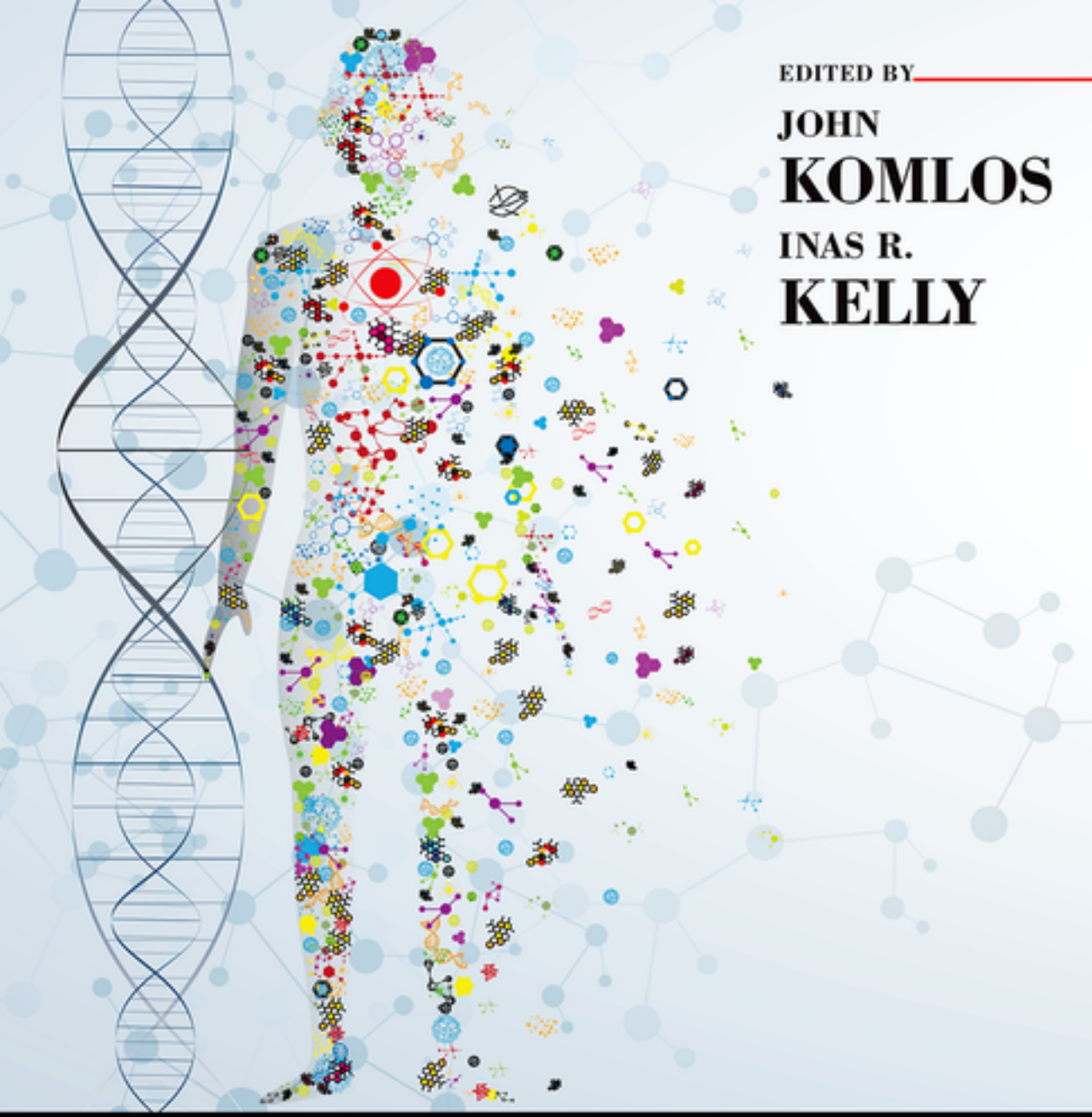

EDITED BY

JOHN KOMLOS

INAS R. KELLY

The Oxford Handbook *of* ECONOMICS AND HUMAN BIOLOGY

THE OXFORD HANDBOOK OF

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PART I

INTRODUCTION TO ECONOMICS AND HUMAN BIOLOGY

INTRODUCTION

INAS R. KELLY AND JOHN KOMLOS

The *Oxford Handbook on Economics and Human Biology* explores a relatively new and expanding field of economics that makes use of biological insights and introduces the reader to the main approaches, insights, and results obtained in this area of research over the previous three decades. We ask questions such as: what role does anthropometrics have in determining economic outcomes? In turn, what roles do economic factors play in transforming anthropometrics over time? The primary focus of this interdisciplinary volume is on examining in detail how economic processes affect human biological outcomes—and the other way around: how biological outcomes affect economic processes. By human biology, we mean all aspects of the organism that are measurable and for which data are available. These include such measures as height, weight, the body mass index (BMI, constructed from height and weight), cholesterol levels, blood pressure, and birth weight. This perspective differs from health and health economics insofar as health is usually defined in terms of the absence of disease, whereas the economics and human biology research program is concerned with the functioning of the human biological system and how it is impacted by its socioeconomic environment.

The volume has three main purposes. The first is to introduce the reader to the research on biological outcomes of historical and contemporary populations and to explore the extent to which human biology is affected by economic processes over time and cross-sectionally. The second is to focus on biological markers (such as height and weight) as inputs, highlighting labor market aspects that emphasize how biological markers (such as body composition) affect labor market outcomes including wages, unemployment, and wealth. The extent to which these biological markers affect labor productivity and human capital accumulation will also be explored. The third purpose is to introduce the reader to developmental aspects and policy, particularly correlates of malnutrition and poverty. The policies that have been introduced worldwide to address these deficits are explored in some detail.

The roots of the systematic study of the relationship between human biology and the economy in which it is embedded go as far back as 1829, when Louis R. Villermé, a statistician of public health, recognized that a population's physical stature was influenced by

economic factors. In the modern period, the field of anthropometric history was born in the mid-1970s with Richard Steckel's studies on slave heights and those of Robert Fogel on the nutritional factors in the demographic transition. The field really expanded after Fogel received the Nobel Prize in economics in 1993. Since then, the field has taken off substantially.

Analyzing biological and economic measures together enables us to gain valuable and hitherto unknown perspectives on the well-being of populations, both historical and contemporary. Some of the topics explored are the momentous changes associated with economic growth and transformations such as the Industrial Revolution, the onset of modern economic growth, and globalization. A basic premise is that as the economy affects our pocketbook and our lifestyles, its effects actually penetrate deep into our cells, our organs, and our bones. It does so, for instance, through the food we eat which, in turn, is obviously a function of incomes, relative prices, agricultural productivity, the organization of markets, and all the other salient aspects of the economy. These economic variables thus have significant effects on our bodies at the microlevel, affecting weight, comorbidities, and height. These biological measures in turn have effects on labor market outcomes and productivity. The effects of crisis (manmade and natural) can shed light on these effects, and differences across native populations and migrants can also be revealing in this context.

At the macrolevel, population density, urbanization, inequality, social and occupational structure, taxation, government redistribution, poverty, entitlements, the disease environment, and the organization of medical services all play a role. These various aspects of institutional development are shown to have significant effects on structural changes in biological measures over time and across geographic regions, and vice versa. It is not necessarily standard to think of these macroeconomic measures as being linked to overall anthropometric measures in a country, and yet they play an important role, particularly from a policy perspective.

Through the strong linkages between economics and human biology, policy makers may wish to redirect resources to alleviate conditions such as obesity and early-life growth faltering. The advantages to this approach run deep. If the economic returns to investing in nutrition, well-being, infant health, and other biological measures are quantifiable, ignoring the salient role that they play would be a dire omission in the economics literature. From a policy perspective, government intervention is justified in economics if externalities are present, as Averett and Wang argue with malnutrition in their chapter in this handbook. In fact, market failure may be less the exception and more the rule, particularly when you factor in considerations surrounding human biology. Offer argues in his chapter that market-liberal welfare regimes, for example, have contributed to the high prevalence of obesity.

The extent to which human biology plays a role in economic development needs to be better understood. If severe inequities arise with income and wages, for example, due to disparities in height, weight, endowment at birth, and the like, the reasons for this need to be explored in order to better understand the workings of economies. If this is due to discrimination, then optimal matches in the workplace are not occurring, and

productivity is lower than optimal. In other words, individuals are not being paid the value of their marginal product of labor. Moreover, the mental health effects in this context cannot be ignored. Differences can arise in health (both physical and mental), in quantity and quality of education, in the labor market, in levels of risk aversion, and they can arise across race, gender, and sexual orientation. An understanding of these disparities can also shed light on the transfer to welfare across generations, as argued by Oxley in her chapter in this handbook. These are all issues that the field of economics and human biology attempts to address, filling a critical gap in the general research in either economics or biology alone.

The use of econometrics for the empirical testing of theories in economics and human biology allows researchers to carefully address issues of self-selection, heterogeneity (both observed and unobserved), endogeneity, and confounding factors. As Colman and Dave argue in their chapter, randomized controlled trials, the gold standard of causal research, are often impossible or unethical in this context. Having alternative tools allows us to establish causal effects using the observational data most often available. The emerging use of biomarkers and twin studies, as shown in this handbook, have also become useful as tools in this field.

The interdisciplinary nature of this handbook is paramount and one of its greatest strengths. Economists, anthropologists, historians, biologists, biochemists, physicians, environmentalists, and researchers in public health and public policy, as well as others often all have similar questions and yet approach them from different perspectives using different methodologies. Often they use terminology specific to their disciplines, thus placing an obstacle to collaboration with researchers from other disciplines. Yet it is this very cross-fertilization that can be very fruitful and allow us to arrive at the answers we seek. The study of economics and human biology also needs to be viewed through the dimension of space and time in order provide an accurate portrait of the variation exhibited both historically and geographically. You will thus find several chapters in this handbook that discuss historical perspectives, as well as some that focus on specific geographical areas.

The handbook is divided into four parts. The first part, the Introduction to Economics and Human Biology, provides the reader with a general background on the topic. Four chapters after the introductory chapter provide a background on anthropometrics: Behrman's chapter on growth faltering and catch-up growth, Steckel's chapter on measuring well-being with biological indicators, Bharadwaj and Vogl's chapter on economic crisis and human biology, and finally Koepke's chapter, which follows the biological standard of living in Europe from the Late Iron Age to the Little Ice Age. Next, the chapter by Colman and Dave explores econometric methods used in empirically identifying relationships in how economics impact the human organism.

The second part of the handbook is on biological measures as outcome variables. Here, measures such as height, weight, body mass, and general biological well-being are analyzed using a number of variables, many reflecting changing economic conditions over time. The first group of papers in this part explores weight, height, and obesity. We begin with Carson's chapter, which explores changes in the body mass index through

time. Lakdawalla and Reif's chapter explores economic reasons for why body mass has increased substantially over time. Blum's chapter on inequality and heights explores determinants of inequality in heights in detail. Rosinger and Godoy's chapter on native populations and Steckel's chapter on slave heights add important dimensions regarding subpopulations.

Next, female biological well-being is addressed. The important role of height as an indicator of women's health, productivity, and welfare is examined from an historical perspective in Oxley's chapter.

The next two chapters in this part of the handbook focus on the young: infants, children, and adolescents. Cabieses, Pickett, and Wilkinson's chapter stresses the role of socioeconomic inequality, whereas Rogol's chapter on auxology, the study of growth and development, highlights genetic and environmental factors responsible for the growth and maturation—or lack thereof—of children and adolescents.

The last group of chapters in this part summarize the importance of taking a global perspective for many of the topics explored thus far. Meinzer and Baten's chapter takes a historical perspective and follows global trends and cycles in physical stature from the Neolithic Agricultural Revolution to modern times. Kromeyer-Hauschild, Moss, and Wabitsch provide an overview of trends in BMI across the world and over time, whereas the focus is on the dual burden of poverty and obesity in developed countries in Meyerhoefer and Yang's chapter.

The third part of the handbook introduces biological measures as key independent variables and determinants of economic outcomes. Genetic markers, a new tool available to researchers, are covered in the first two chapters: both Lehrer's and Wehby's chapters provide insights into the burgeoning role of genetic biomarkers and genes as inputs in determining economic outcomes. Behrman's chapter then provides a thorough introduction to the economics of twin studies; it describes the strengths and limitations of various twins methods developed in economics.

The next chapters in this part explore investments in health. Alderman and Sahn's chapter examines the impact of nutrition on productivity and the general economic returns of investing in proper nutrition. The double burden of malnutrition—that both undernutrition and obesity are drivers of adverse health outcomes—is explored in Averett and Wang's chapter on a topic that is increasingly important globally, especially in developing economies. Biological health risks (such as hypertension, cholesterol, high glucose levels, and inflammation) in the context of economic development are explored in Frankenberg, Ho, and Thomas's chapter.

We then turn to monetary outcomes of biological attributes. Su's chapter provides a global perspective—with a focus on countries belonging to the Organization for Economic Cooperation and Development (OECD)—by examining the effect of obesity on income inequality. Hübler's chapter summarizes the literature on the effect of height on wages and provides an extensive theoretical background. The effects of body mass on both wages and wealth are then explored in Greve's and Zagorsky's chapters, respectively, with possible reasons for the prevalent negative relationship found in several studies. Wilson's chapter on family economics and

obesity reminds us that the economics of obesity should be studied within the context of the family, especially with childhood obesity on the rise in economically prosperous nations.

Obesity and welfare regimes are then discussed with Offer's chapter on how market-liberal welfare regimes tend to have the highest prevalence of obesity. The welfare regime hypothesis put forth in this chapter argues that obesity may be a response to stress and that stress is generated by market competition; more specifically, by the uncertainty that the market competition generates. Reasons for this uncertainty are explored in detail in this chapter.

We then turn to child and infant anthropometrics. Silventoinen's chapter discusses children's anthropometrics and later disease incidence, highlighting the adverse effects of low birthweight and short stature. Birthweight as a health indicator is discussed in further detail in Ward's chapter. Torche and Conley's chapter extends the discussion on birthweight as a widely used yardstick for welfare by analyzing its use as a measure of human capital endowment in economic research. These two chapters on birthweight highlight its role in medicine, biology, and economics.

The last chapter in this part is on neuroeconomics, a flourishing field that promises new insights into the field of economics and human biology. Aimone and Houser's chapter provides a summary of this burgeoning field, the limitations of standard economic theory, and how neuroeconomics can help explain behaviors such as trust, reciprocity, and betrayal.

The fourth part of the handbook encompasses regional studies in economics and human biology. The focus is on Africa in Hirvonen and Moradi's chapter, which explores the phenomenon of surprisingly tall African men and women in spite of low incomes. The focus then turns to Asia in Schwekendiek's chapter, which highlights the rich history of anthropometric growth over time in China, Japan, and Korea and the role that economic performance plays. We turn to North America in both Boustan and Margo's chapter, which discusses racial differences, and Craig's chapter, which provides reasons for the "antebellum puzzle"—the mysterious finding that the American population became shorter at the onset of modern economic growth in the decades prior to the US Civil War.

This finding dovetails with a literature that is critical of the excessive emphasis on gross domestic product (GDP) growth as the primary if not the sole indicator of living standards (Stiglitz, Sen, & Fitoussi, 2010). In contrast, the anthropometric evidence at the onset of modern economic growth emphasizes that a rising GDP was compatible with a decline in nutritional status of children and youth who had no agency to determine their own destiny. And these are exactly the age groups that are almost always left out of conventional analysis inasmuch as monetary measures pertain exclusively to adults and not to children and youth who are not employed. That is another major distinction between conventional views and anthropometric views of welfare. Methodological individualism is very concerned about agency, but those who were hurt by economic growth in the Antebellum United States had no agency at all, and that is worth emphasizing. Hence, economic growth was by no means a Pareto-optimal

process. Instead, there were gainers and losers. That is also a new and useful insight into the dynamics of economic development.

A'Hearn's chapter presents an anthropometric history of the Mediterranean world. These insightful chapters on anthropometrics and economic performance in different regions across the globe conclude this interesting, novel, and interdisciplinary volume that we hope will be a useful resource for researchers in economics and other social sciences. As editors of this handbook, we feel privileged to be able to present the reader with this impressive body of research and hope it will serve as a valuable resource for a long time to come. Finally, we would like to thank David Pervin for his vision, guidance, perseverance, and patience in bringing this volume to fruition.

Reference

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CHAPTER 1

GROWTH FALTERING IN THE FIRST THOUSAND DAYS AFTER CONCEPTION AND CATCH-UP GROWTH

JERE R. BEHRMAN

1.1 Background

Early-life growth faltering is of considerable concern for many low- and middle-income countries (LIMICs). Average growth paths for children in undernourished populations typically indicate birth lengths/weights below medians for well-nourished populations, then fairly sharp relative declines until leveling off around 2 years of age, and possibly a slight catch-up with further aging (Figure 1.1). Approximately 170 million children under 5 years old, mostly in LIMICs, suffer growth faltering as measured by being stunted (i.e., heights/lengths <2 standard deviations [SD] below World Health Organization [WHO, 2006] medians for well-nourished populations; de Onis et al. [de Onis, Blössner, & Borghi, 2011]). Growth faltering has prevalences of approximately 40% in a number of South Asian and sub-Saharan African countries (de Onis et al., 2011). Recent surveys indicate that early-life growth faltering is associated with less satisfactory outcomes in education, labor markets, marriage markets, health, and parenting (e.g., Victora et al., 2008, summarized herein).

