

IZA DP No. 4340

Population and Health Policies

T. Paul Schultz

August 2009

Population and Health Policies

T. Paul Schultz

*Yale University
and IZA*

Discussion Paper No. 4340
August 2009

IZA

P.O. Box 7240
53072 Bonn
Germany

Phone: +49-228-3894-0
Fax: +49-228-3894-180
E-mail: iza@iza.org

Any opinions expressed here are those of the author(s) and not those of IZA. Research published in this series may include views on policy, but the institute itself takes no institutional policy positions.

The Institute for the Study of Labor (IZA) in Bonn is a local and virtual international research center and a place of communication between science, politics and business. IZA is an independent nonprofit organization supported by Deutsche Post Foundation. The center is associated with the University of Bonn and offers a stimulating research environment through its international network, workshops and conferences, data service, project support, research visits and doctoral program. IZA engages in (i) original and internationally competitive research in all fields of labor economics, (ii) development of policy concepts, and (iii) dissemination of research results and concepts to the interested public.

IZA Discussion Papers often represent preliminary work and are circulated to encourage discussion. Citation of such a paper should account for its provisional character. A revised version may be available directly from the author.

ABSTRACT

Population and Health Policies

The program evaluation literature for population and health policies is in flux, with many disciplines documenting biological and behavioral linkages from fetal development to late life mortality, chronic disease, and disability, though their implications for policy remain uncertain. Both macro- and microeconomics seek to understand and incorporate connections between economic development and the demographic transition. The focus here is on research methods, findings, and questions that economists can clarify regarding the causal relationships between economic development, health outcomes, and reproductive behavior, which operate in many directions, posing problems for identifying causal pathways. The connection between conditions under which people live and their expected life span and health status refers to “health production functions.” The relationships between an individual's stock of health and productivity, well-being, and duration of life encompasses the “returns to health human capital.” The control of reproduction improves directly the well-being of women, and the economic opportunities of her offspring. The choice of population policies may be country specific and conditional on institutional setting, even though many advances in biomedical and public health knowledge, including modern methods of birth control, are now widely available. Evaluation of a policy intervention in terms of cost effectiveness is typically more than a question of technological efficiency, but also the motivation for adoption, and the behavioral responsiveness to the intervention of individuals, families, networks, and communities. Well-specified research strategies are required to address (1) the economic production of health capacities from conception to old age; (2) the wage returns to increasing health status attributable to policy interventions; (3) the conditions affecting fertility, family time allocation, and human capital investments; and (4) the consequences for women and their families of policies which change the timing as well as number of births.

JEL Classification: D13, I18, J13, O12

Keywords: health, fertility and family planning, biology of health human capital, economic development

Corresponding author:

T. Paul Schultz
Department of Economics
Yale University
Box 208269
New Haven, CT 06520-8269
USA
E-mail: paul.schultz@yale.edu

1. INTRODUCTION

Population policies are primarily a response to the anticipated consequences of fertility and mortality, and secondarily to internal and international migration that also modify the size, age composition, and regional distribution of the population. It is not novel that economists, especially macro- and micro-oriented economists who tend to examine empirically different types of data, country aggregates or individual and household observations, have not always reached a consensus on population issues. However, recognizing the differences between these research traditions and their findings in this chapter may help formulate working hypotheses that warrant more study, and may guide research on and empirical testing of these hypotheses in a common framework to close some of the existing gaps in our knowledge.

One underlying development that motivates much population policy research, directly or indirectly, is the demographic transition that involves first a decline in age-specific mortality rates, and then is generally followed by a decline in fertility rates. During the interval between the onset of the decline in mortality and the sustained and substantial decline in fertility, the natural rate of population growth tends to increase and the age composition of the population changes. This may create opportunities or imbalances in the aggregate economy for which social welfare policies are sometimes proposed to improve prospects, typically through the introduction of subsidies, taxes, transfers and regulatory policies to deal with externalities. These changes in mortality and fertility are related to the reallocation of family resources over its life cycle, and these interdependent behavioral responses of women and men, parents and children, provide the core microeconomic issues for study. Without an understanding of how people respond to the provision of new health opportunities and means for controlling births, it is difficult to discuss the tradeoffs on which population policies seek traction. Without knowledge of the technical features of health production functions, and the marginal health gains associated with the use of health-related inputs and behaviors, policies affecting health and well-being will be difficult to evaluate. Biological issues also affect how reproduction is modeled and technological change in birth control is viewed from a policy perspective.

