrollment. Deriving a health-adjusted educational attainment measure of the working-age population based on their health status during the time they did their education, Madsen (2016b) shows that health improvements have enhanced the quantity and quality of schooling and innovation over the past 140 years in the OECD countries. The principal difference between Madsen (2016b) and this paper is that this paper focusses on disease-induced cognitive impairment in early life, whereas Madsen (2016b) focused on the productivity effects of the *interaction* between disease during school age and education and the *interaction* between disease in adulthood and ideas production.

3. THE IMPACT OF HEALTH IN EARLY LIFE AND COGNITIVE AND PHYSIOLOGICAL DEVELOPMENT

In most industrialized countries, the high burden of PIDs and protein-energy malnutrition in childhood before World War I had the potential to significantly impair the cognitive function and the physiological development of much of the population. Cognitive function was further impaired by essential mineral deficiencies, most importantly iron and iodine deficiencies, and the widespread use of lead pipes for water supply in big cities. This section discusses how persistent PIDs and micronutrient malnutrition during infancy may permanently impair cognitive development and how protein-energy malnutrition before maturity has been reached in the early-20's impairs physiological capital. The distinction between cognitive impairment and physiological damage is somewhat expositional since diseases and malnutrition impinge on physiological capital as well as cognitive development, as discussed in the following sections.

3.1. Health and Cognitive Development

Health insults in utero and in early childhood can significantly and irreversibly reduce the cognitive development of individuals. The cognitive development of very young children is highly sensitive to the presence of PIDs because their brains compete fiercely with parasites and the immune system over the energy supply. PIDs take the energy needed for a child's developing brain, and some also cause anemia, which can also affect brain development. As the brain absorbs 87% of the body's metabolic budget in newborn infants, 44% at the age of 5, and 20% in adulthood, cognitive development is substantially more sensitive to energy and oxygen supplies in early periods of childhood than later on in life [Holliday (1986)].

Several studies have shown that PIDs significantly weaken the cognitive ability and development of children [see Jardim-Botelho et al. (2008) for an overview]. Surveying more than 150 scientific papers on the effects of PIDs on cognitive disorders, Watkins and Pollitt (1997) find almost unanimous support for the hypothesis that most PIDs have the potential to impair cognitive ability and that the

burden of PIDs peaks at a very young age. The PIDs that are most influential for cognitive development are helminth, malaria, cholera, and typhoid, diseases that were widespread in the industrial countries until the mid-20th century. Although malaria hardly exists in the OECD countries today, it was widespread before it was eradicated in the United States and in the Mediterranean countries over the period 1930–1950 and earlier in other European countries [Huldén et al. (2005)]. In the US southern states, four million cases were reported every year in the 1930s and 1940s [Dobson and Carper (1996)]. Malaria existed in Western Europe and Japan during the 18th century, and in the 19th century, it was even common in the north and north-east of Europe [Huldén et al. (2005)]. Malaria in infants and young children causes widespread anemia during the period of rapid brain development and it can also directly cause brain damage [Snow et al. (2005)].

Iron and iodine deficiencies during infancy and, particularly, in utero are highly influential for the cognitive development of children. Several studies identify impaired cognitive abilities among children born to mothers who had iodine and/or iron deficiencies during their pregnancy [Scrimshaw (1998)]. Based on meta-analysis of 18 studies, Bleichrodt and Born (1994) find that iodine- and non-iodine-deficient groups were 13.5 IQ points apart. Iron-deficiency anemia results in cognitive deterioration and the alternation of neurological functions.

Waber et al. (2014) find strong support for the hypothesis that malnutrition among young children impairs their cognitive ability significantly and, often, potentially permanently by reducing the number of brain cells. Several studies find that IQ can be reduced by approximately 10 points in children who have been exposed to longer spells of malnutrition during their childhood [for a survey, see Scrimshaw (1998)]. Furthermore, the brain damage induced by malnutrition leads to behavioral problems among children and poor relationships with their peers, short attention spans, distractibility in the class room, irritability, apathy, and a lack of interest in subject topics [Holding and Snow (2001)].

As is well known today, lead is toxic and leads to impaired cognitive ability, fatigue, increased blood pressure, impaired neurological and behavioral function among adults, and a decrease in gross and fine motor skills [see, for a survey, Shih et al. (2007)]. Clay et al. (2010) find that in cities with lead-only water pipes, average wages were about 6% lower for manufacturing workers than in cities with no-lead water pipes, which suggests that lead poisoning had potentially adverse effects on cognitive ability and, consequently, productivity.

3.2. Health and Physiological Development

Fogel (1994, 2002, 2004) argues that protein-energy malnutrition from early life up to maturity results in temporary and, often, permanent physiological dysfunctions such as degradation of tissue structure in the lungs, the heart, and the gastrointestinal tract. Malnutrition is also associated with the mucosal cells of the gut, the inhibition of wound healing, increased likelihood of traumatic shock and of sepsis, impaired functioning of the endocrine system, increased tendency to edema,

electrical instability that can provoke acute arrhythmias, and degenerative joint diseases [Fogel (1994)]. The immediate economic consequences of chronic malnutrition are reduced work intensity and reduced hours worked [Dasgupta (1993)].

Evidence in Fogel (2002) suggests that energy-protein malnutrition was widespread during industrialization in the United Kingdom and France and, presumably, the other countries studied here. In the middle of the 19th century in Britain, the average caloric intake was at or below the average caloric supply of today's low-income countries [Fogel (2002)]. It was not until well into the 20th century that the caloric intake in France and Britain reached levels that are required for a healthy life [Fogel (2002)]. Fogel (2004) estimates that improved nutrition has been responsible for 30% of the British per capita income growth rate over the period 1790–1980 through increasing labor force participation and work intensity. Fogel (2004) argues that the impact of nutrition on long-term economic growth accounts for most of the previously unmeasured increase in British TFP (total factor productivity). He even suggests that his estimates are on the low side because they neglect the indirect educational effects.

As an example of the improvement in physiological capital in the United States due to improved nutrition over the past century, Fogel (2004) estimates that the capacity of Union Army veterans circa 1900 to engage in manual labor by the ages 60–64, had declined to about a third of what they had been at their peak at about the age of 35, which is about 15 years earlier than today. Furthermore, Fogel (2004) finds that the supply of calories per equivalent adult male available for work increased from about 848 per day in 1800 to about 1,793 in 1980 in Britain, reinforcing the very little opportunity for the majority of the population to develop and utilize their physiological capital to its full potential back in 1800.

4. EMPIRICAL ESTIMATES

The preceding section suggests that PIDs, malnutrition, and exposure to lead during early life up to the late teens impair the level of cognitive skills and physiological capital reached in adulthood, which, in turn, adversely affects productivity through channels that are discussed below. This section establishes the basic econometric framework and argues that cohort infant mortality and the average height of the population of working age are good proxies for health insults in infancy and during childhood.

The following model is estimated over the period 1865–2011[1]:

$$\ln(Y/L)_{it} = \alpha_0 + \alpha_1 \Phi_{it}^{IM} + \alpha_2 \Phi_{it}^{H} + Z_{it}\xi' + CD_i + TD_t + TT_{it} + \nu_{it}, \quad (1)$$

where Y is real GDP, L is hours worked (annual hours worked multiplied by employment), Φ^{IM} is cohort infant mortality, Φ^{H} is cohort height, Z is a vector of control variables, ν is a stochastic error term, TD is time dummies, CD is country fixed effect dummies, TT is country-specific time-trends, i is country i, and t is time. The confounding factors contained in the vector Z, which are introduced in

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Section 5.3, are variables that underwent a significant transition at approximately the same time as infant mortality and height and, as such, are key control variables.

Cohort infant mortality and height are the two focus variables proxying for health insults experienced by the working population in utero and during their infanthood (cohort infant mortality) as well as during their early years of life until maturity (height at maturity). They are constructed as follows:

$$\Phi_{it}^{IM} = \left(\sum_{\tau=15}^{64} Pop_{\tau it} IM_{i,t-\tau} \right) / Pop_{it}^{15-64}, \tag{2}$$

$$\Phi_{it}^{H} = \left(\sum_{\tau=15}^{64} Pop_{\tau it} H_{i,t-\tau} \right) / Pop_{it}^{15-64}, \quad \tau = 15,\ 16, \ldots,\ 64, \tag{3}$$

where $Pop_{\tau it}$ is the size of the population for age cohort τ for country i at time t, Pop^{15-64} is the population of working age, $IM_{t-\tau}$ is the infant mortality rate at period $t-\tau$, and $H_{t-\tau}$ is the terminal height reached at maturity of a cohort born in period $t-\tau$. Equation (2) shows that the working population cohort at time t comes from a birth cohort in which the average infant mortality was Φ^{IM} and equation (3) shows that the working population cohort at time t comes from a birth cohort, which reached the terminal height of Φ^{H}. For the 60-year-old cohort, for example, Φ^{IM}/Φ^{H} is the infant mortality/height experienced by the cohort that was born 60 years earlier.

Studies suggest that infant mortality is a good indicator of the burden of health insults during infancy and in utero and that these health insults have the potential to permanently impair cognitive skills[2]. First, Case and Paxson (2009) find that infant mortality in the United States in the first half of the 20th century was significantly and negatively related to the cognitive abilities of the cohorts at older ages. Second, Crimmins and Finch (2006) find infant mortality to be an excellent indicator of inflammation during infanthood. Third, Atkins (1992) and Lee (2007) note that PIDs were the main infant killers before the great demographic transition and that the burden of PIDs was reflected in infant mortality rates. Fourth, infant mortality is significantly and positively related to the incidence of iron and iodine deficiency in utero and during the first months of life [Maberly (1994), Scrimshaw (1998)]. Fifth, anemia among pregnant mothers, which is often caused by iodine and iron deficiency, malnutrition, and PID, leads to low birth weight, which, in turn, increases the risk of infant mortality [Brooker et al. (2008)].

Sixth, McCormick (1985) also finds that low-birth-weight infants have high infant mortalities compared to the normal-weight group, and Lynn (2009) reports studies showing that IQ is 5 points lower among infants with low birth weight compared with the control group. Studies also indicate that low birth weight reduces the strength of the immune system and is associated with poor neurosensory, cognitive, and behavioral development [Holding and Snow (2001)]. Seventh, lead poisoning also increases infant mortality as infants are highly sensitive to lead [Troesken (2008)]. Troesken (2008) estimates that the use of lead water pipes

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increased infant mortality by 25%–50% in the average town in the United States in 1900. Finally, Reidpath and Allotey (2003) find a strong positive relationship between infant mortality and disability-adjusted life expectancy (DALE), where DALE combines information on mortality as well as morbidity associated with non-fatal health outcomes. They estimate a correlation coefficient of 0.91 between the two and conclude that one measure could stand as a proxy for the other and that either is an excellent proxy for population health.