Prominent studies based on such associations argue for redirecting resources to alleviate early-life growth faltering, particularly during the first thousand days (FTD) after conception (up to 2 years of age). Martorell et al. (Martorell, Khan, & Schroeder, 1994), for example, conclude that catch-up growth after early childhood might occur in some

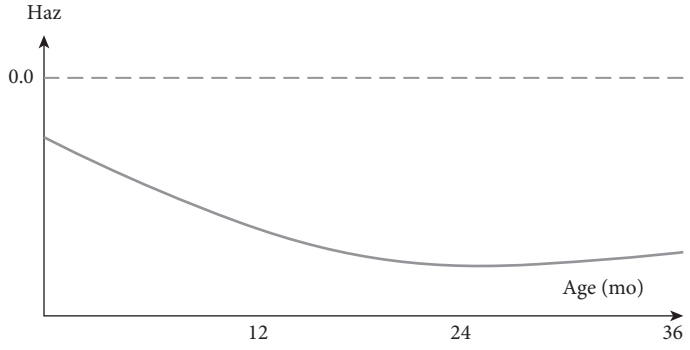

FIGURE 1.1 Stereotypical average HAZ versus age in undernourished population, with median HAZ = 0 for well-nourished population.

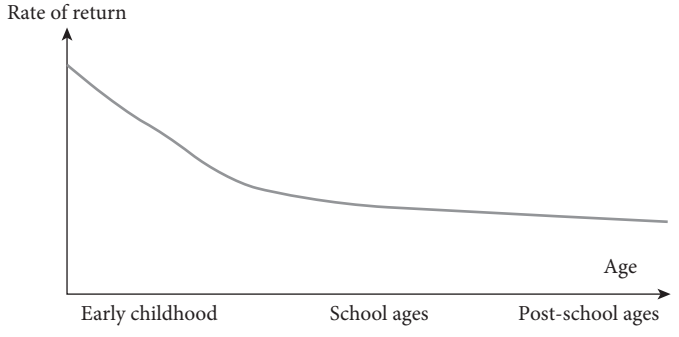

FIGURE 1.2 Rate of return to investments versus child age, as suggested by Heckman (2006).

circumstances but that "subjects who remained in the setting in which they became stunted experience little or no catch-up in growth later in life." Victora et al.'s (2008, p. 340, emphasis added) influential *Lancet* article stated that their first key message is "Poor fetal growth or stunting in the first 2 years of life leads to *irreversible* damage, including shorter adult height, lower attained schooling, reduced adult income, and decreased offspring birthweight." Victora et al.'s (Victora, de Onis, Hallal, Blössner, & Shrimpton, 2010, p. e473) *Pediatrics* article summarized their study of child growth patterns in 54 countries: "Children from low- and middle-income countries are born with weights and lengths below WHO growth standards, and early growth faltering is even faster than currently assumed. The window of opportunity for preventing undernutrition ends at 2 years of age." These conclusions resonate with Heckman's (2006) claim that rates of return are highest for early-life human capital investments and decline as children age (Figure 1.2).

This chapter considers selected empirical evidence on impacts of FTD growth faltering and possible subsequent catch-up growth and addresses those questions that merit further investigation.

1.2 Life Cycle Framework of FTD Growth Faltering and Catch-Up Growth

Figure 1.3 presents a simplified framework with six life-cycle stages: (1) the FTD, (2) subsequent early childhood (i.e., from 2 years to normal school-entry ages), (3) late childhood, (4) adolescence, (5) adulthood (including intergenerational effects through fertility and parenting), and (6) old age. Children start life with endowments, both genetic and otherwise. Conditional on these endowments, FTD development (upper right box) is affected by risk factors (upper left box) including malnutrition, infections, pregnancy and birth complications, and inadequate stimulation/nurturing. These risk factors are mitigated by familial inputs (e.g., family-provided stimulation, nutrition) and community inputs (e.g., accessibility and quality of day care, preschool programs, and health care). The FTD outcomes (upper right box) in turn affect outcomes in subsequent sequential life cycle stages that may be exclusively for the children under consideration and their families or may include spillovers on others. FTD growth faltering (e.g., stunting) is the most common measure of the long-run nutritional status component of the physical status outcome. Familial and community inputs (center left box) may moderate the four FTD risk factors and their impacts on outcomes such as FTD growth faltering and how FTD outcomes produce impacts over the life cycle. These investment inputs occur for each stage within particular contexts and have associated resource costs. Across contexts, the effects of these investments and their resource costs are likely to vary.

Caregivers (mainly parents) make decisions that affect child developmental outcomes, including FTD growth faltering. Outcomes in subsequent life cycle stages are conditional on FTD outcomes (or inputs that determine them), subsequent life cycle familial and public inputs, and stochastic factors (e.g., variations in disease/nutrition/cognitive environments). Production functions give vectors of outcomes for the s^{th} life cycle stage (Y_s) dependent on a matrix of familial production inputs from conception to the s^{th} life cycle stage (F_s), a matrix of community-level production inputs from conception to the s^{th} life cycle stage (C_s), a vector of genetic and other endowments at conception (E_o), and a matrix of stochastic production factors from conception to the s^{th} life cycle stage (U_s):

$$Y_s = Y_s\left(\mathrm{F}_s, \mathrm{C}_s, \mathrm{E}_0, \mathrm{U}_s\right). \tag{1}$$

Production function features highlight several issues: (1) endowments at conception and the history of familial, public, and stochastic production inputs since conception may matter, so right-side matrices have multiple rows for life cycle stages since conception and multiple columns for inputs in each stage; (2) within life cycle stages, inputs

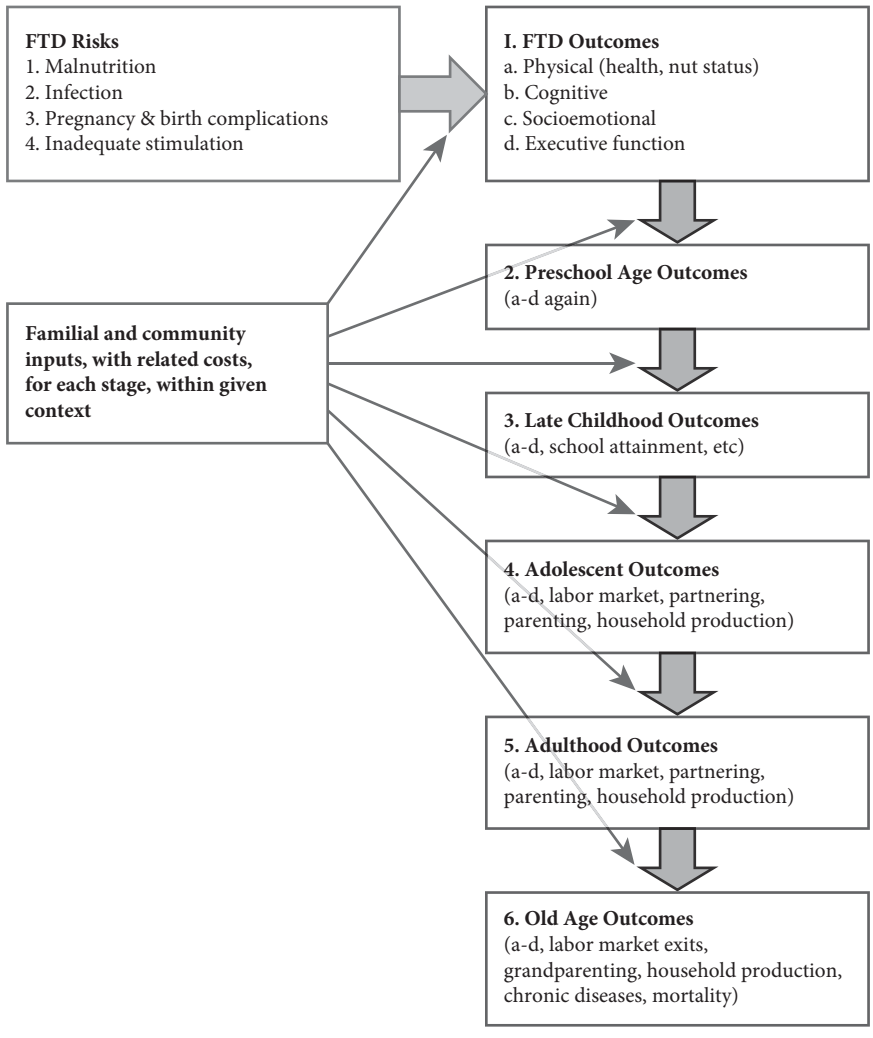

FIGURE 1.3 First thousand days (FTD) growth faltering within life cycle framework.

may compensate or reinforce each other; and (3) across life cycle stages, inputs may compensate or reinforce each other through dynamic complementarities (Cunha & Heckman, 2007). Better FTD nutrition-related inputs, for instance, are hypothesized to improve learning through stimulation at that stage and through preschool or school programs in later life cycle stages.

Production functions are only technical relations, including biological aspects, between inputs up to life cycle stage *s* and child outcomes in life cycle stage *s*. But what determines inputs is critical. Within standard economic models, familial inputs are determined by familial decisions (perhaps with some intrafamilial bargaining) that aim to maximize expected familial welfare (perhaps with different weights on different family members) subject to the production functions in relation (1), a matrix of welfare function

preferences (W_s), a matrix of knowledge about technology and markets (K_s), a matrix of prices and policies that the household has faced and expects to face in the future (P_s), a matrix of resources that the household has had and expects to have in the future, including child endowments in Equation (1) (R_s) and a matrix of stochastic shocks, including those in Equation (1), that the household has faced and expects to face in the future (V_s):

$$F_s = F_s\ (\mathrm{W}_s, \mathrm{K}_s, \mathrm{P}_s, \mathrm{R}_s V_s)\,. \tag{2}$$

There are six dimensions to these demand relations:

1. Right-side variables refer not only to periods from conception to the s^{th} stage, but also, if investors are forward-looking, include expected future distributions (or parameters underlying such distributions).
2. If right-side variables vary across child life cycle stages, then relations need to be added to represent how they evolve over time (e.g., preferences may depend on communication campaigns, knowledge may depend on investments in learning, policy-related factors may depend on whether relevant governments allocate public inputs to favor the better-off because of their political influence or the poorer because of anti-poverty concerns).
3. Through substitution into Equation (1), demand relations can be obtained for outcomes in stage s (Y_s).
4. Behavioral decisions are critical in determining outcomes and whether they compensate or reinforce prior differences (e.g., if parents have strong "inequality aversion" [Behrman, Pollak, & Taubman, 1982] regarding outcome distributions among their children, a program that provides nutrients to other than the most malnourished of their children might induce considerable reallocation of parental resources from the target child to other children).
5. To evaluate the total impact of FTD growth faltering or of interventions to reduce such faltering, various individual impacts over the life cycle must be combined (with care not to double count; e.g., if one impact such as schooling attainment is primarily a channel through which growth faltering or interventions affect adult productivities) by using weights (real prices) for individual impacts (some of which, e.g., adverting mortality,[1] pose challenges) and discounting back to the FTD. For example, US$1,000 in 40 years—when some productivity effects may occur—with a 3% discount rate has a present discounted value [PDV] of US$307 (US$252 with adjustment for Indian survival probabilities); with a moderate discount rate of 6%, this is US$97 (US$80).
6. To estimate benefit–cost ratios (or internal rates of return) to interventions to reduce FTD growth faltering, the PDV of total resource costs (including private and public resource costs and distortion costs but not transfers) of the intervention and costs induced by the intervention (e.g., if increased schooling attainment is induced years later, there are likely to be resource costs for this additional schooling) need to be calculated.

Finally, estimation of Equations (1) and (2) is very challenging because of tremendous information requirements (many variables, some of which are difficult to observe, over many years), random and systematic measurement errors in observed variables, and unobserved factors including endowments at conception that may affect both left-side and right-side variables and thus cause spurious correlations. FTD growth faltering is determined, inter alia, by endowments and familial investments (left-center box) and cannot practically—to say nothing of ethically—be distributed randomly (although public investments that affect growth faltering might be explored with randomized designs). The challenges are greater, moreover, if heterogeneous responses are allowed across contexts, families, or individuals. Studies provide estimates, although with considerable simplifications with regard to number of variables, number of periods covered, impacts of unobserved factors, treatment of right-side behavioral relations, functional forms, and homogeneity of parameters.

1.3 FTD Growth and Outcomes in Subsequent Life Cycle Stages

1.3.1 Direct Estimates of FTD Growth and Outcomes in Subsequent Life Cycle Stages

Several empirical approaches relate FTD growth directly to outcomes in subsequent life cycle stages. These can be considered special cases of conditional demand functions derived from Equation (2), in which outcomes at some later life cycle stage are conditional on indicators of FTD growth faltering. Common simplifying assumptions are:

(A1) First-order or main effects in Taylor-series expansions of conditional demand functions capture the essence of the phenomena being investigated. Therefore, these studies provide no illumination about static and dynamic interactions that are emphasized in recent economics literature.

(A2) FTD outcomes other than physical growth can be ignored, consistent with other FTD outcomes being uncorrelated with FTD growth—a strong assumption because all of these outcomes are determined primarily by the same family inputs, community inputs, and genetic and other endowments. This assumption also is consistent with FTD growth serving as a proxy for correlated components of other outcomes, which leads to interpretation problems because *ceteris paribus* coefficient estimates for FTD growth represent not only causal impacts of FTD growth but also of correlated components of other FTD outcomes.[2]

I now summarize selected studies on FTD growth and later-life outcomes that fit into this general simplified framework, some of which depend on further simplifications.

1.3.1.1 *Longitudinal Associations between FTD Growth Faltering and Later Life Outcomes*

Most related studies in the nutritional and public health literatures that cover fairly long life cycle segments are associational. Perhaps the most prominent of these, Victora et al. (2008), is based primarily on the COHORTS data that cover at least 15 years of life cycles for undernourished birth cohorts with sample sizes of 1,000+ for subpopulations defined by specific geographical locations when born in Brazil, Guatemala, India, the Philippines, and South Africa (Richter et al., 2012). Table 1.1 summarizes associations of FTD height/length (cm or height-for-age *z*-scores [HAZ]) and weight (kg or weight-for-age *z*-scores [WAZ]), which are significant for various outcomes. As noted earlier, the authors state that their first key message is that "Poor fetal growth or stunting in the first 2 years of life leads to irreversible damage, including shorter adult height, lower attained schooling, reduced adult income, and decreased offspring birthweight." This conclusion depends on interpreting associations between FRD growth and later-life outcomes as causal, which depends on assumptions (A1) and (A2) and on further assumptions:

(A3) FTD growth indicators are sufficient statistics for everything through the FTD. That is, there are no inputs into FTD growth that have impacts on later-life outcomes that occur through channels other than through FTD growth. For instance, genetic endowments relating to innate health and innate abilities have no *direct* impact on later-life outcomes such as adult health and cognitive skills, but only *indirect* impacts through FTD growth outcomes (or, if A2 is not maintained, through other FTD outcomes).

(A4) Familial and community inputs for life cycle stages subsequent to the FTD that are correlated with those during the FTD do not affect later-life outcomes of interest, or, if they do affect these later-life outcomes, they are not correlated with the FTD growth indicator. That is, persistent familial factors (e.g., parental schooling attainment, long-run wealth or income) and persistent community factors (e.g., disease environments, accessibility and quality of social services) are assumed to not be correlated with FTD growth (even though a priori they would seem to determine FTD growth through Equation (2)), or they are assumed to have no post-FTD impact on any other variables that affect the outcomes of interest (e.g., if adult labor market outcomes are of interest, these persistent familial and community background variables are assumed to not affect any post-FTD learning or any other post-FTD channels that affect adult labor market outcomes).

Thus, the assumptions seem very strong for the Victora et al. (2008) conclusions about longer run effects of FTD growth failure based on their empirical analysis. They also present no evidence about the extent, impacts, and costs of post-FTD growth, which would seem necessary for their conclusion about irreversibility.