The average length of life in the world has approximately doubled from the start of the nineteenth to the start of the twenty-first centuries, from 30-35 years to 60-70 years, with the recent notable exception being several states in Southern and Eastern Africa where life

expectancy has declined due to the HIV/AIDS epidemic. This increase in length of life has paralleled the spread of modern economic growth and the increase in household personal consumption per adult (Kuznets, 1966). But the connections between the economic conditions under which people live and their longer lives, on the one hand, and the relationship between the improved health status of adults and their economic productivity as workers, on the other hand, are two possible causal relationships underlying this covariation of life span and economic growth. Understanding these causal relationships could inform the choice of population policies related to health, family planning, and migration, and improve the basis for predicting future economic development. A goal of this chapter is therefore to describe what we are learning about these underlying causal relationships, and how social scientists are improving the empirical measurement of these causal relationships that could become the basis for better development policy.

Evidence on these relationships is accumulating in a variety of disciplines and subfields of economics, and a number of emerging hypotheses merit refinement and concerted empirical study to test the magnitude of behavioral and technical responses, to determine which biological and behavioral pathways are involved in these responses, and to assess longer-run consequences of programs and policies after individuals and families reallocate their lifetime resources. Household sample survey data are an important resource for this research, especially panel surveys which interview the same individuals and families repeatedly over time, to assess more rigorously causal relationships affecting behavior and welfare. These empirical assessments are generally premised on exclusion restrictions embedded in implicit structural models. The biological and behavioral processes underlying the current improvement in health status are complex, with long-gestating lags linking the growth of the fetus and early childhood biological development, all the way to late life mortality, disability, and health status, as well as impacting intermediate observable outcomes such as cognitive achievements (IQ), schooling, productivity, fertility, and other forms of behavioral adaptation to local environments and policy conditions. The political economy governing the performance of social organizations affecting health and schooling may also be impacted. There is substantial uncertainty about even the short-run effects of social policy interventions on health, fertility, and population growth, and even less certainty regarding the longer-run effects as can only be inferred from the study of long time series and panel data, which are exceedingly rare.

The agenda for research on population policies requires a simultaneous description of the determinants of the *supply* of public produced health-related services and birth control, and the determinants of private household *demands* for those services and technologies, including preventive or curative health services, social insurance, subsidies for family planning technologies, schooling attainment of boys and girls, the health and reproductive health content of schooling, etc. Given the complexity of these interdependent behavioral, organizational, and technological processes, some economists propose that social experiments are needed to evaluate reliably the long-run social consequences of population programs and policies. Randomized allocations of program and policy treatments can be especially informative in this field, but may not provide a general basis to forecast policy effects. Experimental studies cannot be extrapolated outside the range of observed samples, and program designs and technologies change over time and across societies. For programs to be extended to new populations, the sources of heterogeneous response to the program treatments become a key issue. Additional methods for policy evaluation may also be useful for analyzing naturally occurring “quasi-experiments,” which tend to impose restrictions on how behavioral and technological relationships occur, yet are necessary to recover estimates of the effects of policy treatments. These combined approaches, experimental and nonexperimental, may reveal the likely effects of population policy on mortality, morbidity, disability, labor productivity, labor supply, and fertility, as well as the life-cycle accumulation of physical and human capital that enhance private well-being, generate significant social externalities, operate as public goods, and spur economic growth. However, this area of population policy research is complicated and cannot yet deliver confident answers to settle many of these central questions confronting policy makers. Many empirical and policy questions related to fertility, women’s human capital, and child quality are addressed in a previous paper (Schultz, 2008a), and therefore the current chapter focuses first and more thoroughly on current health issues.