Height is a good indicator for protein-energy nutrition in early life up to the late teens [Hatton (2014)], and Fogel (1994, 2002, 2003, 2004) uses height as one of his most effective instruments in his studies of the development of physiological capital during the past few centuries. Fogel (2003) argues that:

> rapid accumulation of physiological capital is tied both to long-term reductions in environmental hazards and to the conquest of chronic malnutrition (made possible by technophysio evolution), and it is reflected in the improvements in stature and the Body Mass Index, a measure of weight standardized for height. Variations in height and weight are associated with variations in the chemical composition of the tissues that make up vital organs, in the quality of the electrical transmission across membranes, and in the functioning of the endocrine system and other vital systems. Nutritional status, as reflected in height and weight, thus appears to be a critical link connecting improvements in technology to improvements in human physiology. (p. S27)

Laboratory experiments on animals and observational studies of human populations indicate that anthropometric measures such as weight and attained final height are reliable measures of malnutrition, particularly during childhood [Fogel and Wimmer (1992)]. Furthermore, there is robust evidence of height being closely correlated with physical strength. Studying Indian female laborers, Koley et al. (2009) find a significant relationship between the strength of hand grip and height ($p < 0.001$), noting that handgrip strength is necessary for performing activities of daily living, which, in turn, are required to maintain functional autonomy. Forde et al. (2000) find that the weight lifted by champion weight lifters "varied almost exactly with height squared" (p. 1061).

The problem associated with the strategy used here to distinguish PIDs from calorie–protein malnutrition is that medical research is still progressing on causes of height and infant mortality and, particularly, trying to understand which health insults show up in infant mortality and height, respectively. Crimmins and Finch (2006), for example, argue that height is also influenced by inflammation in childhood as chronic infections and the inflammatory response elevate the cortisol level and, potentially, cause growth stunting by impairing the protein synthesis. Furthermore, studying Peruvian children, Checkley et al. (2003) show that children ill with diarrhea 10% of the time during the first 24 months of life were 1.5 centimeters shorter than children who never had diarrhea, suggesting that physiological developments are also influenced by PIDs. Consequently, the distinction between Φ^{IM} and Φ^{H} as capturing cognitive skills and physiological capital of the working-age population can, to some extent, only be considered expositional.

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A potential downside of using infant mortality and stature as indicators of PIDs and malnutrition is that the culling effects may exceed the damaging impacts, the so-called cohort damaging hypothesis in which ambient shocks to gestation may cull infants, leaving behind less frail members of that cohort. However, there is strong empirical evidence suggesting that the damaging effect exceeds the culling effects and, therefore, that infant mortality and height are reliable proxies for the environmental exposure to PIDs and inflammation by the survivors of a cohort [see, for evidence and discussion of the literature, Crimmins and Finch (2006)].

Finally, the model specification given by equation (1) follows the empirical endogenous growth literature in which productivity advances are driven by technological progress, which, in turn, is driven by innovations [see, e.g., Aghion and Howitt (1998); Peretto (2015)]. The stock of foreign knowledge, S^f, is assumed to be transmitted internationally through imports. Cohort infant mortality and height are not measured in logs and, therefore, it is the change in the mortality rate/height, and not the growth in the rate that matters for economic growth, suggesting that the productivity effects of a change in the mortality rate/height are independent of the level of infant mortality/height. This follows the same principle as Mincer's human capital approach in which the returns to an additional year of schooling are independent of the level of education.

4.1. Identification

Cohort infant mortality and cohort height may be exogenous from the perspective that they are determined several years before they affect productivity; however, since they are serially correlated, they may not be strictly exogenous. Furthermore, that Φ^{IM} and Φ^{H} precede $\ln(Y/L)$ need not reveal causality because a third factor may impinge on infant mortality/height and productivity simultaneously, as illustrated by the following examples. First, suppose improvements in a country's institutions start impacting on public health with a disproportionate impact on infants and youngsters; then, with a lag, this affects the quality of education, technology in production, and the efficiency of market allocations. Second, if falling infant mortality triggers a reduction in fertility that influences productivity growth with a lag, then infant mortality may, again, exaggerate the productivity effects of health. Third, omitted variables could have operated with a lag as well, and they could be jointly determined with decreases in mortality. For example, if a country came out of a war at a certain period, and started a process of rebuilding its infrastructure and its institutions at the same time that public health provision returned to normal, then all those effects could be confounded. Indeed, any factor that might impact growth with a similar lag to that assumed for infant health, and which moved in the same general direction as infant mortality, will confound the results.

To address these problems, the following strategies are pursued for identification. First, instruments are used for infant mortality and height to deal with endogeneity and measurement errors. Second, control variables and cohort indicators are included in the regressions to ensure that Φ^{IM} and Φ^{H} are not capturing

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the effects of confounding variables that transitioned at approximately the same time as Φ^{IM} and Φ^{H}. The mortality transition, for example, was associated with an almost simultaneous decline in fertility, the introduction of mass education, institutional improvements, financial liberalization, the industrial revolution, etc., and cohort infant mortality could have captured the effects of these variables on productivity. Cohort institutional quality, fertility, innovations, productivity, credit provision, and literacy are introduced into the regressions in the robustness section to allow for confounding effects.

From the outset, it is important to stress that the instrumental variable strategy pursued here may not fully solve the endogeneity problem because the proposed instruments are not entirely exogenous and, more importantly, the instrumental variable (IV) strategy does not solve the time-varying omitted variable problem. For that reason, IV as well as ordinary least-squares (OLS) regressions will be presented in most of the tables shown below.

The following first-stage regressions are carried out:

$$\Phi_{it}^{X} = \vartheta_0 + \vartheta_1 \ln\Phi_{it}^{Med} + \vartheta_2 \ln\Phi_{it}^{P^f/P} + \vartheta_3 \ln\Phi_{it}^{I^{Infr}/Y} + Z_{it}\zeta' + CD_i + TD_t + TT_{it} + \varepsilon_{2,it}, \quad (4)$$

where $X = (IM, H)$, $\Phi_{it}^{X} = (\Phi_{it}^{IM}, \Phi_{it}^{H})$, *Med* is significant medical innovations, $(P^f/P)_{it}$ is real food prices, $(I^{Infr}/Y)_{it}$ is share of public infrastructure investment in total GDP, Φ_{it}^{Med} is cohort significant medical innovations, $\Phi_{it}^{P^f/P}$ is cohort real food prices, and $\Phi_{it}^{I^{Infr}/Y}$ is the cohort share of public infrastructure investment-to-GDP ratio. The cohort instruments are generated using the same formulae as cohort infant mortality and height [equations (2) and (3)].

Real food prices, P^f/P, act as a potentially good instrument for the following reasons. First, they are influenced by domestic weather conditions, plant and animal diseases, and international food prices. The significant decrease in real food prices over the past four centuries [Harvey et al. (2010)] is evidence that food prices have not been driven by the increasing income and that real food prices are not pro-cyclical, as food demand is relatively independent of the business cycle. Second, real food prices are good proxies for food crises because they signal food shortages and food affordability and, therefore, the energy-nutrition availability for the poor population, which was a large fraction of the population during the First and the Second Industrial Revolutions [Fogel (1994)]. Since increasing real food prices are associated with lower calorie intake, they will automatically increase mortality and delay physical growth that may permanently lead to stunting, as discussed above. A downside of using real food prices as an instrument is that they may not entirely satisfy the exclusion restriction because food prices are, in addition to weather conditions, also influenced by demand factors and productivity growth in agriculture and because weather conditions not only affect food prices, but may also influence the spread of infectious diseases.

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Significant medical innovations, *Med*, collected by Adlington and Humphries (1999), encompass what they consider to be the most important medical innovations in the world and are available annually over the time-period considered in this paper, where the coding year is the innovation year and, therefore, not the year at which the innovation is adopted. They are accumulated in the first-round regressions because each innovation is assumed to permanently lower mortality rates. Each innovation gets the score of 1 as it is impossible to weight the innovations by their influence. Accumulated significant innovations are assumed to affect infant mortality rates and height equally across the countries considered here because they are freely available worldwide and because the evidence suggests that medical innovations are easily diffused across the globe [see, for a discussion, Papageorgiou et al. (2007)]. The Bacteriological Revolution associated with the development of germ theories of diseases, in the late-19th century, for example, is probably the most significant event for infant survival probabilities, and it spread widely and quickly across the industrialized world [Black (1996)]. Furthermore, using data for Swedish counties, Ager et al. (2015) find that the start of vaccination in 1801 together with the introduction of compulsory vaccination in 1816 had profound negative effects on the infant mortality rate.

Public infrastructure investment as a percentage of GDP, I^{Infr}/Y, acts as a proxy for investment in public infrastructure such as hospitals, schools, sewage drainage, clean water supply, central heating and public housing, investments that were major contributors to the mortality transition in the rich countries in the first half of the 20th century [see, e.g., Cutler et al. (2006)]. Public infrastructure investment is exogenous from the perspective that it is determined by policy decisions and there are likely to be few feedback effects from mortality to a government's investment in hospitals, sewage systems, public housing and central heating. The high morbidity during the 19th century acted as an impetus to investment in better health infrastructure; however, the timing of the investment spurt in the beginning of the 20th century was unrelated to changes in mortality as mortality had always been high before the mortality transition and, therefore, it was an ongoing problem. That some countries improved their public health infrastructure while most other countries did not suggests that the decision to make major inroads into the public health infrastructure was independent of the level of morbidity and mortality and determined at the political level. The only downside associated with public infrastructure as an instrument is that it is a component of GDP, suggesting that the exclusion restriction does not hold. However, the violation is not likely to be of any significance since public investment in health infrastructure has historically probably been a minuscule fraction of GDP.

4.2. Data and Graphical Evidence

The data are collected for the following 21 OECD countries over the period 1800–2011: Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Japan, the Netherlands, New Zealand, Norway, Portugal,

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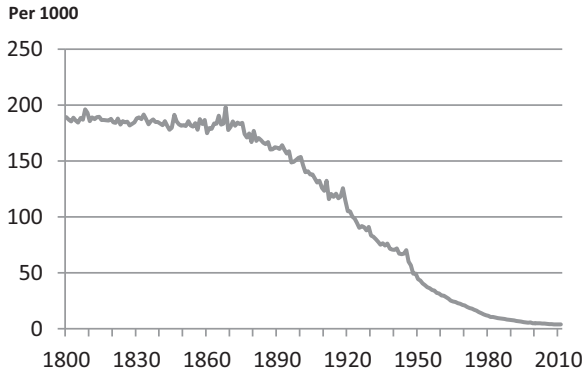

FIGURE 1. Infant mortality, OECD countries.