Table 1.1 Selected associations between first thousand days (FTD) anthropometric measures and adult outcomes

Schooling Attainment	0.5 Grades for 1 HAZ at Age 2
	0.5 grades for 1 WAZ at age 2
	0.3 grades for 1 kg at birth
Adult height	3.2 cm for 1 HAZ at age 2
	0.7–1.0 cm for 1 cm at birth
Labor income	8% for 1 HAZ at age 2 males
	8–25% for 1 HAZ at age 2 females
Birthweight of offspring	70–80 g for 1 HAZ or 1 of mother at age 2

Source: Constructed by author based on Victora et al. (2008)

1.3.1.2 *Birthweights and Subsequent Outcomes with Control for Endowments Shared by Identical Twins*

Monozygotic (MZ, identical) twin fixed-effects (FE) (or within-MZ twins) estimates control for all factors shared by identical twins including the life cycle stages of their parents and genetic endowments at conception.[3] MZ-FE estimates thus have advantages over the associations just discussed because they do not confound effects of persistent components of familial and community background and genetic endowments with impacts of FTD growth (i.e., they do not require assumptions A3 and A4). A prominent critique of MZ-FE estimates for estimating schooling impacts (the most widespread use of this method) is that between-MZ twins schooling attainment differences are assumed to be random but in fact may be due to other parental responses to differences between twins that may have direct impacts on outcomes of interest (e.g., birthweight differences associated with schooling differences and also with differences in outcomes subsequent to schooling; see Bound & Solon, 1999). This critique holds for FTD growth at 2 years, but not for birthweights because between-MZ twins birthweight differences do not reflect efforts by parents to favor one twin but are merely chance outcomes of placement relative to placentas. For the same reason, MZ-FE estimates of birthweight impacts on later outcomes are not confounded by persistent familial and community background characteristics shared by twins. Table 1.2 summarizes estimated impacts of birthweight (from birth certificates) on selected outcomes with comparisons of ordinary least squares (OLS) and MZ-FE estimates for US twins. Under the assumption that MZ-FE estimates give true causal effects of birthweight, the OLS positive significant associations with body mass index (BMI) and with birthweight in the next generation reflect substantial upward biases due to shared family and community background and genetic endowments that are controlled in MZ-FE estimates. Under the same assumption, on the other hand, the OLS positive associations of birthweight with schooling

attainment and particularly ln wages understate considerably the true (MZ-FE) effects. These results may reflect that genetic endowments are multidimensional, and those components associated with physical growth on average are inversely associated with those associated with intellectual development, so that OLS estimates are biased upward for BMI and birthweight and toward zero for schooling and wage rates (Behrman & Rosenzweig, 2004; Behrman et al., 2014). MZ-FE estimates for Chinese women indicate greater birthweight impacts on schooling attainment than for men because of women's comparative advantage in skill-intense occupations, thus illustrating that context matters (Rosenzweig & Zhang, 2013). The relevance of such results for broader populations requires:

(A5) Inferences can be made for broader populations from MZ-FE estimates. Debate is considerable about this assumption because twins are small percentages of populations, and being twins is perceived as being different. However, some of this debate is misplaced (e.g., the "common environment" assumption between MZ and DZ twins is critical for heritability estimates but not for MZ-FE estimates; to a first-order approximation, MZ-FE estimates control for what is different about being twins) or not reflective of recent developments in twins analysis (e.g., showing that if whatever causes differences between twins for right-side variables such as birthweights also has direct impact in the same direction on outcome variables of interest, then the MZ-FE estimates establish upper bounds on absolute magnitudes of true effects) (Kohler, Behrman, & Schnittker, 2011). Of course, one important respect in which twins differ from the general population is that distributions of birthweights for twins are to the left, although considerably overlapping, of those for singletons (Behrman & Rosenzweig, 2004). This means that the Table 1.2 results are more relevant than would be results from singleton populations for low birthweight babies of particular interest for catch-up growth.

Table 1.2 Monozygotic-fixed effects (MZ-FE)/ ordinary least squares (OLS) estimates for impact of birthweight over life cycle

	MZ-FE/OLS (%)
Schooling	210
BMI	28
Ln Wage	706
Child Birthweight	25

Constructed by author based on Behrman and Rosenzweig (2004)

1.3.1.3 *Birthweight and Post-FTD Outcomes for Synthetic Cohorts by Linking Estimates Across Life Cycle Segments*

FTD growth and outcomes in later life can be linked by several approaches,[4] including using longitudinal data that follow cohorts from early life over a number of years (as in the first study reported), linking data on adult life cycle stages with earlier birth records (as in the second study), or linking estimates from different studies across different life cycle stages.[5] The last approach requires an assumption related to (A3):

(A6) Linking variables across life cycle stages provide sufficient statistics for what preceded them. That is, if one study linking birthweight to schooling attainment is combined with another study linking schooling attainment to adult productivities, then schooling attainment is a sufficient statistic for all birthweight impacts up to the end of schooling that may affect adult productivities.

Table 1.3 summarizes estimates of impacts of moving children from low birthweight to normal birthweight status in low-income contexts using the synthetic-cohort approach and the best estimates that the authors could find for each linked life cycle segment (based on randomized controlled trials or econometric methods) to avoid omitted variable biases (e.g., due to unobserved genetic endowments-related innate health and abilities). Other than an illustration of this approach, these estimates illustrate three important points. First, particularly for outcomes later in the life cycle, discount rates matter. Second, even with discounting at the usual rates, outcomes later in the life cycle may be very important in judging impacts of reducing FTD growth faltering, particularly if they persist over long life cycle segments (e.g., persistent productivity effects). Third, whether interventions to reduce low birthweight babies make economic sense depends in part on costs.

1.3.1.4 *Instrumental Variable (IV) Estimates of HAZ or Stunting at 24 Months and Multiple Outcomes into Adult Life Cycle Stages*

Hoddinott et al. (2013*b*) investigated impacts of FTD growth faltering on a more comprehensive set of outcomes than in any other previous prospective study, including human capital (schooling, intelligence, reading), marriage, fertility, adult health (cardiovascular disease risk factors, physical performance), labor markets, and household poverty. They used an IV approach to attempt to control for endogeneity and measurement error in their FTD growth indicators. They use the INCAP Guatemalan data, based on random assignment of more (protein-dense) and less (no-protein) nutritious supplements available free at community centers in four villages in 1969–76, which led to different exposures during the FTD depending on village and birth timing. The sample villages had experienced considerable prior FTD growth faltering by 24 months, with mean HAZs of less than −3 and stunting prevalences of 86%. They use

Table 1.3 Estimates of present discounted values in US dollars of seven major impacts of moving one infant out of low birthweight status in a low-income developing country

	Annual Discount Rate (%)		
Impacts	3	5	10
1. Reduced Infant mortality	$95	$99	$89
2. Reduced neonatal care	$42	$42	$42
3. Reduced costs of infant and child illness	$36	$35	$34
4. Productivity gain from reduced stunting	$152	$85	$25
5. Productivity gain from increased cognitive ability	$367	$205	$60
6. Reduced costs of chronic diseases	$49	$15	$1
7. Intergenerational effects	$92	$35	$257
Total Benefits	$832	$510	$257

Amounts are in US dollars.

Source: Constructed by author based on Alderman and Behrman (2006).

as instruments child-specific FTD exposure to nutritional supplementation, depending on village and birth date, which means that their estimates are not contaminated by family and community background and genetic endowments during the FTD also having post-FTD impacts (and, if successful, avoiding A3).[6] To limit estimation problems related to post-FTD familial and community inputs (and therefore limit A4 to unobservables), they controlled for birth-year dummy variables, maternal schooling, paternal schooling, parental wealth, whether parents had died before participants were 15 years old, school quality at age 7 years, distance to village center, and village of origin. Their preferred IV estimates indicate some important effects: a 1 unit increase in HAZ was significantly associated with 0.8 grades more schooling, approximately 0.25 SD higher test scores for reading and nonverbal cognitive skills, better marriage partners (1.4 years older, 1.0 grades more schooling, 1.0 cm taller), 21% greater household per capita expenditure as adults, and 10 percentage points lower probability of living in poverty as adults. For women, these results included 0.8 years older at first birth, 0.6 fewer pregnancies, and 0.4 fewer children but no significant impacts on adult health. They conclude that attrition is not a major problem because they control for numerous observables that might be associated with attrition, and their results are robust to explorations of impacts of attrition related to observables. They find that, for a number of outcomes, the IV point estimates differ fairly substantially from the OLS

estimates, thus suggesting that controlling for endogeneity and measurement error is important and that associational studies that do not may be misleading regarding the magnitudes of effects. Arguably, this is the most persuasive study yet available about the causal effects of FTD growth—or whatever FTD growth is proxying for—on life cycle outcomes in poorly nourished populations. They acknowledge (see note 2) that they do not confidently identify effects of FTD growth from other FTD outcomes unless they accept A2. This leaves open the question of to what extent these results (as the previous studies reviewed) justify interventions directly to increase FTD growth versus other outcomes correlated with FTD growth. The study, finally, is based on a very particular context: children born between 1962 and 1977 in four Guatemalan villages with high growth-faltering prevalence. If the results hold roughly the same for other poorly nourished populations, then they are very important because of high growth-faltering prevalence in South Asia and sub-Saharan Africa. However, the external validity of these results is difficult to assess because of the lack of other data from poorly nourished populations with initial randomized assignment and follow-ups for decades into mid-adulthood.

Hoddinott et al. (Hoddinott, Alderman, Behrman, Haddad, & Horton, 2013*a*) utilize the cost data for reducing growth faltering from 17 different LIMICs with high growth-faltering prevalences from Bhutta et al. (2008) and the estimated relation between stunting at 2 years and household consumption when the children became adults from Hoddinott et al. (2013*b*) to estimate the benefit–cost ratios for interventions to reduce stunting. Their estimates indicate that benefit-cost ratios are generally substantially greater than 1 (median of 18.4) with substantial cross-country variance due to variations in costs and projected wage growth (range 3.8–47.9). These costs are conditional on the same assumptions as the results in Hoddinott et al. (2013*b*) plus:

(A7) The relation between reducing stunting and adult consumption similar to that found in Hoddinott et al. (2013*a*) holds across the 17 countries considered, with adjustments for different projected earnings growth across countries; this is a strong assumption given different skill mixes across labor markets of the sort emphasized by Rosenzweig and Zhang (2013).

1.3.2 Estimates of FTD Growth Determinants on Subsequent Life Cycle Outcomes

Direct estimation of Equation (2) may be informative about the impacts of FTD growth determinants on later outcomes. Such estimates do not require A2 (i.e., there are no FTD growth outcomes correlated with FTD growth) because the question is to elucidate long-run impacts of FTD growth determinants whether or not FTD growth is the sole channel for such impacts. Household and community factors are the major proximate

Table 1.4 Benefit–cost estimates for nutritional interventions for preschool children with discount rates of 3–5%

	Benefit–Cost Ratio
1. Reducing LBW for pregnancies with high probabilities LBW	
1a. Treatments for women with asymptomatic bacterial infections	0.58–4.93
1b. Treatment for women with presumptive STD	1.26–10.71
1c. Drugs for pregnant women with poor obstetric history	4.14–35.20
2. Improving infant and child nutrition in populations with high prevalence of child malnutrition	
2a. Breastfeeding promotion in hospitals in which norm has been promotion of use of infant formula	5.6–67.1
2b. Intergrated child care programs	9.4–16.2
2c. Intensive preschool program with considerable nutrition for poor families	1.4–2.9
3. Reducing micro nutrient deficiencies	
3a. Iodine (per woman of child-bearing age)	15–520
3b. Vitamin A (per child under 6 years)	4.3–43
3c. Iron (pregnant women)	6.1–14

determinants of FTD growth. However, generally, household and community resources are highly correlated across life cycle stages and have impacts across such stages, which makes it challenging to identify their impacts during the FTD alone on subsequent life cycle outcomes; A4 is very strong for these determinants of FTD growth. But some studies, examples of which we now consider, consider marginal determinants that arguably give more credible estimates.

1.3.2.1 *Protein-Dense Nutritional Supplements and Multiple Outcomes into Adulthood*

The Guatemalan INCAP longitudinal data used by Hoddinott et al. (2013*b*) also has been used to investigate the impacts of the *atole* protein-dense supplement relative to the *fresco* no-protein supplement on outcomes over the life cycle. The randomized nature of these data means that estimates using them are likely not to be particularly contaminated by familial, community, and genetic endowment determinants later in the life cycle, so A3 and A4 need not be invoked (or perhaps invoked in weaker forms). Estimates suggest fairly substantial effects decades later when the sample members were 26–42 years old: approximately 40% increased real wage rates for men; approximately 0.25 standard deviation increases in reading skills and cognitive abilities for both men and women; and higher birthweights by approximately 100 g for the children of women who had received the supplements (Behrman et al., 2009; Hoddinott, Maluccio, Behrman, Flores, & Martorell, 2008; Maluccio et al., 2009). These results are consistent with FTD growth being important or an important proxy for other

developments, though, as noted, they do not demonstrate that FTD growth itself is the only or even the primary channel through which these effects occur because they refer to the effects of the supplements that may occur through channels other than FTD growth, and they do not compare effects through various channels. For the educational and labor market outcomes, these studies report no significant difference between being exposed to protein-dense nutritional supplements for up to 24 versus up to 36 months of age, but there is no significant impact from being exposed for 36–72 months. For intergenerational effects, by contrast, exposure throughout childhood up to 15 years of age, not just up to 24 or 36 months, produces significantly positive estimates.

1.3.2.2 *Other FTD Nutritional Interventions and Post-FTD Outcomes for Synthetic Cohorts that Link Estimates Across Life Cycle Segments*

As in Table 1.3 for birthweight, there are estimates for FTD nutritional interventions for synthetic cohorts that link estimates across life cycle segments for low-income populations. To provide perspective about the magnitudes of such impacts, it is useful to consider the implied benefit–cost ratios. Table 1.4 gives some illustrative values. Based on these estimates, benefit–cost ratios from selected FTD nutritional interventions in poorly nourished populations appear considerable.

1.4 Catch-Up Growth and Implications

1.4.1 Is There Catch-Up Growth?

1.4.1.1 *Average Patterns Across Ages*

Figure 1.1 gives a stylized characterization of average patterns of HAZ within undernourished populations. Prentice et al. (2013) note that, in terms of HAZ, this pattern implies some slight catch-up that continues into early adulthood based on the COHORTS data. Partially in response, several subsequent studies point out that although there may be some catch-up on average in terms of HAZ, the HAZ for adults in these populations still are considerably below those for well-nourished populations, and the deficit in height on average increases in these populations (Leroy, Ruel & Habicht, 2013; Leroy, Ruel, Habichat & Frongillo, 2014; Lundeen et al., 2014a, 2014b). Height deficits can increase with age even if HAZ deficits decline because height standard deviations that reference well-nourished populations increase with age. Thus, whether these linear growth patterns are consistent on average with some catch-up linear growth (as indicated by increasing HAZ) or increased deficits in linear growth (as indicated by increasing height deficits) depends on the appropriate measure of linear growth deficits. If our interest is not in relative adult height (HAZ) or absolute adult height (height deficits) per se, a

natural question is to determine which is more highly associated with the outcomes we are concerned with (i.e., in labor markets, marriage markets, and adult health). Several studies for different economies report significant associations between height and adult outcomes (e.g., Behrman & Deolalikar, 1989; Case & Paxson, 2008; Persico, Postlewaite, & Silverman, 2004).[7] But data on adults alone cannot identify whether HAZ or height deficits are more important because HAZ is just a linear transformation of height—thus, HAZ, height, and height deficits all are perfectly correlated. Some insight about the relative importance of HAZ versus height deficits might be gained by examining empirical associations or, better yet, the causal effects of pre-adult trajectories of HAZ and height on important adult outcomes.

1.4.1.2 *Regression Estimates*

There are several regression estimates pertaining to catch-up growth in the literature. These estimates can be viewed as linear approximations to a version of Equation (2) that has H_s, an indicator of height and one of the elements in the vector of outcomes Y_s, as the dependent variable, and with the history of Y_s for previous life cycle stages substituted into the right side to represent conditional demand functions. For most (but not all) of these studies, the height element in Y_{s-1} is assumed to be a sufficient statistic for all past history, thus involving assumptions A2 and A3 (although not necessarily explicitly).[8]

$$H_s = \alpha_0 + \alpha_1 H_{s-1} + \alpha_2 W_s + \alpha_3 K_s + \alpha_4 P_s + V_s . \tag{2A}$$

The critical parameter for catch-up growth in this formulation is α_1, under the assumption that the indicator for α_1 controls for age.[9] A value of $\alpha_1$that is not significantly different from 1.0 is interpreted to mean no catch-up growth, and a value of $\alpha_1$that is not significantly different from 0.0 is interpreted to be complete catch-up growth or path independence. Presumably, a value of α_1 that is significantly less than zero implies regression toward the mean.

One group of estimates of Equation (2A),[10] mostly found in the nutritional literature, uses OLS and generally assumes α_2 is zero (though in some cases controls are included for child age and gender and family background characteristics, particularly those related to parental schooling and family resources). These studies report estimates of α_1 that are not very different from 1.0, suggesting little to modest recovery from childhood stunting on average (Adair, 1999; Stein et al., 2010; Lundeen et al., 2014*b*). The estimates of α_1 are affected by upward omitted-variable bias if, as seems plausible, there is serial correlation over time in unobserved family and community factors (middle box on the left in Figure 1.1) that determine both H_s and H_{s-1}.[11] A second group of studies uses econometric methods such as instrumental variables or dynamic panel estimators to attempt to eliminate such omitted-variable bias, control for measurement error, and find smaller estimates of α_1. These suggest that between one-third and one-fourth of earlier linear growth deficiencies on average are reversed (Alderman, Hoddinott, & Kinsey, 2006; Behrman, Deolalikar, & Lavy, 1994; Georgiadis et al., 2015; Hoddinott & Kinsey,

2001; Mani, 2012; Outes & Porter, 2013). The estimates in all of these studies, whether they use instrumental variables to attempt to control for endogeneity and measurement error or not, probably are biased upward because of A3 (i.e., other outcomes can be ignored) since there probably are positive correlations among child outcomes so that the growth indicator in part proxies for them.