The outline of the chapter is as follows. Section 2 surveys the historical changes in demographic rates. Section 3 reviews some stylized facts about health and fertility, and economic development. Section 4 outlines a framework for studying health determinants and consequences. Section 5 surveys the macroeconomic evidence on health and growth, which has relied largely on cross-country comparisons, whereas Section 6 surveys the microeconomic evidence on health and development, where households and communities are the units of

observation, and the primary goals are to estimate health production functions and the productive labor returns to health human capital. [Section 7](#) turns to fertility, and focuses on the macroeconomic evidence of the consequences of fertility change and specifically its effect on economic growth, whereas [Section 8](#) reviews briefly the microliterature on the determinants of fertility decline and the effects of policy-induced voluntary declines in fertility on the welfare of women, their families and communities that might be attributed to effective family planning and reproductive health programs. [Section 9](#) notes the connections between the demographic transition, development, and internal migration, and the problems it raises for policy evaluation studies. [Section 10](#) concludes.

2. HISTORICAL TRANSITION OF DEATH AND BIRTH RATES AND POPULATION POLICIES

The demographic transition occurred first in what are today high-income countries, when age-specific death rates started to decline gradually after about 1750, whereas sustained mortality declines in low-income countries may have only started in the 1920s, but proceeded much more rapidly after the Second World War. Consequently, there has been a catch up by the low-income countries, or a convergence in life span after 1930 which emerges more clearly after 1950, and persisted until the 1990s. The decline and convergence in fertility has tended to follow that of child mortality.

There are differences between the age-specific pattern of mortality decline experienced in various historical periods in high- and low-income countries. In the early-industrializing countries, the initial declines in mortality from 1750 to 1875 may have favored slightly adults. Only toward the end of the nineteenth century did the heavy mortality among infants and young children begin to fall rapidly. Where the early agricultural and industrial revolutions attracted a growing fraction of the population to work in the increasingly unhealthy cities, the economic growth associated with urbanization did not immediately lead to increased life spans, at least not in England, Scotland, or the United States ([Tanner, 1982](#)). By the end of the nineteenth century, it is widely believed that improvements in urban public health infrastructure was responsible for mitigating the urban health problems associated with rising population densities, specifically through public investments and regulation of sanitation and water supplies, while the growing

acceptance of the germ theory of disease provided individuals and institutions with a motivation to adopt more hygienic practices that could control the spread of communicable infectious diseases. Many factors are attributed a role in the salient declines in infant and childhood mortality rates, including the rising economic standard of living, and in particular the declining price of food relative to wages, the increasing quality and diversity of food in urban markets, the decreasing crowding of urban worker housing, and extending of welfare systems designed to provide a safety net for food and medical care for the poor, disabled, vulnerable widows, women with children to support, and the elderly. Although advances in medical knowledge and public health interventions were probably unimportant in reducing mortality before the beginning of the twentieth century, the impact of medical science became notable after the 1930s (Fogel, 2004; McKeown, 1976, 1979). Until the 1960s, most lives saved by these improvements in health technology, income, and the living environment were those of children, and only thereafter in the industrially advanced countries does the reduction in deaths among older persons, over age 50, add more to the population than the reduction in deaths among children.

As indicated above, a second phase in mortality decline in the early-industrializing countries occurred from roughly 1875 to 1960, as the proportion of children surviving birth and childhood diseases increased substantially, and these improvements in child health conditions from communicable diseases was often associated with a lag to the decline in fertility. Observers hypothesized that fertility was in part responding to the increase in child survival in a homeostatic manner controlling population growth (e.g., Freedman, 1975). Ultimately, fertility fell from roughly 6 to little more than 2 children per woman, more than enough to offset the effect of the decline in child mortality on population growth, and the number of surviving children per woman declined from approximately 5 to 2 as the rate of natural population growth subsided in a few generations to approximately zero by the end of the twentieth century in Europe, Japan, and areas settled by Europeans.

The post-Second World War baby boom in some countries could be in part attributed to shifts in the timing of fertility, births delayed during the Great Depression and the Second World War occurred during the postwar economic expansion. Completed fertility for a sequence of births cohorts fell gradually toward replacement levels of about two children per woman in most developed countries. Differentials in fertility across developed countries and within these countries by region and socioeconomic classes gradually narrowed. Life expectancy at birth and

lifetime fertility have become more homogeneous across and within more developed countries by the end of the twentieth century.