Notes: The figures are unweighted averages of Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Japan, the Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, the United Kingdom, and the United States. Note that height terminates in 1990 as data are first available with 20 years delay.

Spain, Sweden, Switzerland, the United Kingdom, and the United States. Summary statistics are provided later in this paper (Table 5) after the confounding variables have been introduced. Labor productivity is estimated as gross domestic product (GDP) divided by total employment and annual hours worked, where it is crucial to account for annual hours worked since they have almost halved over the past century. The infant mortality data are based on national sources and go back to around 1840 for most countries and further back in time for about half of the countries. The height data measure the height of fully grown males and are mostly collected from military recruits' files. These data are available from year 1800 for almost all countries in the sample.

The average infant mortality and height in the countries considered here over the last two centuries are displayed in Figures 1 and 2. The height figures refer to the birth year of each age cohort when they have reached their full height, which implies that the data are available with a time-lag of approximately 20 years. The short cycles in infant mortalities are due to the cyclical epidemiology of diseases such as measles (every 2–5 years), whooping cough (every 2–5 years), diphtheria (approximately every 10 years), and influenza (every 2–3 years) [Armstrong et al. (1999)]. The slight increase in height in the first decades of the 19th century is short-lived and the infant mortality and height trends are relatively constant before around 1870. The figures indicate a marked health transition in the period 1870–1950; however, their time-profiles vary somewhat. The strongest absolute decline in infant mortality occurs in the period 1870–1950, whereas the height-growth spurt is concentrated in the period 1885–1960, indicating that the epidemiological transition started earlier than the calorie-protein nutritional transition.

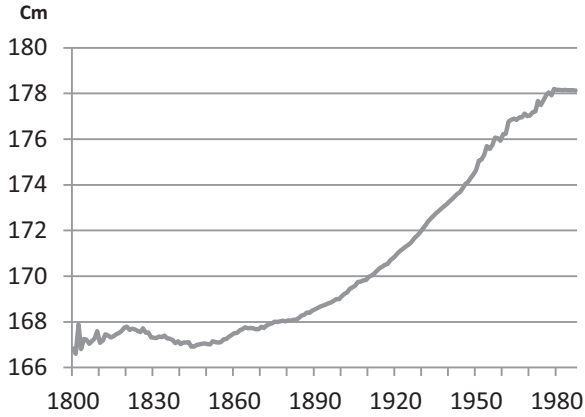

FIGURE 2. Height, OECD countries.

Notes: The figures are unweighted averages of Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Japan, the Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, the United Kingdom, and the United States. Note that height terminates in 1990 as data are first available with 20 years delay.

The reduction in PIDs was particularly influential for the decline in infant mortality during the first half of the 20th century, and infant mortality due to PIDs decreased more than 50% from the end of the 19th century to the mid-20th century[3]. According to Lee (2007), the reduced burden of PIDs among infants was mainly a result of the improved quality of cows' milk and a reduction in malnutrition. Mothers over several centuries have supplemented breastfeeding with cows' milk and other food [Castilho and Barros Filho (2010)]. The fraction of mothers who supplemented milk by bottle feeding infants under the age of 6 months across countries often exceeded 50% around the turn of the 20th century [Lee (2007), Castilho and Barros Filho (2010)]. Consequently, infants were often exposed to diseases such as cholera, polio, anthrax, scarlet fever, bovine tuberculosis, brucellosis, and botulism found in milk [Atkins (1992)]. Furthermore, as the milk was often diluted by contaminated water, other diseases such as salmonella and typhoid were widespread in milk during the 19th century [Atkins (1992)]. The quality of milk improved substantially after the turn of the 20th century in the industrialized countries. In the 1930s, pasteurization was used almost everywhere in the United Kingdom and the United States [Atkins (1992)]. Interestingly, despite a strong decline in breastfeeding during the period from 1910 to 1930, the diarrheal mortality declined substantially in the United States pointing toward better milk standards as a major contributor to the declining infant mortality [Lee (2007)].

Another important reason for the decline in infant mortality was increasing intake and absorption of essential micronutrients. Iron deficiency was widespread in industrialized countries in the 18th and 19th centuries but had almost disappeared by the mid-20th century due to a switch from a cereal-based diet that was rich in

inhibitors of iron absorption to more mixed diets rich in iron, and the eradication of helminth and other infectious diseases [Ramakrishnan and Yip (2002)]. Iodine deficiency was also widespread before iodine fortification of salt began after the mid-1920s in the industrialized countries [Maberly (1994)]. The high content of cereals and low content of fish and meat in the diet during the 19th century, furthermore, rendered iodine deficiency a potentially major problem as the soil has a low concentration of iodine in many areas across the world [McClendon (1939)]. King Louis Philippe I commissioned what was probably the first national goiter survey in 1845 in France, noting that goiter is caused by iodine deficiency. From a total population of 36 million, the survey revealed 370,403 people with goiter and 120,000 cretins or idiots [Maberly (1994)]. These conditions are at the extreme end of the spectrum, which suggests that the 1.4% of the population in France with goiter and cretinism was only the tip of the iceberg, and much less dramatic, but still significant iodine-deficiency-induced cognitive impairment would have been widespread in France before salt was fortified with iodine. In Michigan, in 1918, goiter was present in 30.3% of individuals registering for military service [Lynn (2009)].

The decline in infant mortality is also highly likely to have been associated with declining infant morbidity. Unfortunately, there is very little, if any, data available on infant morbidity far back in time. There are, however, two direct pieces of evidence pointing toward parallel movements of infant mortality and morbidity during the epidemiological transition. First, following Grossman's (1972) pioneering study, it is increasingly acknowledged that health insults reduce the health capital each individual is born with [Barker (1990)]. Childhood infections, malnutrition, and risk factors all reduce the individual's health stock over time, leading to increased observed morbidity incidence and higher mortality risk as persons age [Grossman (1972)]. The strong declining infant mortality during the epidemiological transitions was followed, with a delay, by reduced mortality in older ages, as demonstrated in the regressions in Section 6 where it is shown that cohort infant mortality is a strong and significant predictor of working-age mortality and life expectancy at birth. Second, as discussed above, Reidpath and Allotey (2003) find a correlation between DALE and infant mortality of 0.91 using present-day data, again supporting the thesis of parallel movements in infant mortality and morbidity.

The eradication of lead pipes for water supply contributed further to declining infant mortality in the beginning of the 20th century. Lead pipes were widely used for distributing water before circa 1920 [Clay et al. (2010)]. Infants are highly sensitive to lead and, given that infants were often fed with milk that was diluted with water, they were often poisoned with lead, especially in the bigger cities before and during the 19th century [Clay et al. (2010)]. Moreover, lead was transmitted from drinking water, through the mother, into the fetus [Clay et al. (2010)]. Modern studies show that even small amounts of lead can lead to fetal and infant mortality [Clay et al. (2010)].

Malnutrition was another contributor to the high infant mortality and was the major cause of the growth stunting before the great demographic transition. The

lack of sufficient caloric intake in the 19th century is reported by Harris et al. (2014), who state that the calorie intake of an adult person increased by approximately 500 calories per day between 1800 and the eve of World War I. This suggests that calorie malnutrition must have been widespread during most of the 19th century, particularly when it is taken into account that the energy losses to physical activities and to diseases, most obviously diarrheal diseases, but also fevers and respiratory infections, were substantially larger in the 19th century than they are today.

4.3. Is the Decline in Infant Mortality Associated with Increasing Cognitive Ability?

It has been argued above that infant mortality is heavily influenced by PID health insults and malnutrition in utero and in infancy and that these factors permanently impair cognitive ability. Consequently, the decline in infant mortality since 1870 would have been associated with a gradual and potentially large increase in the IQ of the adult population over approximately the past 120 years. This path is consistent with the findings in the literature. Psychologists have identified massive IQ gains among western populations over the past century, the so-called Flynn effect. Using IQ test data for military conscripts and students in their teens, several studies find IQ gains of approximately 3 points per decade, which translates into IQ gains of approximately 30 points during the last century in several industrialized countries [Flynn (1999), Lynn (2009)]. These results are consistent with the path in infant mortality. Multiplying the average change in cohort infant mortality in the OECD countries over the period 1870–2011 and the coefficient of infant mortality estimated from regressing IQ estimates from Lynn and Vanhanen (2006) on infant mortalities in 2006 across countries in the world yields an IQ gain of 30 [Madsen (2016a)]. This gain is slightly smaller than the gains estimated by psychologists; the difference may reflect various other factors [see, for a discussion of potential factors, Lynn (2009)].

The finding of massive IQ gains is also consistent with the increasing size of cranial vaults over the past one to two centuries, giving some indication of an increasing IQ during the same period. Jantz (2001), for instance, finds a significant increase in cranial vaults in the United States during the period 1850–1975. Miller and Corsellis (1977) find that in the United States over the period 1860–1940 brain weight increased by approximately 6 g per decade. Finally, empirical evidence points toward health in utero and during childhood as the main force behind the secular increase in IQ. The largest gain in cognitive function has been recorded among children in the 6–18 month age group during the last century [Lynn (2009)]. Thus, the improvement in education could not have been the main cause of the increasing cognitive ability during the last century. Furthermore, better nutrition is found to influence fluid intelligence (the capacity to think logically and solve problems) and not crystallized intelligence (ability to use skills, knowledge, and experience) [Flynn (1999), Lynn (2009)]. This is consistent with the hypothesis

TABLE 1. First-stage IV regressions [equation (4)]

Dependent variable	(1) Φ_{it}^{IM}	(2) Φ_{it}^{H}	(3) Φ_{it}^{IM}	(4) Φ_{it}^{H}
$\ln\Phi_{it}^{Med}$	−43.0 (25.5)***	9.89 (47.5)***	−54.1 (21.4)***	13.3 (12.4)***
$\ln\Phi_{it}^{I^{Infr}/Y}$	−16.6 (13.7)***	−5.17 (0.31)	−5.70 (5.76)***	−101 (3.03)***
$\ln\Phi_{it}^{P^f/P}$	3.07 (13.3)***	−0.22 (7.64) ***	0.60 (5.66)***	−0.09 (3.80)***
R^2	0.79	0.72	0.98	0.85
p-value	0.00	0.00	0.00	0.00
Observations	3087	3087	3087	3087
CD	Y	Y	Y	Y
TD	N	N	Y	Y
TT	N	N	Y	Y
Estimation period	1865–2011	1865–2011	1865–2011	1865–2011

Notes: The numbers in parentheses are absolute *t*-ratios based on White's heteroscedasticity consistent covariance matrix and the Newey–West autocorrelation consistent covariance matrix of order 1. *CD* = inclusion of country dummies, *TD* = inclusion of time-dummies, and *TT* = inclusion of country-specific time-trends, *p*-value = *p*-value of *F*-tests for the joint significance of the instruments, Φ^H = cohort height, and Φ^{IM} = cohort infant mortality. *** $p < 0.01$.

that those health improvements in utero and in early life, which, as discussed earlier, caused a drop in infant mortality rates, are also responsible for increases in cognitive ability over time.