1.4.1.3 *Individual Patterns*

These regression estimates yield average patterns. Even if average growth faltering does not change much, as in Figure 1.1, there may be considerable movement for individual children, with some children experiencing growth recovery and others experiencing growth faltering. If so, and if determinants of these movements can be identified, then there may be significant possibilities for increasing growth recovery and, perhaps equally important, reducing growth faltering after the FTD. One way of exploring whether there is much individual movement around the average growth paths is to explore how much of the variation in HAZ at a later age is predicted by HAZ at an earlier age (basically estimating Equation (2A) without any other right-side variables except for HAZ at the earlier age). Based on longitudinal data on 7,266 children in the Young Lives (YL) study in Ethiopia, India, Peru, and Vietnam, HAZ at about age 1 year predicts much of HAZ variation at age 5 years, but 40–71% is not predicted. Similarly, HAZ at age 5 years does not predict 26–47% of HAZ variation at 8 years (Schott et al., 2013). These data do not yet permit following the same children when they become older, although subsequent rounds of data will become available in the future.

1.4.2 Implications

1.4.2.1 *Associations with Cognitive Achievement*

Crookston et al. (2013) use the YL data to determine whether changes in growth after infancy are associated with schooling and cognitive achievement at age 8 years. They represent growth by HAZ at 1 year and HAZ at 8 years that was not predicted by HAZ at 1 year (or residual HAZ at 8, conditioning on HAZ at 1). They find that HAZ at 1 is inversely associated with overage for grade and positively associated with mathematics achievement, reading comprehension, and receptive vocabulary. Unpredicted growth from 1 to 8 years of age is also inversely associated with overage for grade (odds ratio range across countries: 0.80–0.84) and positively associated with mathematics achievement (effect size range: 0.05–0.10), reading comprehension (0.02–0.10), and receptive vocabulary (0.04–0.08). Children who recovered in linear growth had better outcomes than did children who were persistently stunted but were not generally different from children who experienced no growth faltering. They conclude that improvements in child growth after early faltering might have significant benefits on schooling and cognitive achievement and that, although early interventions remain critical, interventions to improve the nutrition of preprimary and early primary school-aged children also merit consideration. Using the same YL data, Georgiadis et al. (2015) find that, within

the FTD, postnatal growth in the first year generally predicts cognitive skills at age 8 in all four countries, but fetal growth and post-infancy growth are at times (but less frequently) significant predictors. They also show that the use of actual HAZ at various ages versus initial HAZ and unpredicted HAZ for older ages are both consistent with the same underlying recursive model even though some of the coefficients of actual versus unpredicted growth differ in their relations to that model. These YL results suggest that there may be some important associations between post-FTD growth and various outcomes, but these results require a strong set of assumptions, including A1–A3, to permit inferences about causality for either FTD growth or post-FTD growth.

1.4.2.2 *Determinants of Growth Patterns*

Controlling for initial HAZ, Schott et al. (2013) characterize child growth up to age 1 year, and from ages 1 to 5 and 5 to 8 years and identify key household and community factors associated with these growth measures using the YL data. Multiple regression analysis suggests that parental schooling, consumption, and mothers' height are key correlates of HAZ at age 1 and also are associated with unpredicted change in HAZ from ages 1 to 5 and 5 to 8 years, given initial HAZ. These results underline the importance of children's starting point in infancy in determining subsequent growth, point to key household and community factors that may determine early growth in early life and subsequent growth recovery and growth failure, and indicate that these factors vary some by country, urban/rural designation, and child sex. That household and community factors are important determinants of both FTD growth and subsequent growth not predicted by HAZ in the FTD suggests possibilities for affecting both early growth and subsequent growth through these determinants.

1.5 Conclusion

Early-life growth failure is widespread in many LIMICs, particularly in South Asia and sub-Saharan Africa. Studies find strong associations between early-life (FTD) growth failure and outcomes over the life cycle and into the next generation. Strong assumptions are needed to interpret these as yielding causal estimates, with the strength of the assumptions differing across studies. MZ-FE estimates suggest that birthweight effects differ substantially from associations when all common endowments that MZ twins share are controlled. Several studies using the Guatemalan INCAP data report evidence of strong impacts of randomly allocated protein-dense nutritional supplements, particularly during the FTD, on adult and intergenerational outcomes, for which lessening FTD growth faltering may have been the channel: about a quarter of a standard deviation on adult cognitive skills and abilities, more than 40% on adult male wage rates, and about 100 g of women's children's birthweights. Another study using different birth cohort exposures to the INCAP alternative supplements finds associations between growth faltering at 2 years and a number of outcomes over the life cycle, arguably

without biases due to unobserved determinants but probable biases due to failure to control for other possible channels. Based on such studies, there are very viable claims that growth faltering in the FTD has high costs through reducing schooling attainment, adult height, and adult earnings, among other outcomes (e.g., Table 1.1), and that it is irreversible.

However, there are estimates that suggest that both post-FTD catch-up growth and growth faltering can be considerable, that household and community factors are likely determinants of both FTD growth faltering and post-infancy growth not predicted by growth measures in the FTD, and that such unpredicted growth is almost as significantly associated with cognitive achievements later in childhood as are growth indicators in the FTD. These results certainly raise questions about the claims of irreversibility and the "closing window of opportunity" at age 2. They suggest that, although it may be that the rates of return to investing in early life are relatively high (as suggested in Figure 1.2), given current investments in children, subsequent nutritional investments may have high rates of returns as well.

There are many limitations in the existing literature, mostly related to the assumptions noted explicitly above, and these limitations suggest the need for further research.

1. Most available studies to date rely on strong assumptions to make inferences of causality, particularly in light of probable endogeneity of growth failure. Even studies that plausibly control for unobserved endowments, for example, generally do not control for other channels through which effects may be transmitted. These weaknesses are pervasive in investigations both of the importance of growth faltering in the FTD and of the importance of subsequent nutritional outcomes.
2. There are few datasets with which to assess long-run effects, particularly data with characteristics (e.g., initial randomization, natural experiments including twins) that permit at least some exploration of the robustness of the estimates to weakening some of the assumptions. Much of what appears to be known with relatively great confidence, for example, depends on studies of one dataset in one particular context: the Guatemalan INCAP data. Also there is a basic quandary regarding how to assess longer run effects over the life cycle because longitudinal data, although having some considerable advantages, also means that the early-life experiences being examined occurred in much different contexts that prevailed decades ago.
3. Another limitation of the current literature is that most studies consider only reduced-form linear relations that provide little insight into static or dynamic substitution/complementarities that might arise from production technologies or from parental behaviors, but that would require much more structural approaches to investigate (and that also would allow exploration of counterfactual policies).
4. There is some information on the costs of altering growth faltering either in the FTD or post-FTD, but many studies do not address the cost side even though it is difficult to see how policy inferences can be made without knowledge of the costs as well as the benefits.

5. Empirical investigations in this area tend to focus on undernourished children who tend to be from poor families, and thus they potentially are informative about policies related to pro-poor redistribution. But they make virtually no effort to inform about policies to increase efficiency due to market imperfections, although there should be potential for "win-win" policies to address both anti-poverty and efficiency aims if the poor are particularly subject to market imperfections, as often claimed.
6. There is little attention paid in the literature to the probable diminishing marginal returns to investments in the FTD and post-FTD. This is an important area of research. Even if it is the case that, given current investments, children in poorly nourished LMIC populations may have rates of returns as shown in Figure 1.2, shifting those investments from older to younger children would seem likely to reduce the rates of return for early-life investments and increase those for older ages, thus twisting this curve.

In conclusion, it is quite possible that early-life growth faltering and subsequent catch-up or faltering are very important for the outcomes over their life cycles of hundreds of millions of children. However, there remains much to be learned about the nature of causal effects and interventions at different ages to determine the (possibly high) benefits relative to costs.

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Notes

1. Estimates for valuing averted mortality per child range widely from cheapest alternatives for averting mortality (e.g., ~$1,000 for vaccinations in Pakistan; Summers, 1992) to what compensating differentials that individuals require to assume more risk (e.g., based on wage tradeoffs, ~$10,000,000 for the United States; Viscusi & Aldy 2003).

2. Hoddinott et al. (2013*b*) explicitly recognize this possibility: "We did not consider HAZ or stunting at 2 y to be the causal factor per se. Rather the cause was the cascade of factors at societal, household, and individual levels, such as those depicted in the UNICEF conceptual framework (36), which ultimately determines nutrient availability at the cellular level and directly has an effect on growth and development in the first 1000 [days after conception]." This implies that to remedy problems for which FTD growth failure proxied in their study, the whole "cascade of factors at societal, household, and individual levels" would have to addressed.
3. Dizygotic (DZ, fraternal) twins-FE estimates also control for all factors shared by twins, including parent life cycle stages, but not genetics at conception. Estimates suggest that DZ genetic differences are important for socioeconomic outcomes (e.g., consistent with about a quarter of US adult twins earnings variance: Behrman, Rosenzweig & Taubman, 1994). Siblings-FE estimates control for all factors shared by siblings but not for parent life cycle stages or individual-specific deviations from common family genetics at conception.
4. Actual cohort studies follow a birth cohort over many years of their lives. Synthetic cohort studies link estimates for multiple birth cohorts for different life segments (e.g., one birth cohort for each of the life cycle stages in Figure 1.3).
5. Another possibility starts with an older sample and solicits recall information on early life; this is subject to recall errors but may be useful for salient early-life events for which recall is likely to be relatively accurate. I am not aware of good examples of this approach on this chapter's topics.
6. Being twins and ln mother's height are also in their preferred instrument sets; alternative estimates indicate that these variables are not significant if included in second-stage estimates.
7. Although generally these studies do not control for endogeneity of height, which may be proxying for unobserved genetic endowments. One study using the Guatemalan INCAP data that attempts to control for the endogeneity of height finds that a significant OLS association of height with wages does not remain significant if height is treated as endogenous, with randomized nutritional supplements among the identifying instruments (Behrman, Hoddinott, Maluccio, & Martorell, 2011).
8. Exceptions include lagged height from earlier life cycle stages and thus do not use A3, but do assume, as in A2, that all other outcomes can be ignored. See Lundeen et al. (2014*b*).
9. If not, the coefficient estimate for α_1 presumably will reflect normal age growth between life cycle stage *s-1* and life cycle stage *s*. In this case, comparisons in the rest of this sentence have to be adjusted for the normal age growth.
10. Or of a slight variant in which H_{s-1} is subtracted from both sides so that the coefficient of H_{s-1} in the relations estimated is $\alpha_1 - 1$.
11. Eckhardt et al. (Eckhardt, Suchindran, Gordon-Larsen, & Adair, 2005) report that diets after age 2 years are significantly associated with heights up to age 18.5 years, which seems to suggest catch-up growth.

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CHAPTER 2

BIOLOGICAL MEASURES OF WELL-BEING

RICHARD H. STECKEL

What is well-being, or, more to the point in the social sciences, how can it be measured? The subject is more subtle than it may appear, but people who study the problem agree that it has three important ingredients, shown in Figure 2.1. These components (material, health, and psychological) interact or depend on one another. One may suppose that, ultimately, well-being is a psychological phenomenon—it's all in the mind, a feeling, or sensation. One could even go beyond this, arguing that the processes of the mind are biochemical and, in principle, could be measured as such. Scientists are a long way from this achievement, however, and for now we must be satisfied with preferences or whatever people say, demonstrate, or choose.

These choices exhibit patterns. Except in pathological cases, people prefer good health to bad and more as opposed to fewer material goods or services. Satiation occurs near the tipping point at which too much of a product is dissatisfying. In colonial New England, for example, indentured servants insisted they not be fed lobster more than twice per week. Therefore, people allocate their expenditures across goods and services so as to balance tradeoffs (i.e., marginal utilities in relation to costs).

One can also distinguish individual from aggregate well-being. The individual level is simpler because it's defensible to assume that tastes or preferences do not change, at least in the short run. These preferences often differ across individuals, although there are similarities across subgroups defined by education, age, gender, and so forth. Under the convenient assumption of comparability, it is then possible to consider this year's market basket of consumption to that of last year, or this year's income to that of the previous year. Typically individuals do not worry about externalities of consumption, such as pollution or congestion, in judging their choices on aggregate well-being because they are a very small part of the market. This proposition does not hold for nations or large regions of countries.

What is an economist or psychologist to do in quantifying national well-being? Most would start measuring at the individual level and then aggregate, with the qualification noted earlier about interpersonal comparisons. Still, in considering income (a measure

FIGURE 2.1 Components of well-being.

of access to resources or material goods and services) versus health, weighting schemes differ. In gauging health, the process is highly democratic—each person counts the same: a death is a death regardless of the status of the person. However, with respect to income, those people having greater access to resources count more in the aggregate statistics.

Readers can readily understand the existence of a literature or philosophy on measuring well-being. This is a field in itself, but practical people—those charged with analyzing evidence, delivering reports, and developing policy—are aware of these issues but cannot dwell on them. They must make choices or recommendations, and this chapter reviews common ones.

Concepts are most useful if they are readily measurable, if there is an empirical counterpart: numbers such as prices and quantities left by markets, length of life as indicated on death certificates, or even feelings of happiness as recorded on responses to questionnaires.

It should be noted that technology and public purpose heavily determine the cost and availability of evidence related to national well-being. Bills of mortality, for example, were recorded for centuries, especially in cities, but systematic national efforts to record deaths soon after they occurred appeared only in the mid to late 19th century, after the germ theory of disease defined a role for these documents. With regard to technology, one might like to have a time series of blood pressure measurements, but practical blood pressure cuffs did not arrive until the early 20th century.

This chapter emphasizes biological measures of well-being, but it is important to place them in perspective relative to monetary measures commonly used by societies. Gross domestic product (GDP) per capita is essentially universal, and for this reason the concept receives special consideration.

Biological measures have certain advantages over monetary quantities such as income or wealth. For one, they are more comparable across time and space. Life expectancy at birth, for example, means about the same thing in ancient Egypt as in modern America.

The benefits of income, on the other hand, depend on the goods and services available for purchase and their prices. Over long periods of time, it is important to correct prices for inflation or deflation, and price levels may differ across regions or countries. One may speculate what GDP means in a hunter-gathering society, yet numerous biological measures of well-being would be well-defined.

In addition, there are diminishing returns to income but not to health, at least within the current range of possibilities. Income relative to that of others matters for people's well-being in the sense that the very rich may provoke envy. There is even evidence that living near people who are much richer is bad for health, perhaps by creating anxiety to "keep up with the Jones." Health inequalities, however, may also reduce health and well-being through exposure to diseases harbored by the poor or the unwashed.

2.1 Common Biological Measures

The number of biological measures available is nearly limitless, particularly when considering the cellular or molecular level. Imagine white blood cell counts, cortisol (a steroid hormone released in response to stress), cholesterol readings, and temperature, which all help diagnose health. Useful as these may be at the individual level, public health officials have not provided long times series of evidence for social scientists or policy makers to study.

Here, I examine the unique and valuable contributions of four biological measures—life expectancy at birth, morbidity, physical stature, and skeletal remains—to understanding levels and changes in human well-being. People desire far more than material goods and, in fact, they are quite willing to trade material things in return for better physical health. Health is so important to the quality of life for most people that the "biological standard of living" is a useful concept. Biological measures may be especially valuable for historical studies and for other research circumstances where monetary measures are thin or lacking. But note that they are informative also for modern populations because they provide another perspective on well-being. A concluding section ruminates on the future evolution of biological approaches in measuring happiness.

2.1.1 Life Tables

The life table or average length of life is the oldest and most widely used biological measure. The concept and the data required to construct this measure were understood by the early 1800s, but, in most countries, it took many decades to form administrative structures to collect the necessary evidence, which are death certificates and estimates of the size of the population at risk (Swanson, Siegel, & Shryock, 2004, ch. 12–14).

Although there is a cohort life table, which is based on the mortality experience of an entire birth cohort, most life tables are the period variety, which imagines a synthetic

or artificial cohort that experiences the probabilities of death of people of different ages and observed in a single year. The probabilities of death are calculated from information on the number of deaths by age, gathered from death certificates, and the number of people alive at each age, usually estimated from census counts of the population. One calculates life expectancy at birth by supposing that an actual birth cohort experiences the mortality rates of people of different ages observed in a single year, say 2000. Thus, a period life table provides a cross-section measure of health that will underestimate the actual life expectancy of people born in 2000 if mortality rates fall over time, as was the case in the 20th century. Likely, those people who were old in 2000, for example, had higher mortality rates than those who will be old in 2050. The actual birth cohort will live longer on average than the cross-sectional evidence would predict. Of course, this is not inevitable because mortality rates may fluctuate over time or rise sharply during an epidemic.

The 20th century witnessed a vast expansion in population studies that were well-grounded in evidence (Caldwell & Caldwell, 2006). By the middle of the 20th century, scholars had formulated an influential generalization called the "demographic transition," which depicted progress from pre-modern regimes of high fertility and high mortality (in the neighborhood of 3.0–3.5%) to the post-modern situation in which both were low (about 1.0–1.5%). Typically, the fertility decline preceded the fall in mortality, and, depending on the country and time period, the difference may have been several decades or longer. The process of change tended to be more rapid in the 20th as opposed to the 19th century, and those of the past half-century occurred even more quickly.