Infectious diseases were first controlled by isolating the sick (i.e., quarantine), and then by the use of preventive health measures and changing personal hygiene to limit the transmission of disease, and eventually by the development and application of sulfa and antibiotic drugs, whose use to control infectious and parasitic diseases was accelerated by the involvement of the armed forces in health research during the Second World War. After the war, new public health technologies diffused rapidly to the rest of the world. By the 1960s, medical science increasingly grappled with the noncommunicable chronic diseases, slowly reducing death and disability due to cardiovascular disease, respiratory diseases, some forms of cancer. The reduction in fertility has also been associated with a number of other changes in coordinated family life-cycle behavior, including increased female schooling (relative to male), increased female participation in the market labor force, increased human capital investments in children's schooling, and perhaps increased physical savings and life-cycle accumulation of wealth. With the declines in fertility and continuing declines in mortality rates among the elderly, national populations have become older, first in the high-income countries, and now emerging as an issue in a growing number of low-income countries where birth rates first started to fall in the 1960s and 1970s.

Beyond Europe, Japan and countries settled by Europeans the secular declines in mortality occurred later but evolved thereafter more rapidly, though incomplete registration of death rates often leaves in doubt the precise timing of these changes, how cause-specific deaths evolved, and the economic, social, and institutional developments that governed this epidemiological transition. The sustained slow declines in mortality in low-income countries begins to be evident after the flue epidemic of 1918-1919, and probably accelerated in the late 1940s as public health measures were coordinated to control infectious and parasitic diseases by WHO, and supported by technical assistance programs, such as Truman's "Point Four program for economically undeveloped countries." These declines in mortality in low-income countries initially benefited largely infants and children, leading to more rapid population growth and more youthful populations in the 1950s, without clearly associated improvements in labor productivity or growth in national income (Acemoglu & Johnson, 2007). By comparison, the early gradual declines in mortality in Western Europe seem to have been related to the initial agricultural and

industrial revolutions, which raised wages relative to the price of food, improved nutrition, adult health, and probably increased labor productivity (Fogel, 2004).

If the increase in life expectancy is accepted as a summary measure of survival and population health, this indicator of life span has been increasing in the countries with the longest life span and most reliable mortality statistics by about a quarter of a year with the passage of each calendar year. This trend can be traced backwards for almost 200 years (Oeppen & Vaupel, 2002). In low-income countries life expectancy at birth has been increasing at twice this rate, or about a half year per calendar year, from 1940 to 1990, after which this pattern of convergence in life span between high- and low-income countries has been interrupted by the HIV/AIDS epidemic in sub-Saharan Africa (Strauss & Thomas, 2008).

Longer-lived parents may need more support in their old age, and since they have chosen to have fewer surviving children to provide such support, this development may lead to an increase in life-cycle savings in the form of physical or financial assets, or to increased pension systems and retirement funds managed by governments or offered through employers. Since there is little exogenous variation across individuals within countries in public policies affecting the tax-transfer incentives to work and save, microestimates from individual data of how the changing retirement program incentives affect labor supply behavior, or crowd out private savings by workers, is not resolved. Comparisons of tax-transfer incentives across a handful high-income country suggest high taxes on the earnings of elderly and generous pensions could contribute to the decline in elderly labor force participation, or in other words the reduced age of retirement despite the increase in health, adding to the fiscal burden of most public pension and health care programs (Gruber & Wise, 1995, 2004).

Becker and Barro (1988) postulate that more generous retirement pensions are responsible for the decline in fertility in more developed countries in the twentieth century. But this correlation could also signal that populations that want fewer children also demand pensions from their governments. In either case, funding public pension system from taxes on the earnings of workers has often led to “pay as you go” systems (i.e., Ponzi scheme), in which the earnings of younger generations of workers are taxed to support part of the pensions of the elderly. This is one reason advanced for nations to subsidize childbearing and investment of public resources in the health and schooling of children, in order to increase the size and productivity of future generations, and thereby reduce the future tax rate on labor required to finance “pay as you go”

old-age pensions (Gruber & Wise, 2004). A reduction in the tax rate on labor is often assumed to encourage more work, effort, and savings among the young, but the empirical documentation of the magnitude of these behavioral responses remains uncertain in high- or low-income countries. In low-income countries, population policies are associated with subsidies for birth control and health. International development assistance in the population field has recently increased, predominantly for health, and much of this chapter deals with research on health and its implications for development policy, and fertility is discussed later and in another review (Schultz, 2008a).