4.4. First-Stage Regression Results

The first-round IV regressions are reported in Table 1. Time dummies and country-specific time-trends are excluded from the regressions in the first two columns and included in the last two columns. The *p*-values of the joint significance of the coefficients of the instruments and the *R*-squared indicate that the explanatory variables are sufficiently correlated with the dependent variables to act as potentially good instruments. Most of the coefficients of the instruments are significant at the 1% level and have the expected signs. The coefficients of cohort significant medical innovations, Φ_{it}^{Med}, are all of the expected sign and are highly significant. An increase in medical innovations results in reduced infant mortality and increased height. The coefficients of cohort infrastructure investment, $\Phi_{it}^{I^{Infr}/Y}$, are significantly negative in the cohort infant mortality regressions, as expected; however, they are, unexpectedly, negative and statistically significant in one of the cohort height regressions. A possible reason for this result is that the government investment in infrastructure includes items that are not always particularly health related and the marginal health effects of public infrastructure investment are likely to have waned after WWII. As argued above, the key health-related public infrastructure projects such as provision of sewage and clean water facilities were likely to have been completed before WWII. More importantly, since height is more associated with nutrition than infections, I would expect a much weaker effect of public

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infrastructure investment on height than infant mortality. Finally, the coefficients of cohort real food prices, $\Phi_{it}^{P^f/P}$, are highly significant and have the expected positive signs for cohort infant mortality and negative signs for cohort height.

Public infrastructure investment as a percentage of GDP, I^{Infr}/Y, acts as a proxy for investment in public infrastructure such as hospitals, sewage drainage, clean water supply, central heating and public housing, investments that were major contributors to the mortality transition in the rich countries in the first half of the 20th century [see, e.g., Cutler et al. (2006)].

4.5. Structural Regression Results

Restricted and unrestricted OLS and IV estimates of equation (1) at annual frequencies are presented in Table 2. Consider first the regressions covering the full estimation period in columns (1)–(7). The coefficients of cohort infant mortality and cohort height are statistically highly significant and of the expected signs in all the regressions, regardless of whether time dummies and country-specific time-trends are included in the regressions. The latter result is important because it suggests that the statistical significances of Φ^{IM} and Φ^{H} are not driven by a common trend between productivity and Φ^{IM} and Φ^{H} or omitted variables that have changed at the same rate across countries.

Economically, the entire health transition of an approximately 150 point decline in infant mortality and an approximately 10 cm increase in height over the period 1865–2011 have contributed to a 480% (cohort infant mortality) and 16% (cohort height) increase in labor productivity using the coefficient estimates in column (6). Thus, the epidemiological transition has been an important factor behind the productivity advances in the OECD countries over the past century and a half. However, the quantitative results found here fall short of Fogel's (2004) conclusion that most of the productivity growth over the past two centuries has been driven by improved health and nutrition, given that labor productivity increased almost 23-fold over the period 1865–2011 for the average OECD country (unweighted). Furthermore, as shown below, the quantitative results here may even be on the high side because the cohort health variables are likely to have captured the productivity effects of confounding factors that have shown approximately the same time profiles as infant mortality and height.

Thus far, it has been assumed that pupils enter the labor market at the age of 15, regardless of whether they are enrolled as students at high school or tertiary education; however, the average age at which youngsters enter the labor market is much higher today than it was a century ago because large fractions of the population in the age group 16–22 are enrolled in secondary and tertiary education. The assumption of entrance to the labor market at the age of 15 has been made to avoid any endogenous feedback effects from improvements in health and cognition that may enhance student enrollment and, consequently, lead to biased coefficients of cohort infant mortality and working-age height. To allow for delayed entry into the labor market of students older than 15 years of age, cohort infant mortality

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TABLE 2. Structural regression results [equation (1)]

Dependent variable	(1) ln(*Y*/*L*)	(2) ln(*Y*/*L*)	(3) ln(*Y*/*L*)	(4) ln(*Y*/*L*)	(5) ln(*Y*/*L*)	(6) ln(*Y*/*L*)	(7) ln(*Y*/*L*)[#]	(8) ln(*Y*/*L*)	(9) ln(*Y*/*L*)	(10) ln(*Y*/*L*)	(11) ln(*Y*/*L*)
Φ_{it}^{IM}	−0.014***	−0.013***	−0.008***	−0.018***	−0.023***	−0.032***	−0.031***	−0.023***	−0.005***	−0.015***	−0.024***
	(27.3)	(28.9)	(11.4)	(7.25)	(14.3)	(8.18)	(8.17)	(20.8)	(4.65)	(11.0)	(13.9)
Φ_{it}^{H}	0.038***	0.005***	0.007***	0.068***	0.054***	0.016***	0.017***	0.001	0.012***	0.001	0.015***
	(15.5)	(3.68)	(5.11)	(4.75)	(4.17)	(6.98)	(7.60)	(0.47)	(5.22)	(0.79)	(6.72)
Method	OLS	OLS	OLS	2SLS	2SLS	2SLS	2SLS	OLS	OLS	OLS	OLS
Frequency	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual
Observations	3,087	3,087	3,087	3,087	3,087	3,087	3,087	1,281	1,491	1,617	672
Sargan's p-value	–	–	–	0.05	0.72	0.15	0.10	–	–	–	–
CD	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
TD	Y	N	Y	Y	N	Y	Y	Y	Y	Y	Y
TT	N	Y	Y	N	Y	Y	Y	Y	Y	Y	Y
Estimation period	1865–2011	1865–2011	1865–2011	1865–2011	1865–2011	1865–2011	1865–2011	1920–1980	1865–1935	1935–2011	1980–2011

Notes: Country fixed effect dummies and constant terms are included in all the regressions. *CD* = inclusion of country dummies, *TD* = inclusion of time dummies, and *TT* = inclusion of country-specific time-trends. The numbers in parentheses are absolute t-ratios based on White's heteroscedasticity consistent covariance matrix and the Newey-West autocorrelation consistent covariance matrix of order 1. [#]Allowing for delayed entry of graduates into the labor force [equation (5)]. *** $p < 0.01$.

and height are computed as follows:

$$\Phi_{it}^{X} = \sum_{\tau=23}^{64}\left(\frac{Pop_{\tau it}}{\Psi_{it}}X_{i,t-\tau}\right) + \sum_{\tau=15}^{17}\left(\frac{Pop_{\tau it}}{\Psi_{it}}X_{i,t-\tau}\right)\left(1-GER_{it}^{S}\right) + \sum_{\tau=18}^{22}\left(\frac{Pop_{\tau it}}{\Psi_{it}}X_{i,t-\tau}\right)\left(1-GER_{it}^{T}\right), \quad (5)$$

where

$$\Psi_{it} = \sum_{\tau=23}^{64} Pop_{\tau it} + \sum_{\tau=15}^{17} Pop_{\tau it}\left(1-GER_{\tau it}^{S}\right) + \sum_{\tau=18}^{22} Pop_{\tau it}\left(1-GER_{\tau it}^{T}\right), \quad (6)$$

$X = IM, H$, $GER_{\tau it}^{S}$ is the age cohort τ that is enrolled in secondary schooling, $GER_{\tau it}^{T}$ is the age cohort τ that is enrolled in tertiary education, and Ψ is the population of the 15–65 age cohort that is not enrolled in education. The second and third right-hand terms in equation (5) extend equations (2) and (3) to allow for the fact that secondary and tertiary schooling reduces the fraction of the population of the 15–22 age cohort that is in the labor force. Equation (6) accounts for the fraction of the working-age population that is undertaking education since the fractions of education-adjusted age cohorts do not add up to 1.

The results of regressing equation (1) while allowing for delayed entrance of graduates into the labor force are presented in column (7) in Table 2. The coefficients of cohort infant mortality and cohort height both remain highly significant and the magnitude of their coefficients and their associated t-ratios is almost identical to the baseline regression counterpart in column (6), where delayed entry is not accounted for. These results suggest that the baseline regressions are not overestimating the productivity effects of health due to delayed entry of graduates not being allowed for in the estimates of cohort infant mortality.

A question is whether the exclusion restrictions are satisfied in the two-stage least-squares (2SLS) regressions. Sargan's tests for overidentifying restrictions in Table 2 are generally not rejecting the exclusion restrictions ($0.05 < p < 0.72$), noting that the probability of rejecting the null hypothesis is an increasing function of the sample size. Importantly, Sargan's test is insignificant at conventional significance levels in the regression in which country-specific time-trends are all included in the regression. This result underscores the importance of including *TD* and *TT* in the regressions. Therefore, the analysis proceeds with these deterministic variables included in the regressions.

Finally, the regressions in the last four columns in Table 5 (shown later in this article) cover the periods 1920–1980, 1865–1935, 1935–2011, and 1980–2011 to gain insight into the stability of the coefficients and the force at which cohort health insults have affected productivity in different time-periods. The coefficients of cohort infant mortality are highly significant and of almost the same magnitude

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in all the regressions, except the regression covering the period 1865–1935, where the coefficient is around a quarter of what it is in the regressions covering later estimation periods. This result may reflect that the quality of the data deteriorates as we go back in time and that there were not large identifying variations in cohort infant mortality before 1935. The coefficients of cohort height are highly significant in the regressions covering the period 1865–1935 but insignificant in the other regressions, indicating that cohort height has become less influential for productivity advances over the intermediate period 1930–1980, probably reflecting that nutrition and health reached a point where further gains of these two factors have little effect on productivity.

5. ROBUSTNESS TESTS

This section goes a step further than the preceding section by examining how robust the results thus far are to inclusion of potentially influential confounding variables, to time-aggregation, and to estimation in first-differences. An important issue at stake is whether the coefficients of Φ^{IM} and Φ^{H} have captured productivity effects beyond cohort health insults because key confounding factors with approximately the same time-profile as Φ^{IM} and Φ^{H} have been omitted from the regressions. The mortality transition approximately coincided with events such as the fertility transition, the introduction of mass education, a wave of institutional improvements, the financial revolution, and the Second Industrial Revolution, and it is quite possible that these factors have had lagged productivity effects that coincided with cohort infant mortality and height.