The health side of change is often called the "mortality transition," and recent large compilations of evidence on the topic can be found in Riley (2001) and in Maddison (2001). Both document and discuss possible explanations for change in the world of 1800 with 1 billion people and life expectancy of perhaps 25 years, to the present world of more than 6 billion people and a life expectancy of about 66 years. By 1900, life expectancy across the world had risen slightly, to more than 30 years, but important differences existed by region, with European countries and their colonial offshoots (plus Japan) having a 20-year advantage (46 vs. 26 years) over the rest of the world, which had changed little if at all. Today, there is even more variation across countries, where life expectancy differs by 2:1 (about 40 years to slightly more than 80 years). Even those nations with the lowest life expectancy today, however, are better off in this regard than the healthiest countries of two centuries ago.

There is little doubt that cost-effective public health measures played an important role by reducing exposure to pathogens via cleaner water, waste removal, sewage treatment, personal hygiene, and chemical control of disease vectors. More controversial are explanations for improving health in Europe and its offshoots prior to 1900, before the public health movement flourished and long before antibiotics and other advances in medical technology were available. One school of thought led by McKeown (1976) and by Fogel (2004) emphasizes improving diets that stemmed from the agricultural revolution of the 18th and 19th centuries, which featured new crops and equipment as

well as enclosures, transportation improvements, and eventually the rise of free trade. Others claim that rising incomes and/or a decline in the virulence of pathogens were important.

2.1.2 Morbidity

Of course, not all years of life are equal in terms of strength and vigor. For this reason, demographers have proposed ways to adjust length of life in terms of quality. Measuring the quality of health is challenging in part because there are numerous measures of morbidity and illness, and even if one standard is widely accepted, consistent collection of evidence over time and across space is usually difficult and expensive. The point generally holds with greater force for the past because few if any surveys are available, although Section 1.7 on skeletal remains demonstrates how bone lesions reflect chronic morbidity conditions.

A few decades ago, health economists devised the concept of quality-adjusted life years (QALY) to help estimate cost-benefit ratios from various health interventions (Klarman, Francis, & Rosenthal, 1968; Torrance, Thomas, & Sackett, 1972). The method places a weight from 0 to 1 on the time spent in different health states. A year in perfect health is worth 1 and death is assigned a 0. There are intermediate values for states of life like living with a pacemaker implant, undergoing kidney dialysis, and many other conditions. Some painful or agonizing states are considered worse than death and receive negative values. After considering the additional years of life created by various interventions and weighting these additional years for the quality of health, the result is a common measure that is useful for assessing the benefits of health care spending or other interventions. The method has a number of practical and technical difficulties related to measuring the quality of life (assigning numerical values to morbidity), but physical examinations and surveys are ways to gain such information. One popular survey (EQ-D5) asks the extent to which individuals have functional problems in five areas: mobility, pain/discomfort, self-care, anxiety/depression, and pursuit of usual activities (http://www.euroqol.org/).

If such data were available over the entire life span of an individual, one could construct a graph by age that depicts health, measured on a scale from 0 to 1, over the life course. In this situation, the area under the curve is a biological measure of the quality of life measured by length of life adjusted for health while living. There is obviously a tradeoff between duration and health quality that provide the same QALY, or many different curves can have the same area.

In the United States, morbidity surveys began with Hagerstown, Maryland, in 1921–24 but an ongoing program did not begin until 1956 (Perrott, 1949). The National Center for Health Statistics interviews the noninstitutionalized population for information on doctor visits, hospital stays, acute conditions, limits on physical activity, and so forth while other surveys gain data through physical examinations and various psychological

and physiological tests. Numerous industrial countries such as Japan, the United Kingdom, and the Netherlands have similar surveillance systems.

Historical Statistics of the United States compiles dozens of morbidity statistics, including the incidence rates of many diseases (Steckel, 2006). For example, immunizations led to abrupt declines in many infectious diseases in the middle of the 20th century. Rates of measles had ranged from 250 to 750 per 100,000 population from 1912 up to about 1960, but, by 1966, the rate sank to about 20 per 100,000 or less. As another example, there is evidence showing little time trend in the average number of restricted activity days per person from 1967 to 1995, based on data from the National Health Interview Survey. Of course, interview data on restricted activity may be subject to cultural norms of what constitutes sickness or disability.

2.1.3 Stature and Nutritional Status

J. M. Tanner's authoritative book on *A History of the Study of Human Growth* recounts the long history of studying body size and proportions (Tanner, 1981). Artists were among the first to study human form quantitatively for purposes of accurately rendering sculptures and paintings. What might be called scientific interest in heights began during the Enlightenment. Early studies of *auxology*—that is, the study of human growth—were sporadic, imprecise attempts made by individuals. However, whereas systematic data on both national income and life expectancy awaited large-scale government action, useful measurements of height and related attributes could be made on a small scale. Thus, auxology made important progress before the end of the 19th century. The results of an explosion of growth studies in the 20th century are contained in *Worldwide Variation in Human Growth* (Eveleth & Tanner, 1976/1990).

Figure 2.2 displays the growth velocity of well-nourished boys taken from the National Health and Nutrition Examination Survey (NHANES) survey (Hamill, Drizd, Johnson, Reed, & Roche, 1977). Even though infants grow rapidly, the rate declines during childhood and reaches a preadolescent minimum around age 11. Nutritional requirements increase substantially during the subsequent adolescent growth spurt. Although the adolescent spurt is somewhat larger for boys, they end up 4.5–5 inches taller primarily because the boys have 2 additional years of growth at preadolescent rates. Several studies confirm the similarity of this pattern across a wide range of well-nourished ethnic groups; children who grow up under good conditions are approximately the same height regardless of ethnic heritage (Steckel, 1995, provides additional discussion and references).

Numerous studies establish the importance of diet, exposure to disease, and physical activity or work for the growth of children (Eveleth & Tanner, 1976/1990). In this context, it is useful to think of the body as a biological machine that operates on food as fuel, which it expends in moderate amounts at idle (resting in bed and replacing

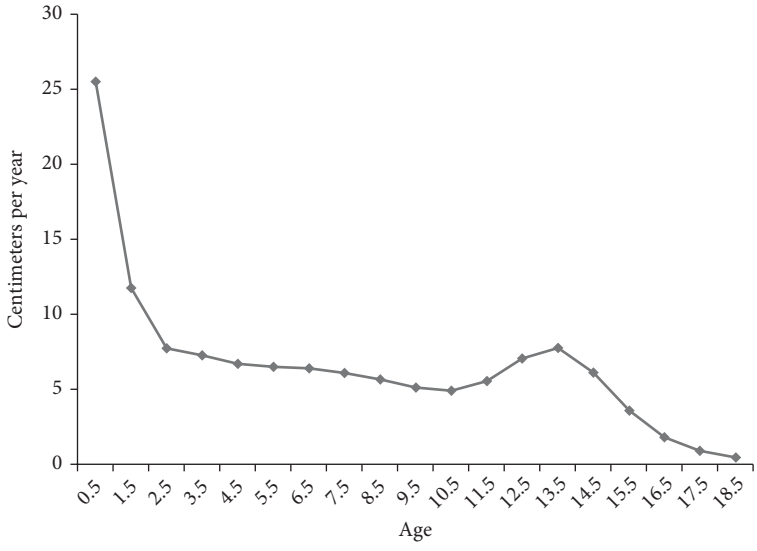

FIGURE 2.2 Growth velocity of boys under good conditions.

Source: Hamill et al., 1977.

worn-out cells) but in larger quantities while working or fighting infection. During World War II, for example, children's heights floundered in Russia and the Netherlands under restricted food intake. Disease may also stunt growth because it can divert nutritional intake to fight infection or result in incomplete absorption of what is consumed. Similarly, physical activity or work places a claim on the diet. For these reasons, average adult height reflects a population's history of *net* nutrition.

If better times follow a period of deprivation, growth may exceed that ordinarily found under good conditions. Catch-up (or compensatory) growth is an adaptive biological mechanism that complicates the study of child health using adult height because it can partially or substantially erase the effects of deprivation. Between birth and maturity, a person could potentially undergo several episodes of deprivation and recovery, thereby obscuring important fluctuations in the quality of life.

Preferably, researchers would have the complete growth history available for study, such as the curve depicted in Figure 2.2. Even these data would be inadequate for a thorough understanding adult height, however, because diet, disease, and physical activity may trade off in combinations that affect growth at each age. Although very useful for analysis, velocity at each age provides only proximate knowledge of why average adult height takes on the value it does (or did). Thus, a thorough understanding requires dozens of pieces of information, and even more if components of diet and varieties of disease are viewed separately. Essentially, such information is never available. In sum, average height is a good measure of welfare or the quality of life during childhood and adolescence but—like any other economic indicator—it can be difficult to analyze or explain because it reflects or captures many conditions over the period of growth.

Income is a potent determinant of stature that operates through diet, disease, and work intensity, but analysis of the relationship must recognize other factors. Personal hygiene, public health measures, and the disease environment affect illness, and work intensity is a function of technology, culture, and methods of labor organization. In addition, the relative price of food, cultural values such as the pattern of food distribution within the family, methods of preparation, and tastes and preferences for foods may also be relevant for net nutrition. Yet, influential policy-makers view higher incomes for the poor as the most effective means of alleviating protein-energy malnutrition in developing countries. Extremely poor families may spend two-thirds or more of their income on food, but even a large share of their very low incomes purchases few calories. Malnutrition associated with extreme poverty has a major impact on height, but expenditures beyond those needed to satisfy caloric requirements purchase largely variety, palatability, and convenience.

Impoverished families can afford little medical care, and additional income improves health through control of infectious diseases. Although tropical climates have a bad reputation for diseases, King (1966) argues that poor health in developing countries is largely a consequence of poverty rather than climate. A group of diseases are spread by vectors that need a warm climate, but poverty is responsible for the lack of doctors, nurses, drugs, and equipment to combat these and other diseases. Poverty, via malnutrition, increases the susceptibility to disease.

At the individual level, extreme poverty results in malnutrition, retarded growth, and stunting. Higher incomes enable individuals to purchase a better diet and height increases correspondingly, but once income is sufficient to satisfy caloric requirements, only modest increases are attainable through change in the diet. Height may continue to rise with income because individuals purchase more or better housing and medical care. As income increases, consumption patterns change to realize a larger share of genetic potential, but environmental variables are powerless after attaining the capacity for growth.[1] The limits to this process are clear from the fact that people who grew up in very wealthy families are not physical giants.

If the relationship between height and income is nonlinear at the individual level, then the relationship at the aggregate level depends on the distribution of income. Average height may differ for a given per capita income depending on the fraction of people with insufficient income to purchase an adequate diet or to afford medical care. Because the gain in height at the individual level increases at a decreasing rate as a function of income, one would expect average height at the aggregate level to rise, for a given per capita income, with the degree of equality of the income distribution (assuming there are people who have not reached their genetic potential).[2] Therefore one should be cautious in estimating and interpreting the relationship between per capita income and average height at the aggregate level without taking into account other factors, such as the distribution of income, entitlements, relative prices, and the organization of medical care.

The aggregate relationship between height and income can be explored by matching the results of 18 national height studies tabulated in Eveleth and Tanner (1976/1990)

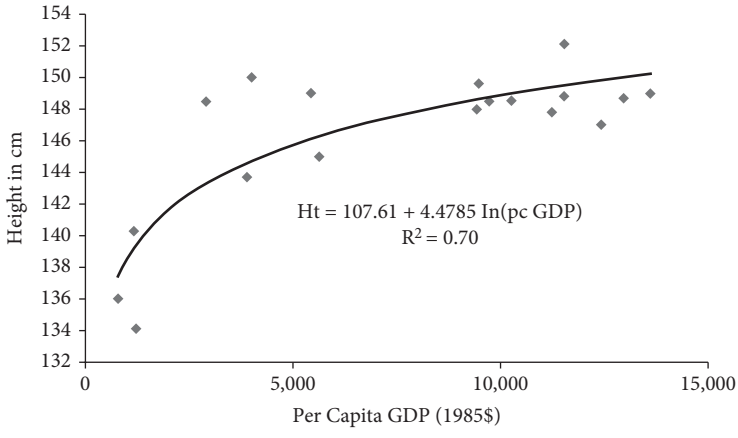

FIGURE 2.3 Per capita gross domestic product and height at age 12 (boys).

Source: Compiled from data in Eveleth & Tanner (1976/1990) and the Penn World Tables: http://pwt.econ.upenn.edu/php_site/pwt_index.php

with per capita income data compiled by Summers and Heston (1991). Despite the large number of factors that may influence the relationship, Figure 2.3 shows that there is a high correlation between a country's average height and the log of its per capita income, which is about 0.82. Although Figure 2.3 illustrates the case of boys, a similar relationship holds for girls and for adults. Figure 2.3 makes clear that income has diminishing returns on average height: once basic necessities are satisfied, higher income has less impact on health and physical growth. Thus, stature is a good measure of deprivation but not of opulence. One should be wary of estimating GDP from height because the curve displayed in Figure 2.3 is a function of health technology and the disease environment. Over time, the curve has shifted upward, receiving, for example, a large boost with the rise of the germ theory of disease, which led to several cost-effective innovations such as water purification.

Regional and national data series exist for heights, but historians have constructed them using data originally collected for other purposes. In the past 30 years, scholars have completed several large historical studies or compilations of evidence on height with an interest in understanding the standard of living. Although there are many data sources, such as slave manifests, muster rolls, convict records, passport applications, and so forth, the most abundant source is military organizations beginning in the middle of the 18th century, which routinely recorded heights for identification purposes, to assess fighting strength, and to make uniforms. Among the country studies are those on Austria-Hungry, England, and Japan (Floud, Wachter, & Gregory, 1990; Komlos, 1989; Mosk, 1996). Steckel and Floud (1997) organized a large effort for a comparative study of England, France, the Netherlands, Sweden, Germany, the United States, Australia, and Japan. John Komlos edited papers compiling evidence for numerous countries around the globe, and Steckel surveyed the state of the field as of the mid-1990s (Komlos, 1994, 1995; Steckel, 1995). Thus, historical perspective is available for numerous countries.

Moreover, the World Bank, the United Nations, and other agencies now regularly collect height data as part of occasional surveillance programs, to evaluate interventions, and to investigate socioeconomic mechanisms that affect physical growth and child health.

Collectively, the existing studies about stature both confirm and contradict certain long-held beliefs about differences and changes in human well-being. Heights substantiate the poor heath in cities relative to rural areas prior to 1900, a pattern long known from historical population studies. In 19th century Sweden, for example, average height was 3–8 cm greater in rural areas compared with Stockholm, depending on the time period and rural area (Sandberg & Steckel, 1988).

Comparing height patterns with traditional monetary measures of social performance across developing and developed countries in the second half of the 20th century revealed a useful role for heights: assessing biological inequality (Steckel, 1983). Steckel found that average height was not only a logarithmic function of average income at the national level, but that holding income constant, average height increased as the degree of income inequality declined. From this insight, researchers began to study occupational and regional differences in stature as a proxy for inequality. In late 18th century England, for example, the average heights at age 14 of poor boys admitted to the Marine Society were 20 cm below those of upper class boys who attended the elite academy at Sandhurst (Floud et al., 1990). During the same era, the difference in average height between the rich and the poor in the United States was roughly 3 cm (Margo & Steckel, 1983).

2.1.4 American Slaves

Anthropometric history has uncovered surprising patterns of evidence that challenged traditional interpretations of the past and sometimes provided new insights for human biology. An example includes the extraordinary growth depression in childhood and substantial later recovery by American slave teenagers, which is based on the heights of approximately 48,000 individuals exported from the cotton states. The children were among the smallest ever measured and would have caused alarm in a modern pediatrician's office. Yet the adults were comparable in height to the contemporary nobility of Europe, about half an inch shorter than Union Army troops, and less than 2 inches below modern height standards (average for males and females). Children adopted from poor into rich countries also show substantial catch-up growth, which proves the pattern is biologically possible. Selectivity cannot explain the pattern because the heights of slaves shipped by traders were little different from those transported by plantation owners, and higher death rates for shorter individuals would explain at most a trivial portion of the growth acceleration by teenagers (Steckel & Ziebarth, 2015).

The extent of deprivation and catch-up was extraordinary and unprecedented in historical or modern populations, which suggests that slavery was somehow responsible. All height studies, whether for the past or the present, show that the height

percentiles attained by children and by adults were similar within the same population or community.

The health deficit of young slaves probably began with low birthweights, which were associated with seasonal rhythms in the diet, work, and disease of pregnant slaves, which was followed by attenuated breastfeeding and a low-protein diet until slaves began working around age 10 (Steckel 1986*a*, 1986*b*, 1987). Nonworking slaves were fed little meat, a result commonly achieved through dietary segregation of food prepared in central kitchens whereby children and adults usually ate at separate times and places, with children in the nursery and working adults in the fields. If rations were allocated to families, then owners placed strict limits on the amount of meat given to slaves who did not work in the fields. Owners discovered that the workers could not perform hard labor without meat in their diet, which implies that parents paid a heavy price for sluggish field work (possibly a whipping) if they shared the meat rations with their children. Such feeding practices no doubt stressed the family as a unit in its ability to protect and nurture children.