5.1. Time-Aggregation

The models are estimated in 5- and 10-year non overlapping intervals and in levels in the first four columns in Table 3, where the variables are measured as the annual averages within each time-interval. The time-aggregation reduces the influence of cyclical and erratic year-to-year fluctuations on the regression results and, at the same time, reduces the statistical significance of the coefficient estimates as a result of the reduced number of observations relative to the annual estimates. The estimated coefficients of Φ^{IM} and Φ^{H} all remain quite significant and have their expected signs, suggesting that the results in the preceding section are robust to time-aggregation.

5.2. First-Difference Regressions

First-difference regressions are presented in the last four columns in Table 3. Country fixed effect dummies, *CD*, and time dummies, *TD*, are included in the regressions in columns (6) and (8) to cater for time-trend (the *CD* dummies are first-difference transformations of the *TT* dummies) and omitted variables that show the same time-varying profile across countries. Only OLS regressions are

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TABLE 3. Estimates of equation (1) in 5- and 10-year intervals in levels and in first differences

Dependent variable	(1) $\ln(Y/L)$	(2) $\ln(Y/L)$	(3) $\ln(Y/L)$	(4) $\ln(Y/L)$	Dependent variable	(5) $\Delta\ln(Y/L)$	(6) $\Delta\ln(Y/L)$	(7) $\Delta\ln(Y/L)$	(8) $\Delta\ln(Y/L)$
Φ_{it}^{IM}	-0.008^{***}	-0.028^{***}	-0.016^{***}	-0.024^{***}	$\Delta\Phi_{it}^{IM}$	-0.011^{***}	-0.003^{***}	-0.009^{***}	-0.005^{***}
	(7.35)	(4.93)	(6.56)	(3.06)		(9.33)	(3.13)	(6.72)	(3.23)
Φ_{it}^{H}	0.008^{***}	0.017^{***}	0.016^{***}	0.029^{**}	$\Delta\Phi_{it}^{H}$	0.003^{***}	-0.001	0.001	-0.001
	(3.15)	(3.66)	(4.01)	(2.10)		(2.89)	(0.56)	(0.48)	(0.45)
Method	OLS	2SLS	OLS	2SLS	Method	OLS	OLS	OLS	OLS
Frequency	5 year	5 year	10 year	10 year	Frequency	5 year	5 year	10 year	10 year
Observation	630	630	315	315	Observations	630	630	315	315
Sargan's p-value		0.534		0.77					
CD	Y	Y	Y	Y	*CD*	N	Y	N	Y
TD	Y	Y	Y	Y	*TD*	N	Y	N	Y
TT	Y	Y	Y	Y	*TT*	N	N	N	N
Estimation period	1865–2010	1865–2010	1870–2010	1870–2010	Estimation period	1865–2010	1865–2010	1870–2010	1870–2010

Notes: CD = inclusion of country dummies, *TD* = inclusion of time dummies, and *TT* = inclusion of country-specific time-trends. The SUR estimator is used in the regressions in columns (5) and (6). The numbers in parentheses are absolute t-ratios based on White's heteroscedasticity consistent covariance matrix and the Newey-West autocorrelation consistent covariance matrix of order 1. $^{***}p < 0.01$; $^{**}p < 0.05$.

presented because the first-stage regressions did not give meaningful results in terms of first-stage F-tests for excluded restrictions—presumably because of time-varying diffusion time-lags for great medical innovations and public investment in infrastructure. The data are again measured in 5- and 10-year intervals as opposed to annual intervals to filter out erratic and cyclical movements at business-cycle frequencies. For example, a few European countries experienced a temporary double-digit year-to-year percentage decline in GDP at the end of or immediately after WWII, observations that are very influential for annual regression results. Furthermore, since the data are not perfect, it is impossible to find an exact correspondence between the changes in productivity and cohort mortality of the working population, particularly because the pre-WWII infant mortality data are often interpolated at 5- and 10-year time intervals in the source data.

The coefficients of Φ^{IM} are all significant at the 1% level and of the expected sign. The magnitudes of the coefficients of Φ^{IM} are smaller than their level counterparts since first-difference regressions tend to capture only the short-run productivity effects of Φ^{IM} and are, generally, much more susceptible to error-in-variable biases than level regressions. The coefficient of Φ^{H} is positive and significant at the 1% level in the regression in column (5) in Table 3 and insignificant in the last three regressions in the table. The latter results suggest that Φ^{H} may not always be a robust determinant of productivity growth—at least not in the short run. That it is more significant in the 5-year than the 10-year regressions probably reflects the differences in sample sizes and that the seemingly unrelated regression (SUR) estimator is used in these regressions. (The SUR estimator cannot be used in the 10-year regressions because the number of countries exceeds the number of time-periods.) The coefficient of Φ^{H} is rendered insignificant in the 5-year regressions when country and time-dummies are included in the regressions, which can be interpreted either as the deterministic variables capture valuable information contained in Φ^{H} or as the deterministic variables capture the productivity effects of omitted variables. In any events, the regression results indicate that Φ^{H} is not as robust a determinant of productivity as Φ^{IM}.

5.3. Adding Confounding Variables

The high significance of the cohort health insult variables in the productivity regressions discussed above suggests that critical confounding variables may have been omitted from the regressions. To address this concern, this section examines how far the health insult hypothesis can be pushed when potentially influential confounding variables are included in the regressions. The key issue at stake is whether the coefficients of Φ^{IM} and Φ^{H} have captured productivity effects beyond cohort health because key confounding factors with approximately the same time-profile as Φ^{IM} and Φ^{H} have been omitted from the regressions. The mortality transition approximately coincided with events such as the fertility transition, the introduction of mass education, a wave of institutional improvements, the financial revolution, and the Second Industrial Revolution, and it is quite possible that these

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factors have had delayed productivity effects that coincided with cohort infant mortality and height. Furthermore, as argued below, the confounding variables considered here are likely, according to the growth literature, to have been the most influential variables for the productivity advances in the West over the past two centuries. Inclusion of these variables in a cohort form, in addition to time dummies, is likely to be a strong stress test for the health insult hypothesis.

Equation (1) is extended by the following variables in the regressions in this section: S^D = domestic stock of knowledge, S^F = foreign stock of knowledge transmitted through imports, EA = educational attainment, Φ^{Exec} = cohort constraints on executive, Φ^{Fert} = cohort general fertility rate, Φ^{Cre} cohort credit-to-GDP ratio, and Φ^{Pat} = cohort patent applications. The variables are converted into cohort variables using equations (2) and (3) with the variable in question replacing infant mortality or height. These control variables have often been highlighted in the growth literature as being influential for the transition from the post-Malthusian growth regime to the modern growth regime [see, e.g., Galor (2011)]. Education and domestic and foreign knowledge have often been stressed as being important for growth over the past two centuries, in general [see, e.g., Madsen (2007, 2010)]. Educational attainment can be considered to be a cohort variable in the sense that educational attainment is determined by school enrollments at the time the working force did their education.

The quality of institutions has often been stressed in the literature as a key factor for economic development. Since significant improvement in the quality of institutions in the countries considered here occurred in the decades surrounding the turn of the 20th century, to some extent, coincided with the declining infant mortality and the increasing height, it is possible that Φ^{IM} and Φ^{H} capture some of the productivity effects of Φ^{Exec}. Furthermore, institutional improvements are likely to take time to take effect as the people need time to adjust their behavior and understand the new incentive structure imposed by new institutions. For example, it takes several years and even decades for entrepreneurs to establish new companies, for new credit institutions to evolve, for new innovators to establish themselves, for new school enrollments to affect productivity, and so on.

Polity IV's constraints on executive, constructed by Marshall et al. (2013), is used as a measure of the institutional quality because it is a widely used and an accepted measure of the quality of institutions and because it is available far back in time. The variable ranges from 1 (unlimited authority of the chief executive) to 7 (accountability groups have effective authority equal to or greater than the executive in most areas of activity), where higher values equal a greater extent of institutionalized constraints on the power of chief executives.

The general fertility rate is a key cohort variable as the literature, particularly unified theories of economic growth [Galor and Weil (2000), Galor (2011)], has often highlighted its positive productivity effects during the transition from the post-Malthusian growth regime with low productivity rates to the modern growth regime. In the Galor–Weil model, the fertility transition resulted in a reduction in the population growth drag introduced by land as a fixed factor of production

and it increased education induced by the quantity–quality tradeoff in the fertility decision. Again, it takes time for the fertility transition to take full effect on productivity through enhanced education, savings, labor force participation, etc., as discussed by Galor (2011). Cohort fertility, Φ^{Fert}, is based on the number of children per female in the reproductive age (15–44). Cohort fertility is potentially a very important confounding variable since the fertility transition occurred at almost the same time as the mortality transition [Galor (2011)]. Note, however, that some caution needs to be exercised when interpreting the regression results relating to this variable because fertility is, at the same time, an outcome variable of cohort health insults, as discussed in more detail in the next section.

Financial development has long been considered to be an essential force behind economic development as it potentially enhances saving, investment, innovation, and schooling, outcomes that tend to have delayed effects on productivity [Madsen and Ang (2016)]. Since the credit-to-GDP ratio followed the same path as infant mortality and height during the early part of the demographic transition, it cannot be ruled out that cohort health has partly captured the delayed productivity effects of financial development. Following the convention, cohort financial development, Φ^{Cre}, is proxied by the credit-to-GDP ratio, where credit is measured by credit to the non financial private sector.

Cohort innovations, Φ^{Pat}, based on patent applications, are included as the final confounding factor that has, at least during the early phase of the demographic transition, followed the same path as cohort infant mortality and height. Cohort innovations may be a confounding factor because the Industrial Revolution was, to a large extent, driven by innovations and, at the same time, coincided with the mortality transition. There is plenty of historical evidence that technological breakthroughs often take decades to take effect on productivity, for example, the innovation of the steam engine, the electrical dynamo, the combustion engine. David (1990), for example, shows that it took about four decades for electrification in the United States to reach a 50% diffusion rate, suggesting very high adjustment costs of major innovations. David (1990), furthermore, argues that there are certain parallels between the introduction of the computer and electrical power, and innovations that are related to infrastructure often take a very long time to take effect. Finally, despite a highly innovative environment, productivity advances during the First British Industrialization were incredibly slow [Clark (2005)], again highlighting delayed productivity effects of innovation waves.

Knowledge spillovers through the channel of imports of intermediate products that contain new technology from country j to country i are computed using the following method of Madsen (2007) to smooth out erratic movements in import ratios:

$$S_{it}^{F} = \sum_{j=1}^{21} \frac{M_{ijt}^{s}}{Y_{jt}^{s,n}} S_{jt}^{D}, \quad i \neq j.j = 1, 2, \ldots, 21,$$

where M_{ij}^{s} is smoothed nominal imports of goods of high-technology products from country j to country i, $Y_{j}^{s,n}$ is country j's smoothed nominal income, and

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S_j^D is country j's stock of domestic knowledge, which is estimated using the perpetual inventory method and a 15% depreciation rate [see, for details, Madsen (2007)]. The 21 countries used to estimate knowledge spillovers are the same as those considered in this paper.

The results of adding the confounding cohort variables to the baseline regression are presented in Table 4. Time and country fixed effects dummies and country-specific time-trends are included in all regressions. The estimation period starts in 1870 instead of 1865 because some of the confounding variables are first available from 1870. The dependent variable is labor productivity in the regressions in columns (1)–(6) and (8), whereas total factor productivity (TFP) is the dependent variable in the last column. TFP is measured as $A = Y/(L^{\alpha}K^{1-\alpha})$, where K is the non residential capital stock, L is the hours worked (economy-wide employment multiplied by annual hours worked), and α is measured as the unweighted average of labor's income share for country i and the United States. Labor's income share is calculated as the economy-wide compensation to employees divided by nominal GDP, where labor's compensation is corrected for imputed payments to the self-employed and employed family members. Finally, following the Mincerian approach in which the return to one additional year of education is independent of the years of schooling educational attainment, EA, is unlogged.

Common for all the regressions in which labor productivity is the dependent variable in Table 4 is that the coefficients of foreign knowledge stock are highly significant and positive, a result that is consistent with the findings in most of the literature [see, e.g., Engelbrecht (1997), Madsen (2007)]. The coefficient of domestic knowledge stock is mostly significantly negative and the coefficients of both S^F and S^D are dragged down by the cohort health insult variables and country-specific time-trends. The parameter estimates of S^F and S^D become 0.24 (13.7) and 0.11 (6.58) (the numbers in parentheses are t-statistics) when Φ^{IM}, Φ^{H}, and country-specific time-trends are omitted from the OLS regression in the first column, suggesting that the coefficients of S^F and S^D are probably severely inflated in the literature due to the omission of confounding variables (results not shown).

Importantly, negative coefficients of S^D do not mean that S^D impacts negatively on productivity but that it is driven down to negativity by omitted variables, measurement errors, and endogeneity. Measurement errors are likely to have played a role since patents are not ideal measures of innovations as influential and uninfluential patents get the same weight in patent counts and not all important innovations are patented. This may also to some extent explain why foreign knowledge stock is significantly positive in the labor productivity regressions since the law of large numbers ensures that the distribution of significant and insignificant innovations is relatively constant across time and space. Cohort fertility, cohort credit, education, and cohort patents are all highly significant and of the expected signs, regardless of whether they are entered separately or jointly in the labor productivity regressions. Thus, the increasing education, innovations, financial development and reduced fertility may all have been influential for the transition

TABLE 4. Allowing for confounding factors in the structural regressions [equation (1)]

Dependent variable	(1) ln(*Y/L*)	(2) ln(*Y/L*)	(3) ln(*Y/L*)	(4) ln(*Y/L*)	(5) ln(*Y/L*)	(6) ln(*Y/L*)	(7) ln(*TFP*)	(8) ln(*Y/L*)
Φ_{it}^{IM}	−0.013(14.7)***	−0.017(17.3)***	−0.012(13.6)***	−0.017(19.5)***	−0.016(18.2)***	−0.010(11.8)***	−0.004(6.38)***	−0.016(8.84)***
Φ_{it}^{H}	0.019(12.4)***	0.013(8.46)***	0.008(5.30)***	0.013(8.45)***	0.013(8.55)***	0.014(9.28)***	0.005(3.49)***	0.028(11.8)***
ln S_{it}^{D}	−0.012(1.19)	−0.028(2.61)***	−0.024(2.32)**	−0.037(3.58)***	−0.042(4.11)***	−0.024(2.40)**	−0.012(0.69)	−0.014(2.06)**
ln S_{it}^{F}	0.016(1.91)*	0.019(3.00)***	0.018(3.32)***	0.018(3.20)***	0.019(3.01)***	0.015(2.34)**	−0.003(0.68)	0.015(5.24)***
ln Φ_{it}^{Fert}	−0.725(13.7)***					−0.566(13.0)***	−0.232(6.04)***	−0.549(5.96)***
ln Φ_{it}^{Exec}		−0.047(1.32)				−0.064(1.87)*	−0.007(0.26)	−0.035(1.56)
EA_{it}			0.054(13.4)***			0.046(9.43)***	0.002(0.52)	0.012(3.26)***
ln Φ_{it}^{Cre}				0.069(6.89)***		0.059(5.56)***	−0.010(1.24)	0.055(7.57)***
ln Φ_{it}^{Pat}					0.034(5.75)***	0.019(3.13)***	0.001(0.20)	0.010(2.17)**
Method	OLS	OLS	OLS	OLS	OLS	OLS	IV	IV
CD	Y	Y	Y	Y	Y	Y	Y	Y
TD	Y	Y	Y	Y	Y	Y	Y	Y
TT	Y	Y	Y	Y	Y	Y	Y	Y

Notes: *CD* = inclusion of country dummies, *TD* = inclusion of time dummies, and *TT* = inclusion of country-specific time-trends. S^D = domestic knowledge stock, S^F = foreign knowledge stock, Φ^{Fert} = cohort fertility, Φ^{Exec} = cohort constraints on executive, *EA* = educational attainment, Φ^{Cre} = cohort credit, and Φ^{Pat} = cohort patents. The numbers in parentheses are absolute *t*-ratios based on White's heteroscedasticity consistent covariance matrix and the Newey–West autocorrelation consistent covariance matrix of order 1. The estimation period is 1870–2011. *** $p < 0.01$; ** $p < 0.05$; * $p < 0.10$.

to the modern growth regime, which suggests that the conventional regression analyses that use only contemporaneous regressors may well underestimate the cohort health insult productivity effects. Cohort constraints on executive remain the only cohort confounding variable that is not significant and even becomes significantly negative at the 1% level if it is entered unlogged.

The coefficients of Φ^{IM} and Φ^{H} remain highly significant and have their expected signs in all regressions. Importantly, the significance of Φ^{IM} and Φ^{H} are remarkably robust to the inclusion of confounding variables, suggesting that cohort health insults have productivity effects that are quite independent of other cohort variables that have been potentially important for the productivity path in the OECD countries. Furthermore, the results are almost unaltered when cohort infant mortality and height are instrumented. The coefficients of cohort infant mortality and height in the IV regression in the last column in Table 4 remain economically and statistically highly significant and of the expected signs when all the control variables are included in the regression. Finally, many of the control variables are insignificant in the TFP regression suggesting that their significance in the other regressions in Table 4 has been partly driven by a high correlation with capital stock; however, probably the most important reason for their low significance is that capital stock is measured by a large margin of error. Only Φ^{IM}, Φ^{H}, and cohort fertility are significant and of the expected signs in the TFP regression.

5.4. Summary Statistics and Simulations

To get an impression of the economic significance of the coefficients in the regression containing all the confounding variables, model simulations are presented along with summary statistics in Table 5. Simulations are carried out to get an impression of the economic significance of the coefficients and, particularly, to get a feeling for how much the health cohort variables have potentially contributed to growth during the health transition over the past two centuries. Caution needs to be taken when the results are interpreted because the variables are not truly exogenous and some potentially important drivers of growth have been omitted from the regression. Furthermore, the negative contribution to the growth of institutions, Φ^{Exec}, and domestic knowledge stock, S^{D}, should not be taken literately, but, as hinted above, these are probably negative because of measurement errors, endogeneity, and omitted variables.

The simulations in the last row in Table 5 show the contribution of each explanatory variable to the arithmetic annual growth rates over the period 1865-2011, where the simulations are based on the regression including all confounding variables and time-dummies in column (6) in Table 4. The simulated growth rates are estimated by multiplying each coefficient by the average arithmetic change of each variable for the OECD countries over the period 1865–2011. Cohort infant mortality, for example, has contributed, on average, $-0.0099 \times -0.69 \times 100 = 0.68\%$ per annum to labor productivity growth, where the number -0.69 is the average annual change in the cohort infant mortality rates in the OECD countries over the

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TABLE 5. Summary statistics and simulations (1865–2011)

	$\ln(Y/L)$	Φ^{IM}	Φ^{H}	$\ln S^{F}$	$\ln S^{D}$	Φ^{Fert}	Φ^{Exec}	EA	Φ^{Cre}	$\ln\Phi^{Pat}$	$Lexp$	$Mort^{Wa}$
Mean	1.7	78.5	110.3	6.3	8.8	77.4	1.2	7.6	19.5	5.48	60.2	0.7
SD	1.1	44.5	6.6	3.3	2.8	19.5	0.4	3.1	17.1	3.33	14.5	0.2
Min	−1.2	7.5	93.9	−3.5	−1.8	42.0	−0.5	0.17	0.1	−6.81	24.7	0.2
Max	3.7	226.5	124.3	32.2	14.7	152.4	1.6	15.4	76.6	11.3	83.3	1.4

Simulations (average annual growth rates, 1865–2011)

	Actual Y/L	Φ^{IM}	Φ^{H}	$\Phi\ln S^{F}$	$\Phi\ \ln S^{D}$	Φ^{Fert}	Φ^{Exe}	***EA***	Φ^{Cre}	Φ^{Pat}	Predicted Y/L
%	2.06	0.68	0.15	0.09	−0.02	0.21	−0.03	0.29	0.12	0.07	1.56

Notes: The simulations are based on the regression in column (6) in Table 4 and the data are OECD averages. The growth rates in the simulations are arithmetic averages.

period 1865–2011 and the number −0.0099 is the estimated coefficient of cohort infant mortality.

The per annum productivity growth has been 2.06%, on average, for the OECD countries and the productivity growth predicted by the non deterministic explanatory variables is 1.56%. The 0.50% growth (2.06 − 1.56) residual is predominantly explained by omitted variables and, to a large extent, captured by the time dummies and the country-specific time-trends. Improved health explains 0.83% of annual productivity growth, where 0.68% is attributed to health proxied by cohort infant mortality and 0.15% is attributed to health proxied by cohort height. Thus, more than a third of the productivity growth experienced in the OECD countries over the period 1865–2011 is due to improved health and nutrition, which is below the figures estimated by Fogel (2004) but is still in a high range.