Remarkably, this pattern of deprivation and catch-up was profitable for slave owners. Dietary studies show that protein is essential for growth. Meat rations limited to workers and protein deficits estimated for poor children in developing countries suggest that the protein deficit was 50% or more. Assuming a protein deficit was the only obstacle to achieving modern height standards, one can calculate the rate of return on feeding children enough meat protein to reach these standards based on the protein content and price of pork, as well as knowledge that slave values increased by 1.37% per inch of height. The rate of return is actually negative if the deficits were as high as 50%, and they remain under 1% if one allows mortality rates to fall in half from better nutrition. Rates of return would have been even lower if well-nourished children were highly active and required more supervision, or if there was a "leaky nutritional bucket"; that is, if these children had parasites, malaria, and other diseases that would have diverted or absorbed some of the better nutrition. It is well-established that poor nutrition in early childhood permanently reduces cognitive ability, which would have limited the capacity of former slaves to compete in the economy following emancipation. It may seem paradoxical, but planters who owned all future labor found that poor nutrition was profitable in their rearing of young children.

2.1.5 Long-Term Trends

Economic historians were surprised to find that heights in America declined during the middle of the 19th century (Figure 2.4), which occurred during the midst of an industrial revolution and rapid economic growth. While per capita incomes grew by 55% from 1830 to 1860, average height declined by 1.2 inches. Hence the term "antebellum puzzle," in which measures of human welfare were moving in opposite directions (Craig, forthcoming). One case see from Figure 2.5 that height declines occurred throughout the country (Zehetmayer, 2011). In additioin, both the United States and England experienced substantial and sustained height declines during industrialization prior to the late

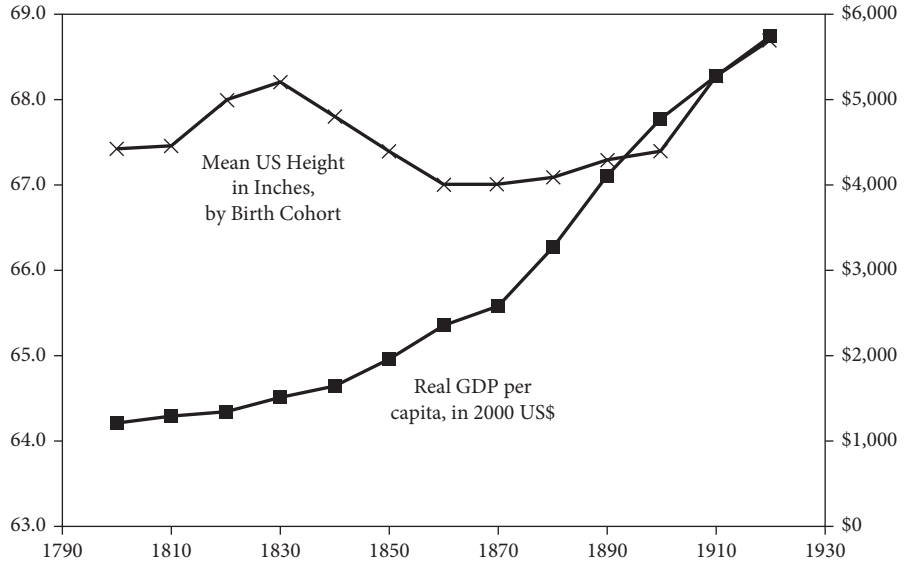

FIGURE 2.4 The trend in height versus real gross domestic product per capita.

Source: Carig, forthcoming

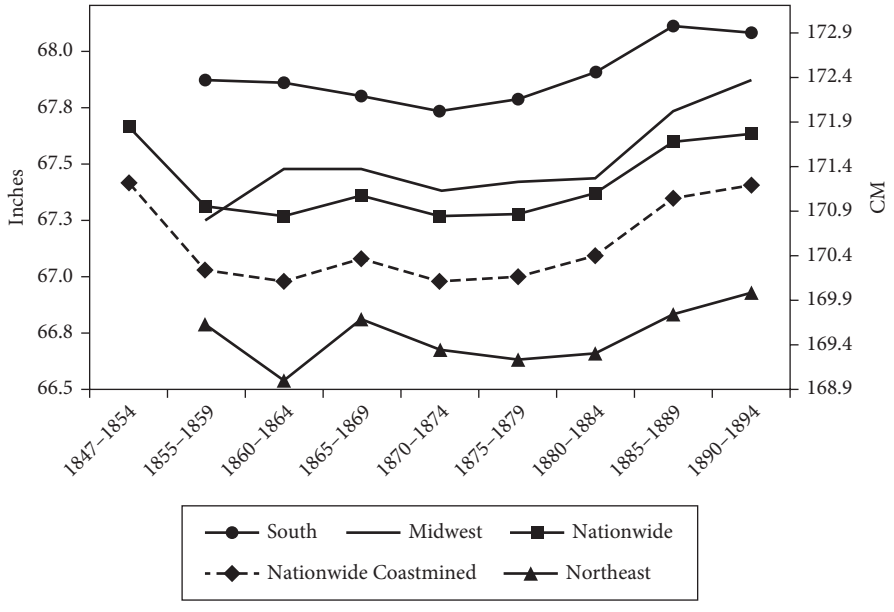

FIGURE 2.5 National and regional estimates of heights for recruits born in the United States.

Source: Zehetmayer, 2011, p. 320.

19th century (Steckel & Floud, 1997), but some European countries experienced shorter term problems during the mid-19th century as well.[3] Numerous explanations for the American case are now under investigation, including urbanization, the rise of public schools that spread diseases among children, higher food prices, growing inequality,

and higher rates of interregional trade and migration that spread pathogens (Komlos, 1998; Steckel, 1995).

Notably, the average height of Americans has leveled off in recent decades, while those of Europeans continue to grow. The Dutch are now the tallest, with the men averaging around 183 cm while Americans fall some 5 cm below. Average heights in northern Europe now exceed those in the United States, but explanations have been difficult to quantify and evaluate. Some people point to differences in the health care system, which is heavily subsidized and widely provided or universal in northern Europe as opposed to the United States. Inequality could play a role also, with democratic socialism leveling disposable incomes and raising average heights relative to the United States. Perhaps diets are the culprit, whereby Americans eat more fast food and snacks that crowd out fruits and vegetables that provide essential micronutrients.

2.1.6 Native Americans

With the possible exception of slaves, no group in American history has suffered greater misunderstanding closer to vilification and manipulation than Native Americans. In many Euro-American eyes of the mid-19th century, they were bad Indians who terrorized settlers and stole horses. Near the turn of the century, they became entertainers and caricatures, as illustrated by Buffalo Bill's Wild-West shows. The *Saturday Evening Post* then serialized romantic stories of the Old West, which were followed by Western movies in which Indians were usually the bad guys. By the 1960s, Native Americans were often portrayed as victims, and, by the 1990s, as ecologically sensitive caretakers of the land. It is difficult to sort fact from fiction in this diverse landscape of images.

Fortunately, height data provide some facts about nutritional status and health-related quality of life. Euro-Americans were not the tallest population in the world, at least in the middle of the 19th century. By a small margin, this honor went to native Americans who used horses to hunt and migrate across the Great Plains (Steckel & Prince, 2001). According to data originally collected by Franz Boas, the men in eight of these tribes averaged 172.6 cm (N = 1,123); the Cheyenne topped the list at 176.7 cm (n = 29), and the Arapaho were second at 174.3 cm (N = 57). The average heights follow an inverted U shape with respect to latitude. The shortest tribes occupied the northern (Assiniboin) and the southern plains (Comanche), whereas the Arapahoe and the Cheyenne of Colorado and Wyoming were the tallest. Their achievement is all the more remarkable because the tribes suffered repeated bouts of smallpox and other epidemic diseases that substantially reduced their numbers. It is unlikely that the Plains tribes were tall due to selective editing or removal of short people by disease. They were tall prior to the epidemics of the 1830s, and the selective effect of mortality on average height is quite small.

Several ecological and socioeconomic variables explain much of the height differences. The tribes were taller if they lived in environments with more green vegetation—a

source of food for people and animals; did not live close to the major trails leading to the west, which were centers for the spread of diseases and conflict (specifically the Santa Fe and Oregon trails); and had smaller land areas per capita, an effect possibly driven by the costs of policing or defending territory. Boas was able to estimate the birth year of each person, which could be linked with conditions during the growing years. Higher rainfall during the growing years (estimated from tree rings) promoted plant growth and the supply of food that increased adult height. On the other hand, epidemics, as assessed from historical accounts, had no effect on height, and, surprisingly, the initial transition to reservations was beneficial for growth (although reservation living was unhealthy near the turn of the century).

2.1.7 Skeletal Remains

Evidence from skeletons vastly extends the reach of anthropometric history by depicting aspects of well-being over the millennia, from hunter-gatherers to settled agriculture, the rise of cities, global exploration and colonization, and industrialization. Skeletons are widely available for study in many parts of the globe. A group of skeletons can provide age- and source-specific detail on nutrition and biological stress from early childhood through old age; indeed, several indicators of health during childhood are typically measurable from the skeletons of adults. Skeletal remains also exist for women and for children, two groups often excluded from more familiar historical sources such as tax documents, muster rolls, and wage records. The value of skeletons is substantially enhanced when combined with contextual information from archaeology, historical documents, climate history, and geography.

Bones are living tissues that receive blood and adapt to mechanical and physiological stress. If a bone is injured by trauma, infection, or erosion of cartilage such that joint surfaces deteriorate, a scar forms and leaves a mark that is usually permanent or at least identifiable if the person dies many years later (Larsen, 1997). More generally, the skeleton is an incomplete but very useful repository of an individual's history of health and biological stress that often takes the form of chronic morbidity. Physical anthropologists have learned that bones can be used to estimate stature and that various lesions (such as tooth enamel deformities) reflect poor health in early childhood. Other lesions on the skull reveal iron deficiencies in early childhood, and serious skeletal infections leave permanent marks on a bone's surface. The front of the tibia is particularly vulnerable in this regard because it has little soft tissue for protection and even small injuries are compounded by dietary deficiencies, such as a lack of vitamin C. Trauma is readily identified by bone misalignment, skull indentations, or weapon wounds, and degenerative joint disease, caries, and abscesses are signs of aging.

Scholars have completed few large-scale comparative studies of community health using skeletal data. The field is relatively new, and building up a database by analyzing skeletons one at a time is highly time-consuming. In addition, the variables collected by physical anthropologists and the details of measurement tend to vary across sites and

schools of thought, so meta-analysis based on evidence from past published studies is generally not an option.

The Backbone of History: Health and Nutrition in the Western Hemisphere is the largest comparative skeletal study undertaken to date, which sought to study not only the Neolithic revolution but health across a broad swath of time, space, and ethnic groups (Steckel & Rose, 2002). Collaborators pooled their evidence on seven skeletal features from 12,520 remains found at 65 localities that were collectively inhabited from 4000 BC to the early 1900s. They distilled the skeletal evidence into a health index, discussed in more detail later, that theoretically could range from 0 (most severe expression in all categories) to 100 (complete absence of lesions or signs of deficiency for every individual at the locality), but in practice averaged 72.8 (standard deviation [*SD*] = 8.0) and varied from 53.5 to 91.8 (Steckel, Sciulli, & Rose, 2002). Surprisingly, Native Americans were among both the healthiest and the least healthy populations, with European Americans and free blacks falling near the middle of the distribution.

The health index was estimated from the 12,520 skeletons of individuals who lived at 65 localities in the Western Hemisphere over the past several thousand years (Steckel & Rose, 2002). Age-specific rates of morbidity pertaining to the health indicators during childhood (stature, linear enamel defects, and anemia) were calculated by assuming that conditions persisted from birth to death, an assumption justified by knowledge that childhood deprivation is correlated with adverse health as an adult (Barker, 1994). The duration of morbidity prior to death is unknown for the infections, trauma, degenerative joint disease, and dental decay (and will be the subject of future research), but was approximated by an assumption of 10 years. Results are grouped into age categories of 0–4, 5–9, 10–14, 15–24, 25–34, 35–44, and 45+. Next, the age-specific rates for each skeletal measure were weighted by the relative number of person-years lived in a reference population that is believed to roughly agree with pre-Columbian mortality conditions in the Western Hemisphere (Model West, level 4), and the results were multiplied by life expectancy in the reference population (26.4 years) and expressed as a percent of the maximum attainable health. The seven components of the index were then weighted equally to obtain the overall index. Of course, numerous assumptions underlying the index can be challenged, modified, and refined. In particular, conditions like dental decay and trauma probably have different effects depending on the social safety net, common production technology, medical technology, and other factors that vary in unknown ways across societies. In addition, the index is an additive measure that ignores interactions.

The most intriguing finding from this project was a long-term decline in the health index in pre-Columbian America. On average, the health index fell by 0.0025 points per year from roughly 7,500 years ago to about 450 years ago, which amounts to 17.5 points over seven millennia. A decline of this magnitude represents a significant deterioration in health; it is larger than the difference between the most and least healthy groups who lived in the Western Hemisphere.

Unfortunately, the observations are concentrated in the two millennia before the arrival of Columbus, when there was clearly a great deal of diversity in health across sites. The highest value for the index did occur at the oldest site, but two sites in the later era also scored above 80. The least healthy sites (scores under 65) were all concentrated within 2,000 years of the present.

Steckel and Rose (2002) estimated a sequence of regressions that examined the statistical connection between health and various ecological categories like climate, size of settlement, diet terrain, and vegetation. Climate—as measured in categories of tropical, subtropical, and temperate—bore no relevance to the health index. This result was unanticipated and bears further study with more refined measures. Living in a larger community was deleterious to health. Groups living in paramount towns or urban settings had a health index nearly 15 points (2 *SD*) below that expressed for mobile hunter-gatherers and others not living in large, permanent communities. Of course, large pre-modern communities faced unsanitary conditions conducive to the spread of infectious disease and other maladies. Diet was also closely related to the change in the health index, with performance being nearly 12 points lower for those subsisting mainly on the triad of corn, beans, and squash, compared with the more diverse diet of hunter-gatherer groups. Because the transition to settled agriculture usually occurred with the rise of large communities, it is difficult to separate their effects on health.

Higher elevations reduced health: people who lived above 300 m scored about 15 points lower in the index. The exact mechanism for this relationship is unknown, but it is likely that a richer array of foods was available (with less work effort) at lower elevations. Vegetation surrounding the site may have affected health via the type and availability of resources for food and shelter. Forests, for example, provide materials for the diet, fuel, and housing and also sheltered animals that could have been used for food. Semi-deserts posed challenges for the food supply relative to more lush forests or grasslands, but the dry climate might have inhibited the transmission of some diseases. Those living in forests and semi-deserts had a health index about 9 points higher than inhabitants of open forests and grasslands. Flood plain or coastal living provided easy access to aquatic sources of food and enabled trade compared with more remote, interior areas, but trade may have promoted the spread of disease. Uneven terrain found in hilly or mountainous areas may have provided advantages for defense but could have led to more accidents and fractures. Apparently, the net benefit to health favored coastal areas, where the health index was about 8 points higher compared with noncoastal regions.

2.2 Frontiers

For more than three centuries scholars have struggled to measure and analyze personal and national well-being. The subject is complicated, and, despite great leaps forward,

much remains to be understood. Although some overlap exists, the customary measures of human well-being used by social scientists may be classified into three broad categories: material, psychological, and health.

Over the past century, researchers have made considerable progress in defining and implementing monetary measures such as GDP. Although research continues to expand on monetary measures, the pace has slowed relative to the high point of the mid-20th century and has reached diminishing returns in adding new useful information. There has been a recent resurgence of interest in measuring well-being through survey techniques that ask about "happiness" (for a starting point in this literature, see the papers by Kahneman and Krueger [2006], and Tella and MacCulloch [2006]). But nagging questions remain about whether people's evaluations of what they report as their "happiness" mean the same thing in one country or era as another. Of course, the same thing can be said about monetary measures: a dollar for me is very different from a dollar to my grandfather when he was my age. At least so far, psychologists have come forward with new approaches to the measurement of well-being that have captured the attention of social scientists.

This chapter focuses on biological measures of well-being, where great progress has been made over the past two centuries in measuring life expectancy, morbidity, and nutritional status. In my view, the next great research frontier will use nano-size biosensors to measure brain activity and assay biochemicals in a search for patterns and determinants of well-being and happiness. For example, miniature total analysis systems, commonly called "lab-on-a-chip devices," contain all the necessary elements for analyzing miniscule amounts of bodily fluids, including the intake, transport, mixing, separation, and measuring of results (Focus, 2006; Whitesides, 2006). Nanotechnology presents legitimate risks and concerns, and the public must be educated to judge the benefits and costs, and, if necessary, be prepared to intelligently regulate the development of these remarkable devices. Nanosensory systems, however, do offer the possibility of vastly improved measures of morbidity. Various concentrations of proteins or other chemicals in the blood may signals high stress levels, increased risk of heart attack, various cancers, epileptic seizure, or inflammation in specific organs. One could ultimately imagine monthly or even daily reports on a country's state of health much like we receive on per capita income or jobs, but based on information gathered by and uploaded from nano-scale devices imbedded in the bodies of a national sample of individuals.

The historical pioneers in the measurement of human well-being have been economists on the monetary measures, human biologists and economists on stature and nutritional status, psychologists on the happiness surveys and brain chemistry, and demographers on issues of life expectancy. Anthropologists, economists, human biologists, medical specialists, historians, and others have also begun to examine these issues in studies of skeletons. The disciplinary boundaries are blurring as researchers increasingly seek and recognize the interrelationships among these traditionally distinct ways of thinking about human well-being.