Of the confounding variables, the simulations show that education, followed by the fertility transition, cohort credit, foreign knowledge through the channel of imports, and cohort patents have been influential for productivity growth. Foreign knowledge stock and cohort patents jointly explain 0.16% of growth and increased education explains 0.29% of annual growth, a result that is consistent with endogenous growth theory in which innovations and human capital are the main forces behind productivity advances as they increase the number of product varieties and the quality of goods produced. Of the remaining variables, the fertility transition explains 0.21% of growth and financial development explains 0.12% of growth. Thus, overall, several factors have contributed to the growth experienced in the advanced countries since 1865, where broadly defined human capital (education plus health) explains the lion's share of productivity growth followed by innovations and the fertility transition.

6. TRANSMISSION CHANNELS

The results in the preceding section suggest that cohort health is a robust predictor of productivity. However, the regressions have been silent about potential channels through which cohort health affects productivity. As an additional robustness check, this section examines the extent to which cohort health, as proxied by cohort infant mortality and working-age height, influences school enrollment rates, *GER*, savings rates, proxied by national savings divided by nominal income, S/Y^n, political participation, *Dem*, life expectancy at birth, *Lexp*, working-age mortality, $Mort^{Wa}$, the general fertility rate, *Fert*, cohort population growth, $\Delta \ln \Phi^{Pop}$, and the innovative activity proxied by patent applications divided by employment, X/L.

Education, innovations, and savings are the key drivers of productivity growth in endogenous and neoclassical growth models, whereas political participation affects productivity positively or negatively through various channels, as discussed in Doucouliagos and Ulubaşoğlu (2008). The fertility transition has been highlighted as an important factor behind the transition from the post-Malthusian to the modern growth regime, as discussed in the preceding section, and the productivity

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effects of life expectancy and working-age mortality have been discussed in the Introduction.

Education is measured as gross enrollment rates at primary, GER^P, secondary, GER^S, and tertiary, GER^T levels, where GERs refer to the fraction of the school age cohorts that is enrolled in education. The cohort health variables, Φ^{IM} and Φ^H, are estimated so that the cohorts refer to the school ages at the different levels in the GER regressions. For primary schooling, for example, the cohort infant mortality is estimated as the average of 6–12 years lag in infant mortality. The reason cohort health may affect GERs is that the expected returns to education are positively related to the cognitive skills of the students because they increase learning capacity.

Fertility takes a special role in the analysis in that cohort fertility is used to explain productivity, while fertility is also considered as an outcome variable that is affected by the cohort health insult variables, Φ^{IM} and Φ^H. Fertility is an outcome variable because of the quantity–quality tradeoff in which parents reduce their fertility and increase the educational resources of their children in response to increasing returns to education, assuming that the returns to education are an increasing function of the health and the cognitive function of the child, on the other hand. The declining cohort fertility over the past two centuries, which has been induced by factors other than cohort health insults, has impacted on productivity independently of cohort health insults.

Cohort population growth, $\Delta \ln \Phi^{Pop}$, is a potential outcome variable because cohort health insults impinge on fertility and mortality rates. Furthermore, population growth is of special interest because Acemoglu and Johnson (2007) in their influential paper find that per capital growth is unrelated to life expectancy predominantly because positive income effects have been counterbalanced by health-induced population growth. The dependent variable, Φ^{Pop}, is measured in log first-differences as opposed to log levels because infant mortality and height impinge on the population through fertility and mortality rates, both of which impact directly on population growth rates and not on population levels.

2SLS regressions are presented in the upper panel, whereas OLS regressions are presented in the lower panel of Table 6. Sargan's test for overidentifying restrictions is significant in the regressions in columns (1)–(3) and (7)–(10), suggesting, by Sargan's criteria, that the instruments are not exogenous in these regressions. Thus, the results in these regressions should be taken with a grain of salt. Considering the GERs, the coefficients of Φ^{IM} and Φ^H are mostly highly significant and of the expected negative (Φ^{IM}) and positive (Φ^H) signs—particularly at the secondary and tertiary levels. The significance of Sargan's tests in these regressions suggests that the instruments may not be exogenous, perhaps because investment in schools, which is likely to be highly correlated with GERs, is included in public infrastructure investment. Endogeneity aside, the results are consistent with the results of Barreca (2010) who finds that educational attainment is reduced by 25% for individuals exposed to early-life malaria in the early-20th-century United States. The results are also consistent with the finding of Hansen and Strulik (2015) that

TABLE 6. Influence of cohort health insults on outcome variables

Dependent variable	(1) GER^P	(2) GER^S	(3) GER^T	(4) $\ln(X/L)$	(5) $\ln(S/Y^n)$	(6) *Fert*	(7) $Mort^{Wa}$	(8) *Dem*	(9) *Lexp*	(10) $\Delta \ln\Phi^{Pop}$
					2SLS					
Φ_{it}^{IM}	−0.008	−0.096***	−0.017***	−0.047***	−0.014***	0.002**	0.006***	−0.360***	−0.007***	0.007***
	(1.53)	(10.3)	(6.29)	(7.08)	(3.02)	(2.37)	(16.5)	(4.24)	(7.17)	(4.07)
Φ_{it}^{H}	0.001	0.036***	0.008***	−0.083***	−0.012	−0.047***	−0.019***	1.454***	0.007***	0.004
	(0.23)	(3.05)	(5.45)	(6.15)	(0.71)	(15.5)	(9.58)	(8.48)	(4.67)	(0.74)
Sargan's p-value	0.01	0.04	0.00	0.43	0.46	0.61	0.03	0.01	0.00	0.01
					OLS					
Φ_{it}^{IM}	−0.016***	−0.055***	−0.012***	−0.016***	−0.005***	0.005***	0.006***	−0.327***	−0.003***	−0.001*
	(4.95)	(11.4)	(6.76)	(7.47)	(3.24)	(13.2)	(11.6)	(9.96)	(7.96)	(1.74)
Φ_{it}^{H}	0.002	0.017***	0.001	−0.007	0.009***	−0.032***	−0.008***	1.021***	0.006***	−0.020***
	(0.83)	(4.67)	(1.07)	(1.47)	(2.71)	(29.6)	(6.67)	(15.3)	(10.8)	(2.79)
Frequency	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual	Annual
Observations	2,982	2,982	2,982	2,982	2,982	2,982	2,982	2,982	2,982	2,982
CD	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
TD	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
TT	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y

Notes: The numbers in parentheses are absolute t-ratios based on White's heteroscedasticity consistent covariance matrix and the Newey-West autocorrelation consistent covariance matrix of order 1. GER^P = gross enrollment rate in primary education, GER^S = gross enrollment rate in secondary education, GER^T = gross enrollment rate in tertiary education, S = gross national savings rate, Y^n = nominal GDP, X = patent applications, L = economy-wide employment, *Fert* = general fertility rate, $Mort^{WA}$ = working-age mortality, *Dem* = democracy, *Lexp* = life expectancy at birth, and Φ^{Pop} = cohort population size (the cohort population growth rate is measured in percentages). Here, Φ_{it}^{IM} and Φ_{it}^{H} are computed as the school age cohorts in the GER regressions. The following countries are excluded from the sample in the savings regression because long-savings data were not available for New Zealand, Austria, Belgium, Greece, Ireland, Portugal, and Switzerland. The estimation period is 1870–2011. ***$p < 0.01$.

the increasing life expectancy induced by the cardiovascular revolution resulted in increasing GERs in the United States.

For primary GERs, the coefficients of Φ^{IM} and Φ^{H} are insignificant in the 2SLS regressions, which is unsurprising since the primary education path has been influenced heavily by the increasing years of compulsory schooling, the introduction of minimum working ages in the 19th century, and the increasing complexity of production [Murtin and Viarengo (2011), Madsen (2014)]. One would, therefore, expect Φ^{IM} and Φ^{H} to be less significant in explaining primary than secondary and tertiary education. This reasoning is also consistent with the finding that the coefficients of Φ^{IM} are higher for GER^{T} than GER^{S}.

Research intensity is regressed on cohort infant mortality and height in column (4) in Table 6. The coefficients of Φ^{IM} are significantly negative in the OLS as well as the 2SLS regressions, whereas the coefficients of Φ^{H} are negative but significant only in one of the two cases. It is not clear why one of the coefficients of Φ^{H} is significantly negative: It may reflect omitted variables that are significantly correlated with Φ^{H} and that, unlike Φ^{IM}, Φ^{H} is chiefly a proxy for physiological health and not cognitive skills. In any event, the net effects of a one standard deviation increase in Φ^{H} and decrease in Φ^{IM} yield the positive net effects of 66.6% (OLS) or 154.3% (2SLS) on research intensity.

Turning to the national savings rate, S/Y^{N}, in column (5) the coefficients of Φ^{IM} are significantly negative as expected, and the coefficient of Φ^{H} is significantly positive as expected in the OLS regression and is insignificant in the 2SLS regression. Cohort infant mortality may influence savings because of delay discounting. Several studies demonstrate that delay discounting is inversely related to IQ [Jones (2011)]. In other words, people with a low IQ tend to have a high time-preference and, therefore, prefer smaller and earlier rewards to larger, later ones.

The fertility regressions, which are displayed in column (6), indicate, as expected, that fertility is significantly positively related to Φ^{IM} and significantly negatively related to Φ^{H}, suggesting that the reduced cohort health insults have been potentially influential for the fertility transition in the OECD countries. We get the same results in the working-age mortality regressions in column (7) in which mortality is significantly positively related to Φ^{IM} and significantly negatively related to Φ^{H}. These results are consistent with the Barker (1990) hypothesis that intrauterine growth retardation, low birth weight, and premature birth have a causal relationship to the origins of hypertension, coronary heart disease, and non-insulin-dependent diabetes, in middle age. Note, however, that Sargan's test is significant at the 5% level in this regression, which suggests that the instruments may not be exogenous, perhaps because a high-mortality environment bolsters medical research that seeks to reduce mortality.