Notes

1. Of course, it is possible that higher incomes could purchase products such as alcohol, tobacco, or drugs that impair health.
2. This argument is reasonable over the range of data used in the empirical analysis discussed herein. However, within an extremely poor country, it might be possible for average height to increase with an increase in inequality if the rich did not approximately attain their genetic potential.
3. Heights in several European countries declined during the late 1830s and the 1840s in connection with harvest failures and/or rising food prices. By the late 19th century, the public health movement noticeably diminished the consequences for health of events associated with industrialization.

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CHAPTER 3

CRISIS AND HUMAN BIOLOGY

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3.1 Introduction

In modern parlance, the word "crisis" describes a pronounced shock, an adverse event, often economic or financial in nature. But, the word's earliest appearances in English texts refer to human illness, with a "crisis" being the critical juncture in the progression of a disease that determines whether the sufferer recovers or dies.[1] The links between crisis and human biology extend well beyond this point, however, and they are the subject of a burgeoning literature in economics. How do the various manifestations of crisis—from deep recessions to pandemics to natural disasters—affect human health, reproduction, and child development, and how do changes in health endowments, incomes, prices, and behaviors mediate these effects?

This chapter reviews the empirical evidence on the effects of crisis on human biology, assessing the capacity of economic theory and methodology to illuminate this evidence. We define crises as acute (as opposed to chronic), severe, and unexpected negative events, thus helping us focus our discussion by circumscribing a subset of the vast literature linking a variety of acute shocks with human biology.[2] We focus on crises that affect entire populations at once, which are likely to have the gravest consequences. Crises strike at various scales, afflicting individuals, families, communities, or entire populations. But only those in the last category supersede the ability of markets and insurance systems to absorb some of the impact of their effects.

Our focus, then, is on events that most would already perceive as terrible and worth avoiding, even at considerable cost. This insight might lead some to wonder why these events merit further study at all. But government decisions regarding crisis prevention and mediation rely on information on the costs and benefits of such interventions. The evidence we review suggests that the effects of aggregate crises are both longer lasting (felt over the lifecycle) and more extensive (involving nonobvious human biological outcomes) than many might appreciate. Thus, from a policy perspective, our review may lend additional support to arguments in favor of increased disaster preparedness

or increased focus on post-disaster adaptation strategies. From a broader scientific perspective, however, the results cast in stark relief the way the human body reacts to a variety of grave insults.

We organize our discussion around five types of aggregate crises: recessions, famines, epidemics, natural and environmental disasters, and wars.[3] Taking a broad view of "human biology"—including health, child development, and reproduction—we detail the effects of each type of crisis on human biological outcomes. Whenever the literature allows, we discuss the roles of health endowments, incomes, prices, and behaviors in mediating the results, and we note how the results may vary between rich and poor settings. Notably, one type of crisis can lead to another crisis—for example, a disease epidemic can cause economic fluctuations, or a war can result in a famine. We note these possibilities as potential pathways where relevant, but, due to space considerations, we focus on the impacts of the crisis that likely occurred first. In the same vein, we do not dwell on the specific pathways through which crises affect human biology. Little existing research sheds light on these mechanisms, so we point to them as a fruitful topic for future research.

3.2 Conceptual Issues

Before reviewing the evidence, we further develop our definitions of the two concepts at the center of this chapter: crisis and human biology. To begin, we set the selection criteria for the crises we study, discussing their theoretical underpinnings and implications. We describe how behavioral responses, price adjustments, and insurance arrangements may mitigate or exacerbate the effects of a crisis, with particular attention paid to how these mechanisms may play out at different levels of aggregation. The research we review does not necessarily shed direct light on these mechanisms, but we draw attention to them as a way to motivate our choice to focus on large-scale crises. After defining crisis, we move on to setting the parameters for the outcomes we consider.

Our definition of crisis narrows the scope of the relevant literature considerably. The *acute* criterion precludes us from focusing on serious, long-lasting problems that many label "crises," such as what the editorial board of *The Lancet* (2013) calls the "global crisis of . . . malnutrition in children." The *severe* criterion leads us to direct our attention to studies of large shocks, rather than marginal changes in rainfall in agricultural areas, for example. And the *unexpected* criterion forces us to exclude important problems that unfold in a gradual and anticipated way, such as the so-called pension crisis. Our restrictive definition does occasionally rule out relevant research that falls slightly outside its boundaries. In these cases, we allow ourselves to deviate. For example, we include research on the HIV/AIDS "crisis," even if the pandemic is by now chronic and expected. We reason that it was once acute and unexpected and that, viewed through a broader lens, its history is still short.

That said, even with the three criteria, our definition includes a very wide range of negative shocks with a wide range of effects. Some crises affect individuals or families in isolation; for instance, when a parent loses his job or dies. Others occur at the local level, such as when a plant closes or an agricultural community experiences a growing season with limited rain. And yet others strike entire populations or economies; these crises, which include famines and sudden epidemics, receive the most attention from policy makers and the media because they are so wide-reaching. Crises at these three levels are likely to have differing effects on human biology depending on the extent of risk sharing, market integration, reserves, and safety nets. Aggregate crises are worse than the sum of many localized crises because they have stronger price effects and are more difficult to insure. If a single agricultural community has a bad crop year, then its members may suffer income losses. If an entire region has a bad crop year, however, these income losses may be compounded by increases in the price of food. More generally, the impact of a crisis is contingent on both the extent of *ex ante* insurance—whether through buffer-stock saving, formal insurance systems, informal risk sharing, or government safety nets—and the extent of *ex post* behavioral responses (see Skoufias, 2003, for more on coping strategies relevant to economic crises and natural disasters). For these reasons, we focus on aggregate crises. The scope of aggregation may vary—a state in one application, a country in the next—but we maintain a focus on crises broad enough to defy many risk mitigation strategies and price adjustment effects.

Apart from the characterization of crisis, our topic also requires us to define human biology. We take a broad view of the concept, including mortality, physical growth, physical morbidity, mental health, cognitive function, and fertility. These domains of human biology have considerable overlap in the literature. In some cases, the impact of crisis in one domain mediates its impact in another. In other cases, the impact in one domain complicates estimation of impacts in another. For example, impacts on mortality or fertility may introduce selection bias in estimates of impacts on morbidity or cognitive function among survivors. This concern arises in all research concerning shocks with potential effects on population composition, but it is especially pertinent for the study of major health crises, such as famines or pandemics.

Much of the research we review follows how the human biology impact of a crisis spills over into social and economic outcomes. We include such analyses in our discussion only when we can confidently attribute the effects of a crisis on social and economic outcomes to a human biology pathway. For example, one can safely trace the human capital effects of disease exposure in utero to a human biology pathway, but the same link is not possible for the lifelong consequences of exposure to war in childhood.

3.3 Recessions

Of all the crises we consider, recessions are the most natural for economists to study. Importantly, recessions typically have other root causes, some of which we consider in

subsequent sections. But a large literature considers the effects of recessions per se, perhaps because recessions with varying causes often share common features. This literature pays special attention to the interplay between *income effects* owing to contractions in economic resources and *substitution effects* from reductions in the opportunity cost of time, for example. Much of the literature estimates the relationship between continuous measures of aggregate economic conditions (such as growth rates or employment rates) and human biology, which bundles together booms, mild recessions, and deep recessions. Although we are most interested in the effects of deep recessions, the work on these continuous measures provides important complementary evidence, so we include it in our review.

Perhaps the most striking finding in this literature is that population health improves during economic downturns in wealthy countries. Using both aggregate time-series and state-level panel data from the United States, Ruhm (2000) finds that a percentage point increase in the unemployment rate is associated with a .5% decrease in all-age mortality. Gerdtham and Ruhm (2006) find similar evidence from across the Organization for Economic Cooperation and Development (OECD), as do Granados, Tapia, and Roux (2009) for the historical United States, even in crises so pronounced as the Great Depression.[4] In seeking an explanation for these results, Ruhm (2000) emphasizes that the opportunity costs of leisure and other health inputs decrease during recessions, as do negative externalities from increased economic activity.[5] Evidence for these mechanisms is mixed in contemporary data. On the one hand, he finds that sedentarism, smoking, drinking, and fat consumption are pro-cyclical, consistent with opportunity cost effects from higher wages and job stress. Additionally, mortality from vehicle accidents is strongly pro-cyclical, consistent with externalities from increased activity.

On the other hand, Stevens, Miller, Page, and Filipski (2011) show that pro-cyclical mortality is concentrated among young children, twenty-somethings, and especially the elderly, for whom the opportunity cost of time is small; the pattern is absent among adults of prime working age. These findings are difficult to reconcile with a theory that indicates individuals take worse care of themselves during recessions. Rather, Stevens et al. propose that mortality declines during recessions primarily reflect cyclicality in the quality of health care, perhaps due to staffing costs. They report that staffing in skilled nursing facilities rises during recessions and also that pro-cyclicality is more pronounced for deaths occurring in nursing homes and in states where more elderly live in nursing homes.

Pro-cyclical mortality and counter-cyclical health are also apparent among the very young, but these patterns may have other causes, most importantly because changes in fertility patterns influence the distribution of health among infants. Fertility falls during recessions in the United States (Currie & Schwandt, 2013, in both national time series and state-level panel data. If this pattern varies across different types of women, then it may affect the distribution of child health. Indeed, Dehejia and Lleras-Muney (2004) present evidence that the composition of new mothers changes during recessions in a way that increases the prevalence of maternal characteristics that promote child health. At the same time, holding these characteristics constant, maternal health

behavior improves during recessions. Both margins of change—selection and behavioral adjustment—improve the average health of US infants born during recessions, both across the country and within states. A percentage point increase in the unemployment rate is associated with a .25–.5% decline in the prevalence of low birthweight.

In poor countries, the opposite pattern generally holds. Analyzing Demographic and Health Survey data from 59 African, Asian, and Latin American countries, Baird, Friedman, and Schady (2011) find strong counter-cyclicality in infant mortality. Within countries over time, fluctuations in gross domestic product (GDP) per capita are negatively associated with fluctuations in infant mortality, a result robust to the inclusion of flexible country-specific time trends and thus accounting for joint secular trends in economy and health. The result is also robust to mother fixed effects—thus controlling for changes in the composition of mothers. A 1% decrease in GDP per capita adds .25–.5 infant deaths per 1,000 live births. Most country-specific studies in the developing world also find that mortality rises in downturns (Bhalotra, 2010; Cutler, Knaul, Lozano, Méndez, & Zurita, 2002; Paxson & Schady, 2005). Where data exist, they suggest similar results for other health outcomes such as birthweight (Bozzoli & Quintana-Domeque 2014). Nevertheless, counterexamples also exist. In Colombia, sudden drops in the international price of coffee cause infant health to improve in coffee-growing regions, arguably because of falling opportunity costs of maternal time with children (Miller & Urdinola, 2010). In Indonesia, child weight-for-age held steady through the financial crisis because mothers buffered children's caloric intake by eating less themselves (Block et al., 2004). Broadly speaking, however, most results point to pro-cyclical health in developing countries.

The differences in results between rich and poor countries suggest that income effects may dominate when a recession reduces resources to dire levels, as in a crisis. This theory would be compelling if the effects of booms and busts had asymmetric effects, in which busts have stronger negative effects than booms have positive effects. The literature offers some evidence to this effect. In poor countries, deep recessions elevate female infant mortality particularly strongly—far exceeding effects proportional to the impacts of small negative shocks or positive shocks (Baird et al., 2011). But in a twist that remains open to interpretation, the effects of aggregate shocks on male infant mortality are both smaller and more symmetric. Furthermore, infant mortality is neither significantly more counter-cyclical in low-income (compared to middle-income) countries, nor in children born to less-educated (compared to more-educated) women.

Beyond these contemporaneous impacts, research suggests that survivors of early-life exposure to economic crisis may suffer life-long sequelae in developing countries. The most convincing evidence comes from the historical record. In Dutch cohorts from the 19th century, those with birth years coinciding with recessions experienced higher mortality risk through the life cycle (van den Berg, Lindeboom, & Portrait, 2006). Around the same time, men born during the Great French Wine Blight exhibited relative shortfalls in adult height if they were born in wine-growing regions that underwent a deep recession (Banerjee, Duflo, Postel-Vinay, & Watts, 2010). More recent evidence also suggests that exposure to economic shocks in early life can result in mental health

impacts in adulthood (Adhvaryu, Fenske, & Nyshadham, 2014). At least for the young, the human biology impacts of deep recessions last far longer than do the direct economic impacts.

3.4 Famines

An extreme manifestation of recession is famine, a phenomenon that has received much attention in the economics literature at least since Amartya Sen's (1981) analysis of the Bengal Famine of 1944 (see also Ó Gráda, 2009). The idea that famines kill is not novel; hence, the literature we summarize in this section addresses the issue of how famines affect the health and human capital of those who *survive*. This relationship is key to the notion that famines can have consequences beyond the loss of life in the short run.

Selection poses a major stumbling block to estimating the effects of famine. If mortality is concentrated among the weakest individuals, then survivors of famine will be positively selected, leading researchers to understate the impact of famine on survivors. Moreover, although the weather plays an important role in triggering famines, the intensity and duration of famines often result from failures of policy and political will. Hence, areas affected by famines might also be areas that otherwise would have received less public transfers, for example. Survivors are therefore likely to be selected in a way that resembles selective sorting into cities or neighborhoods, possibly confounding the analysis.

An influential series of papers examines the long-run impacts of the Dutch famine, which occurred in 1944–45. Reviewing these papers, Roseboom et al. (2001) conclude that prenatal exposure to the Dutch famine resulted in worse adult health along various dimensions, especially coronary heart disease (CHD). The studies focus not only on long-term health as an outcome but also on whether the *timing* of the famine in utero matters in different ways. Cohorts exposed during the first trimester were affected differently than cohorts exposed during the third trimester. For example, cohorts exposed late in gestation tended to have lower weight at birth and increased glucose concentrations (a marker for diabetes) in adulthood. Cohorts affected in the first trimester, in contrast, tended to not have any lower birthweight but had higher tendencies for CHD in adulthood. Perhaps the most interesting conclusion of the Dutch famine studies is that undernutrition during gestation can affect long-term health even if there is no indication of poorer health at birth (as measured by birthweight). Other studies on this particular famine have also found intergenerational birthweight effects, suggesting that the effects of famine last across multiple generations (Stein & Lumey, 2000).[6]

Researchers have examined the long-run health consequences of famines in many other contexts as well. In China, Gørgens, Meng, and Vaithainathan (2012) studied the Great Famine (1959–61) and found that survivors of early-childhood exposure were significantly shorter than people who were not exposed. An important contribution of this study is its idea to use the *children* of survivors to control for selection into survivorship.

Past research on the Chinese famine has struggled to find effects on survivors because famine mortality was apparently linked with potential height. The authors argue that although children inherit the genotype of their parents with regards to height, they do not inherit their phenotype. So they adjust for height differences in the next generation and interpret the residual height deficit in affected cohorts as the effect of famine. Notably, this strategy assumes that the scarring effects of famine do not transcend generations, which seems difficult to defend a priori. Nonetheless, the study makes important progress on dealing with selection effects.[7]

In a different context, Dercon and Porter (2010) highlight similar results stemming from one of Africa's worst famines, which occurred in Ethiopia in 1984. Using a household measure of famine intensity and comparing siblings with different famine exposure, the authors find that early-life famine exposure significantly reduces height in adulthood by at least 3 cm. Comparing exposed and nonexposed siblings is another way to control for selection effects because siblings have similar height potentials.

Although some studies show conflicting results of famine exposure (Luo, Mu, & Zhang, 2006; Stanner et al., 1997), the studies that explicitly account for selection and measurement error seem to consistently show a negative effect on health of famine exposure. A logical next step, then, is to examine how survivors fare in school, on the marriage market, and on the labor market, as recent studies have done.[8] Overall, these studies find that famine exposure at young ages (typically measured in utero or before the age of 2) negatively impacts long-term nonhealth outcomes. Most of these studies use similar strategies of comparing exposed and nonexposed cohorts in areas with differing famine intensity. We highlight two complementary studies that examine the long-run labor market impacts of the Chinese famine.

Almond, Edlund, Li, and Zhang (2010) use multiple sources of variation to study the impacts of the famine. Choosing a narrow window of birth cohorts (1956–64) to reduce confounding, they compare cohort-level changes across provinces with differing famine intensity. They also use residents of Hong Kong, which was under British rule and was thus unaffected by the famine, as a second control group. Among men, they find that in utero exposure to the famine increases illiteracy by 9%, reduces labor force participation by 6%, and reduces the probability of marriage by 6.5%. Women are similarly impacted, albeit with smaller magnitudes.

Meng and Qian (2009) build on this study by using a finer source of variation (county-level rather than province-level) and by using institutional features of the centrally planned procurement system to instrument for famine intensity. To account for positive selection into survival, they estimate the effect of exposure on the upper quantiles of the outcomes of interest. Their findings are consistent with prior studies on the famine (in terms of health and educational attainment), but accounting for measurement error and selection leads to larger magnitudes than those found in prior studies. They find that in utero famine exposure reduces the 90th percentile of adult height by 3 cm, weight by 1.5 kg, and educational attainment by half a year.

3.5 Epidemics

Like famines, epidemics have direct effects on human biology, and hence, economic research has contributed to our understanding of these crises mainly by looking for broad, long-term, or indirect impacts. Here again, changes in patterns of mortality and fertility complicate the estimation of impacts among survivors, but, in this case, they may also have interesting macroeconomic implications. At least since Malthus (1798/1966), economists have noted that widespread disease may increase the ratios of land or capital to labor, thus raising per capita living standards.[9] Along these lines, Voigtländer and Voth (2013) present evidence that the Black Death was a crucial turning point in the emergence of modern economic growth in Europe. A key feature of the bubonic plague in this respect is its rapid progression to death; as a result, it caused widespread mortality without sustained morbidity. More generally, the macroeconomic effects of an epidemic depend on its relative mortality and morbidity burdens. With greater morbidity, the surviving population becomes less productive, pushing back against the Malthusian benefits of epidemic mortality.

A fitting example is HIV/AIDS, which Young (2005) argues may bring increased prosperity to Africa. Incorporating positive wage effects from AIDS mortality, negative fertility responses to HIV, and negative effects of orphanhood on the next generation's human capital, Young calibrates a positive net effect of the epidemic on future living standards. However, the calibration depends heavily on his assumption that morbidity from HIV/AIDS does not meaningfully decrease productivity and on his estimate that fertility falls with rising HIV prevalence.

Subsequent research casts doubt on both of these crucial ingredients to Young's argument. First, HIV has become a chronic disease, and mounting evidence suggests that it has serious productivity consequences (Habyarimana, Mbakile, & Pop-Eleches, 2010; Levinsohn, McLaren, Shisana, & Zuma, 2013; Thirumurthy, Zivin, & Goldstein, 2008). Second, follow-up research has found that increases in regional and community-level HIV prevalence are not associated with falling fertility (Fortson, 2009; Juhn, Kalemli-Ozcan, & Turan, 2013). Using these revised fertility estimates, Kalemli-Ozcan and Turan (2011) recalibrate Young's model and find no macroeconomic benefit from HIV/AIDS.

A further indirect effect of the HIV pandemic on human biology is that it diverts resources and attention away from other important health care goals. Analyzing data from 14 sub-Saharan African countries from 1988 to 2005, Case and Paxson (2011) find that subnational regions with larger increases in HIV prevalence experienced erosions in antenatal care, institutional deliveries, and immunization. In corroborating evidence, Grépin (2012) analyzes country-level panel data to show that expansions in international aid for HIV programs are associated with declines in immunization. Such crowd-out effects are not limited to HIV. In Taiwan, for example, Bennett, Chiang, and Malani (2015) find that the onset of the severe adult respiratory syndrome (SARS) epidemic reduced outpatient medical visits by nearly one-third within a few weeks. Thus,

epidemics affect health care access not only through policy makers' resource allocation decisions but also through fear of contagion in health care settings.

At the microeconomic level, studies show lasting effects of early-life morbidity due to epidemics. The 1918 influenza pandemic has attracted particular attention due to its sharp, unexpected, and indiscriminate nature. As with other crises involving significant mortality, positive mortality selection is likely to bias researchers toward finding no long-term effect on survivors. Nevertheless, drawing on both cohort-level variation and state-cohort panel variation in the United States, Almond (2006) estimates that in utero exposure to the flu pandemic reduced educational attainment, reduced adult income, and raised adult disability.[10] Cohorts that were in utero during the pandemic were 4% less likely to finish high school, 3% more likely to be poor, and 8% more likely to have a disability that prevented work in middle age.

Other research examines the lasting consequences of early-life exposure to malaria, a disease known for its widespread toll. Some of this literature studies the consequences of malaria eradication in settings where the disease is endemic (Bleakley, 2010; Cutler, Fung, Kremer, Singhal, & Vogl, 2010; Lucas, 2010), leveraging regional differences in baseline prevalence to isolate variation in the extent of malaria decline due to eradication. Comparing cohorts born before versus after eradication in high- versus low-prevalence areas, these studies find largely positive effects of malaria-free childhood human capital and labor market outcomes. The elimination of an endemic disease has little to do with crisis, but Lucas (2010) notes that Paraguay underwent a pronounced epidemic just before its eradication campaign. In that setting, the elimination of *epidemic* malaria at birth raised schooling by three-quarters of a year in the most infected region. Further evidence on the consequences of in utero exposure to a malaria crisis can be found in Barreca's (2010) analysis of malaria outbreaks in the early-20th-century United States. Drawing on an ordinary least squares (OLS) specification with state and birth cohort fixed effects, as well as an instrumental variables strategy that relies on climatic fluctuations to identify the effects of malaria outbreaks, Barreca finds that early-life exposure to epidemic malaria reduces schooling and adult labor supply while increasing poverty. A standard deviation increase in the malaria death rate in high malaria states during gestation led to a 0.04-year decrease in educational attainment, a 0.35 percentage point decrease in full-year work, and a 0.38 percentage point increase in poverty. These analyses shed much light on how health insults in early life affect the trajectories of social and economic outcomes over the life cycle.

3.6 Natural Disasters and Environmental Crises

Natural disasters like earthquakes and floods often result in loss of life and property, as do environmental crises like nuclear accidents and dramatic increases in air pollution.

Although both are forms of "crisis," one can perhaps distinguish them on the basis of the extent of human involvement in their genesis. This differentiation is rather loose, however, because some natural disasters are at least partly attributable to human action via climate change. Hence, we treat them jointly in this section.

We begin with papers that examine the impact of natural or environmental disasters on fertility. In the case of fertility, the effect of natural or environmental disaster can be positive or negative. Fertility may fall due to a loss of property or income, or it may rise if parents wish to "replace" deceased children. At least in developing countries, the evidence points toward replacement effects. Examining responses to three different earthquakes, Finlay (2009) finds that fertility rises after an earthquake. Similarly, studying the aftermath of the 2004 Indian Ocean Tsunami in Indonesia, Nobles, Frankenberg, and Thomas (2014) find increased fertility in harder-hit areas. The effect reflected both the behavior of women whose children died (thus replacing their *own* lost children) and the behavior of women who did not yet have children (thus replacing the *community*'s lost children). These results stand in contrast to that of Lin (2010), who finds that in Italy and Japan, short-run instability due to natural disasters is associated with decreased fertility.

Many papers have documented the mortality effects of natural disasters and extreme weather events. For example, in a paper examining the effects of natural disasters in more than 141 countries over two decades, Neumayer and Plümper (2007) find that mortality effects of natural disasters tend to be concentrated among women. This is an important study because it suggests relevant inequalities in the impact of natural disasters. Since mortality effects of natural disasters are well-documented in other review articles (see, e.g., Cavallo & Noy, 2010, and Bourque, Siegel, Megumi, & Wood, 2007) we now focus our attention to perhaps less widely studied extreme weather events. For example, Deschenes and Moretti (2009) find that both extreme heat and extreme cold result in short-run mortality increases, with different causes of mortality at different ends of the temperature spectrum. Heat primarily advances the mortality of those who are already weak by a few days or weeks. In response to cold extremes, however, people who might otherwise live a few more years might die; hence, the mortality impact of extreme cold is longer lasting. Given the general pattern of mobility in the United States, with people moving from colder to warmer climates, this finding suggests that migration could be one driving force behind increasing life expectancy. An important addition to this body of work is the recent research of Barreca, Clay, Deschenes, Greenstone, and Shapiro (2013), who find that the heat-mortality relationship in the United States has declined in recent decades. They attribute this decline to the adoption of air conditioning.

Extreme weather also has short-run consequences in the developing country context. In a broad study examining the impacts of early-life weather conditions on infant mortality in 28 African countries, Kudamatsu, Persson, and Strömberg (2012) find that extreme weather fluctuations have a significant impact on infant mortality in Africa via malnutrition and malaria. Analyzing even more extreme weather variation, Anttila-Hughes and Hsiang (2013) study the aftermath of typhoons in the Philippines, finding elevated infant mortality rates that far exceed the direct effects of the storms. Burgess, Deschenes, Donaldson, and Greenstone (2013) also estimate a significant relationship

between weather and mortality in India, which is mostly driven by high temperature extremes at the time of crop growth in rural areas. In a related paper that speaks to differences between aggregate crises and local shocks, Burgess and Donaldson (2010) show that the expansion of India's railroad network diminished the mortality impact of agriculturally damaging weather shocks.

The evidence of the impact of pollution crises on health in developing countries is considerably less. Jayachandran (2009) is one of the few researchers to examine the mortality effects of forest fires. Forest fires produce atmospheric pollution that can travel large distances, with the potential of affecting the health of people far away. Using data from Indonesia, she finds that prenatal exposure to particulate matter due to forest fires in 1997 led to around 16,400 fewer surviving infants in Indonesia. Changing focus to urban pollution, Arceo-Gomez, Hanna, and Oliva (2012) use variation due to thermal inversions (which despite their frequency might qualify as crises) to find a similar result that pollution exposure is a significant contributor to infant mortality in Mexico City.[11] Several other recent papers examine the effects of marginal changes in environmental damage in developing countries, which fall outside the purview of our review.

Along similar lines, a large literature considers the long-term effects of early-life exposure to pollution (Bharadwaj, Gibson, Zivin, & Neilson, 2014; Currie, Zivin, Mullins, & Neidell, 2013, but much of this literature considers marginal changes in pollution levels or environmental policies that improve pollution, neither of which qualifies as a crisis. A notable exception is Almond, Edlund, and Palme (2009) analysis of the Chernobyl nuclear disaster's aftermath. Leveraging spatial and temporal variation in exposure to the radiation cloud in Sweden, Almond et al. find that prenatal exposure to radiation decreases cognitive achievement in later life, albeit without observable health impacts.[12]

3.7 Wars

Many of the crises we review in part reflect human action, but nowhere is human responsibility graver than in the case of war. Wars and other conflicts are disruptive along social, political, and economic lines, with significant potential to affect human biology.[13] Most research on this topic has focused on the impact of conflict exposure in early childhood on subsequent physical growth. Analyses of this question must grapple with standard concerns about selective mortality, fertility, and migration, in addition to the fact that wars often accompany other undesirable phenomena: recession, disease, food shortage, and deterioration of health infrastructure, *inter alia*. Although separately identifying each mechanism would be an interesting area for future research, existing research does not concern itself with isolating the underlying mechanism.

The Nigerian Civil War was one of the earliest civil wars in postindependence Africa, making it a suitable context for studying long-run effects of childhood exposure. Akresh, Bhalotra, Leone, and Osili (2012) examine its impact on adult stature, finding that individuals from ethnic groups most heavily exposed to the war attained

significantly lower stature as adults. Exposure to conflict during adolescence was more damaging than exposure only in early childhood, which the authors attribute to possible disruptions in the normal growth spurt experienced in adolescence. Adult height is also the primary outcome in Agüero and Deolalikar's (2012) study of the Rwandan genocide of 1994. Whereas they, too, find that exposure to the genocide leads to decreased height (relative to trends in neighboring countries), they find the effects to be greater when exposure occurs at a younger age. Thus, while both Akresh, Lucchetti, and Thirumurthy (2012) and Agüero and Deolalikar (2012) find that childhood and adolescent exposure to wars reduces adult height, they differ on when exposure matters more.

Shifting from adult outcomes to child outcomes, Bundervoet, Verwimp, and Akresh (2009) examine the consequences of exposure to civil war in Burundi on child height-for-age *z*-scores. The authors use variation in the timing and geographic spread of the war to estimate that an additional month of civil war exposure reduced height for age by about 0.05 *z*-scores. The effects are concentrated in children who were exposed to conflict between ages 0 and 2 years, consistent with the idea that the first few years of life are a critical period for physical growth.

When two countries go to war, residents of the winning country might benefit by suffering less destruction or disruptions to economic systems or public health delivery. Hence, when thinking about the impacts of intercountry conflict, a crucial question arises regarding the *net* health impacts of such conflict. Akresh, Caruso, and Thirumurthy (2014) address this issue by examining the health impacts on both sides of the Eritrean-Ethiopian conflict of 1998–2000. Children on both sides suffered equally in terms of the effect on height-for-age *z*-scores, but children in the losing nation suffered more than those in the winning country.

Whereas war-induced migration is a central concern for interpretation of the results so far, the arrival of refugees in large numbers could also pose a health risk to the locals in the areas where they arrive. Baez (2011) examines the health of local children as a function of the refugee influx from the genocides in Burundi and Rwanda. In 1994–95, North-Western Tanzania received hundreds of thousands of refugees; however, topographical characteristics induced geographical variation in refugee inflows. Baez uses this variation to estimate negative effects on the health (as measured by anthropometrics and child mortality) and human capital attainment of local children 1 year after the arrival of refugees. We hope this important study opens the door to further research on the health impacts of conflict for people not directly involved in the conflict.

3.8 Conclusion

A burgeoning economics literature considers the effects of various shocks on human biology. Mixed into this literature are shocks large and small, positive and negative, local and aggregate. In this review, we have homed in on a subset of these shocks—aggregate crises—in the hope of highlighting commonalities in their effects. The

literature suggests that these unexpected, pronounced, negative, and population-wide events affect human reproduction, mortality, and morbidity over the life cycle. To shed further light on the roles of prices and insurance arrangements, a fruitful line of future inquiry would compare crises at different scales (Bundervoet et al., 2009 and Caruso, 2015 are recent examples) or those occurring in environments with differing levels of market integration or insurance system development (Kahn, 2005, is an excellent example).

More broadly, however, the literature highlights the lasting effects that social, economic, political, environmental, and pathological crises have on the human body. Children, who are never complicit in creating crisis, carry the burden of exposure for the rest of their lives. Although advances in methodology and data availability have allowed researchers to uncover these nuanced but powerful effects, much work remains in improving crisis response, especially in poor countries. As the findings demonstrate, improvements in this arena would have beneficial effects long after the acute period of a crisis subsides, on outcomes far beyond its most obvious sequelae.

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Notes

1. In the *Oxford English Dictionary*, all quotations with the word before the year 1600 used this definition.
2. See Ruhm (2006), Strauss and Thomas (2007), and Currie and Vogl (2013) for reviews.
3. We omit major political crises, for example regime change (as in the Soviet Union) or political partition and reunification (as in the partition of British India or the reunification of Germany).
4. Despite the overall mortality decrease during the Great Depression, relief spending during this era was associated with further decreases in mortality (Fishback, Haines, & Kator, 2007).
5. For additional evidence on these mechanisms, see Ásgeirsdóttir, Corman, Noonan, Ólafsdóttir, and Reichman (2014).
6. For more recent results on the Dutch famine, see Painter et al. (2006); Stein et al. (2007); Rooij, Painter, Holleman, Bossuyt, and Roseboom (2007); and Rooij Wouters, Yonker, Painter, and Roseboom (2010).
7. Mu and Zhang (2011) find that female survivors have a higher incidence of disability, which they attribute to greater selective male mortality during the famine. Two other papers use similar strategies to find that exposed cohorts are shorter in the long run (Chen & Zhou, 2007; Meng & Qian, 2009).

8. See Shi (2011), Almond (2006), Brandt, Siow, and Vogel (2008), and Meng and Qian (2009) on China; Dercon and Porter (2010) on Ethiopia; Scholte, van den Berg, and Lindeboom (2015) on the Netherlands; and Neelsen and Stratmann (2011) on Greece.
9. The same general equilibrium reasoning also applies to famine and war, but it has drawn more interest in the literature on disease.
10. Brown and Thomas (2011) point out that the 1918 pandemic coincided with World War I military deployments that changed the composition of new parents, which they argue can account for much of Almond's estimated flu impact. However, data from countries with less involvement in World War I indicate similar impacts of in utero exposure to the pandemic (Lin & Liu, 2014; Richter & Robling, 2013).
11. A thermal inversion occurs when warm air settles over a layer of cooler air near the ground, trapping the cool air and any pollutants inside it.
12. In a related paper using nuclear weapons testing rather than nuclear disaster as a source of variation, Black, Bütikofer, Devereux, and Kjell (2013) find similar long-run impacts of prenatal exposure.
13. Whereas the term "war" is typically used to denote fighting across borders, and the term "conflict" often denotes within-country fighting (such as civil or ethnic conflicts), we use the terms interchangeably in this chapter.

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CHAPTER 4

THE BIOLOGICAL STANDARD OF LIVING IN EUROPE FROM THE LATE IRON AGE TO THE LITTLE ICE AGE

NIKOLA KOEPKE

4.1 Introduction

It is generally accepted that human well-being is a multidimensional concept that encompasses much more than command over goods and services (Boarini, Johansson, & Mira d'Ercole, 2006; World Health Organization [WHO], 1995) and that nutrition and health (individually and in synergistic relationship) are fundamental aspects of living standards. Because nutrition and health are not necessarily related to material well-being, the study of the biological standard of living (BLS) is an important complementary dimension of overall well-being.[1]

Anthropometric indicators, especially physical mean height as a proxy of a population's BLS, are advantageous measures of well-being insofar as they capture how well a society is able to provide adequate living conditions for the population (Komlos, 1998). This is the case because about 20% of the variation in height—and the chance of growing to one's full height potential—is determined by environmental factors (McEvoy & Visscher, 2009; Silventoinen, 2003). Moreover, mean height as a measurement of well-being has the advantage of being applicable to the diverse social and economic systems that existed over the course of time, and its use makes it possible to adequately depict the various spheres of inequality in a population.

Using the anthropometric approach is particularly important for pre-modern periods because only a very few and limited written and archaeological sources give information on other measures of well-being in the very long run. Instead, skeletal remains provide information on mean height and thus enable us to investigate BLS much farther