The influence of cohort infant mortality and height on the strength of democracy is tested in column (8) in Table 6. Here, I would expect the coefficient of Φ^{IM} to be negative and that of Φ^{H} to be positive because lower cognitive skills induced by cohort health insults tend to lead to lower political participation. Rindermann (2008), for example, argues strongly that IQ is highly influential for

political participation, critical thinking, rational behavior, questioning traditional dogmas, good political decision making, effective administration, and effectiveness of governance, among others. Democracy is measured as voter participation in elections as compiled by Vanhanen (2003), where the voter participation in elections is estimated in proportion to the population and not in proportion to the electorate because the electorate often can exclude a large disenfranchised fraction of the population. The coefficients of cohort infant mortality and cohort height are highly significant and of the expected signs (Φ^{IM} negative and Φ^{H} positive), giving some support to Rindermann's hypothesis that the democratization over the past two centuries has been influenced positively by an increasingly informed and cognitively able population. Again, however, care needs to be exercised in the interpretation of the results as Sargan's test indicates that the instruments may not be exogenous.

The life expectancy regressions in column (9) are consistent with those of working-age mortality, in that life expectancy is significantly negative related to Φ^{IM} and significantly positively related to Φ^{H}, regardless of whether Φ^{IM} and Φ^{H} are instrumented. Again these results are consistent with the Barker (1990) hypothesis that morbidity in early life leaves permanent scars and influences mortality later in life.

Finally, the regressions in the last column of Table 6 give mixed signals about the effects of health insults on population growth. The coefficients of cohort height are insignificant in the 2SLS regression and significantly negative in the OLS regression. The coefficients of cohort infant mortality, by contrast, are significantly positive in the 2SLS regression but significantly negative, but only at the 10% level, in the OLS regression. From this, it can be concluded that there is some weak evidence pointing toward a positive association between cohort health insults and population growth and, therefore, that the epidemiological transition in the OECD countries reduced rather than increased population growth. The evidence, however, is far from conclusive given the somewhat conflicting results and the possibility that the instruments may not be exogenous as indicated by Sargan's test.

Thus, it appears the health-insult-induced fertility decline over the past 142 years may have had a stronger effect on population growth than the negative working-age mortality effects of the reduced cohort health insults. However, the regression results are not sufficiently clear-cut to make strong claims about the relationship between health insults and population growth. Note also that these results do not contradict the finding of Acemoglu and Johnson (2007) and Hansen and Lønstrup (2015) because these authors focused on the fraction of the epidemiological transition driven by the invention of penicillin and the subsequent commercialization of antibiotics during the late 1940s, public health inventions supported by the establishment of the World Health Organization, and a change in international values [Acemoglu and Johnson (2007)]. The focus here, by contrast, has predominantly been on the economic effects of the improvements in the hygienic standard prior to the commercialization of antibiotics. Furthermore,

the sample used here is composed of the post-transitional countries and, as such, excludes the countries that have not finalized their demographic transition. As shown by Cervellati and Sunde (2011), life expectancy has a positive effect on per capital income for post-transition countries, whereas it has a negative effect on per capita income in countries that were at their pre-transitional stage in 1940. The key insight in their paper is that the causal effects of life expectancy on per capita income differ systematically during different phases of demographic transitions.

7. DISCUSSION AND CONCLUDING REMARKS

One of the leading issues in the economic growth literature has been to identify the driving forces behind the increasing productivity growth rates in the 20th century following the transition from the post-Malthusian to the modern growth regime. This paper argues that the high burden of PIDs, malnutrition, iron and iodine deficiencies, and lead poisoning before the turn of the 20th century potentially impaired the cognitive and physiological capabilities of the population, on average. It is further argued that the improvement in these health factors may have been important forces behind the productivity increases in the advanced countries over the past $1\frac{1}{2}$ centuries.

To test this proposition, cohort infant mortality and height indexes were constructed as proxies for health insults and malnutrition experienced during the first 20 years of life and it was shown that improved health had been a major contributor to the productivity growth in the OECD countries over the period 1865–2011. Medical innovations, real food prices, and public infrastructure investment were used as instruments for infant mortality and height to alleviate potential feedback effects of economic development on infant mortality and height. Furthermore, the following confounding cohort variables, which transitioned at approximately the same time as infant mortality and height, were included in the regressions to ensure that cohort infant mortality and cohort height were not capturing the productivity effects of these confounding variables: educational attainment, fertility, institutional quality, financial development, and innovations. Although the economic significance of cohort infant mortality and height was reduced by the inclusion of these confounding variables, they have, nevertheless, accounted for more than a third of the average productivity growth in the OECD countries since 1870, thus explaining a large share of the increasing growth rates following the transition from the post-Malthusian growth regime to the modern growth regime.

It has been hypothesized that the declining health insults and nutrition in childhood as proxied by cohort infant mortalities and increasing height have been influential for the productivity advances in the OECD countries because they have been associated with increasing cognitive abilities, as principally captured by cohort infant mortality, and increasing physiological capital, as mainly captured by the height of the working-age population. The medical literature shows that cognitive ability may be permanently impaired by health insults and lack of essential micro nutrition, which, in turn, expresses itself in infant mortality

rates, and that persistent protein-energy malnutrition reduces physiological and, to some extent, neurological development. Unfortunately, I cannot say exactly the extent to which increasing IQ and improved physiological health have contributed to the productivity advances since 1865—the only thing I can say is that the reduction in health insults and malnutrition have been influential for growth since 1865 and that the associated increase in IQ has probably played a significant role for the productivity advances. It is not possible to firmly conclude that cohort health transmits to economic activity through cognitive and physiological improvements because no long-time-series data for IQ and physiological health currently exist.

There are, however, two pieces of evidence suggesting that improved IQ has been a significant factor behind the productivity gains in the advanced countries over the past 150 years and that declining cohort infant mortality has, at least to a first approximation, captured this development. First, the estimates suggest that the declining infant mortality over the period 1865–2011 has been associated with a gain of around 30 IQ score points for the average adult. Even if this number seems extremely high, it, nevertheless, falls short of the findings in the psychological literature. In his survey of the psychological literature on IQ changes over time, Flynn (1999) concludes that:

> data are now available for 20 nations, and there is not a single exception to the finding of massive IQ gains over time. . . . Recent data show that IQ gains in Britain began no later than the last decade of the 19th century. . . . All nations but Norway have shown gains at a rate of about 20 IQ points per generation (30 years). (pp. 26–27)

Second, cohort height and particularly cohort infant mortality were found to be highly significant determinants of school enrollment rates (GERs) at all levels, innovative activity, fertility, and democracy. Increasing IQ increases the returns to schooling and, therefore, gives stronger incentives to undertake education, particularly at the secondary and tertiary levels. The innovative activity is strongly influenced by the cognitive ability of the population and, particularly, of high achievers, and cognitive ability does not enter into the innovation functions in standard endogenous growth models because it is assumed that the average IQ is constant across populations and over time. Finally, political participation is highly influenced by cognitive ability and the positive significance of cohort health in the political participation regression points toward cohort health as an indicator of cognitive ability.

The argument that the increase in cognitive ability has been prominent for growth is consistent with the findings in the literature that IQ is highly influential for productivity. Based on cross-country regressions, Jones and Schneider (2006) demonstrate that IQ is the most robust determinant of growth of the factors that are generally considered as important for growth in the existing literature. These results reinforce the hypothesis in this paper, which suggests that reduced infant morbidity, as evidenced by reduced infant mortality that accelerated at the end of the 19th century, has been influential for growth through the channel of increasing

cognitive ability and health capital among the surviving population during their working life.

The large productivity effects of cohort infant mortality found in the preceding section raise the question as to why the macro effects exceed those of microeconomic studies. Microeconomic studies typically find that an increase in IQ by 1% is associated with an increase in earnings of approximately 1% [see, for a survey, Jones (2011)]. However, there are at least three reasons for large externalities to cognitive skills that do not show up in microeconomic studies. First, macroeconomic studies have demonstrated that the social returns to investment in research and development (R&D) and education exceed their private counterparts [see, e.g., Madsen (2010)]. The regressions above showed that cohort infant mortality and height were influential for education and innovative activity, indicating that health has large externalities on productivity through R&D and education. Second, Jones (2011) argues that high-IQ leads to better cooperation between economic agents by referring to behavioral economics experiments showing that high-IQ players are more cooperative in repeated prisoner's dilemma, trust, and public goods games than low-IQ players. Third, as shown by Kremer (1993), skill complementarities are important in producing O-Ring forms of fragile, delicate output. Small differences in worker skill may cause marked differences in cross-country productivities because weak links in the production process can have large macro effects.

The findings that the health transition has been a significant force behind the historically high growth rates experienced by today's advanced countries in the second half of the 20th century do suggest that growth rates in the advanced countries will slow down in the future and perhaps even revert to their post-Malthusian growth rates as cohort health is showing signs of leveling out. However, it is limited how low the productivity growth rates can fall, unless R&D as a percentage of GDP (research intensity) is reduced. As shown by Madsen (2010) and as predicted by Schumpeterian theories of economic growth [see, e.g., Peretto (1998, 2015)] productivity growth is explained well by research intensity, implying that R&D-induced growth rates will remain steady as long as research intensity is kept at a steady level.

NOTES

1. The dependent variable is not lagged in the regressions because slow adjustment is catered for in the regressions in the 5- and 10-year intervals. Furthermore, the principal results remain unaltered if the dependent variable is lagged one period in the 1-year regressions.

2. Ideally, we would need an indicator of infant morbidity to capture the development of health capital during early years of life. However, the only currently available indicator of morbidity is disability-adjusted life years (DALY) lost due to diseases and injuries in the 0–14 year age group; however, this indicator has only very recently become available and it is not available for groups younger than the 14 years of age. Regressing the DALY among children in the 0–14 year age group due to infectious, respiratory, and perinatal diseases in 2006 on infant mortality for 188 countries in 2006 from WHO (2008) yields t-statistics between 10 and 19 and correlation coefficients between 0.8 and 0.9, thus giving strong support to infant mortality as an excellent indicator for morbidity, even

when infant mortality refers to the age of zero, whereas the DALY data refer to the 0–14 year age group.

3. Over the period 1916–1950 in the United States, the infant mortality due to diarrheal mortality declined by 95.5% [Lee (2007)]. In Norway, from 1899–1902 to 1936–40, the infant mortality due to PIDs declined by 84% (Statistisk Årbok, Statistisk Sentralbyrå, 1961, Tab. 38). Infant mortality due to PIDs in Paris declined by 54.4% from 1886–90 to 1901–03 (Département Seine, 1904, Direction des Affaires Municipales, Bureau d' hygiène ville Paris. 1e règlement sanitaire ville Paris). In Italy, infant mortality due to PIDs declined by 81.1% over the period from 1887 to 1955 (Istituto Centrale di Statistica, 1958, Cause di morte; 1887–1955, The Institute, Rome).

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