2.1 International population policy assistance: Levels and trends

According to UNFPA/NIDA (2009) resource flows project, the funding of population assistance activities by the OECD countries in family planning, reproductive health, and other health activities in less developed countries has increased in the last 11 years from \$1883 million (1993 US dollars) in 1996 to \$5290 million in 2006. Population assistance as a percent of total official development assistance increased from 2.5% to 6.1% in this period. The functional composition, geographic focus, and institutional delivery system have all changed appreciably. In 1996, 37% of population assistance was allocated to family planning services, which increased to 43% in 1998, and thereafter declined to 5% in 2006, leaving donor real expenditures on family planning at about a half the level they were in 1996. Reproductive health services as a share of population assistance have gradually declined from 33% in 1996 to 20% in 2006, yet they more than doubled in 1993 dollars. Research, program evaluation, and data collection, such as the demographic health surveys (DHS), have declined from 14% of the total to 5%, holding approximately constant in real dollar terms. The remainder of population assistance is devoted to the prevention and treatment of sexually transmitted diseases and HIV/AIDS and presumably other unspecified diseases, and it increased as a share of population assistance from 16% to 70%, growing 15-fold in 1993 dollars. Sub-Saharan Africa received 28% of population assistance in 1996, and its share increased to 35% by 2006, whereas Asia and the Pacific received 24% in 1996 and 14% by 2006, and Latin America and the Caribbean received 13% in 1996 and 5% in 2006, with the regional remainder to Western Asia and North Africa, Eastern and Southern

Europe, while the global/interregional share increased from 26% to 40% of population assistance. NGOs were the largest channel of distribution of population assistance in 1996, whereas the importance of bilateral and multilateral organizations was the dominant source of funding by 2006 (<http://www.resourceflows.org/index.php/articles/288>, Tables 1-6; 3 March 2009).

Comparable figures for expenditures on health and family planning activities by the governments in less developed countries and by private households and local NGOs in these countries are not readily available to complete the accounting of social resources allocated to these population activities. WHO and World Bank report data for total public (government) and private (household) expenditures on health, some of which are reported in [Table 1](#) for 1990 and 2000. There is much variation across countries in the fraction of GDP allocated to health, and the share of all health expenditures in the public (vs the private) sector differs markedly across countries at all levels of development, for example, India, China, USA, and Norway. It would be informative to disaggregate public expenditures on health among public health (preventive) services, out patient care, and hospital (curative) services, as reported in the IMF *Government Financial Statistics*. But these annual figures from IMF generally omit state and local public health programs, and even for the central government expenditures these distinctions are missing for many countries. To account for private expenditures on health, a comprehensive representative household survey including consumption and expenditure data is required, such as the living standard measurement surveys of the World Bank (<http://www.worldbank.org/LSMS/>). But these surveys are only available for an unrepresentative subset of countries and years. Out of pocket private expenditures on health as estimated by WHO (2003) excludes health payments to enterprises which deliver medical benefits to their employees, insurance premiums, or other prepayments and reimbursements to third-party payers. Moreover, it is rarely possible to allocate private or public health expenditures according to function, for example, family planning versus reproductive health, or for specific diseases. Finally, no estimates were found of the value of consumer time spent obtaining public and private health services, or the opportunity cost of the time of household member who care for their own sick ([UNFPA/NIDA, 2009](#)).

Another perspective on trends and differentials in population policies may be inferred from what governments indicate are their attitudes and priorities. The [United Nations \(2003\)](#) collected

questionnaires from governments on population policies and their responses are summarized in [Table 2](#) by a country's level of development and over time. Virtually all least and less developed (LLD) countries view mortality rates as unacceptably high, as they do infant, child, and maternal mortality (not reported here), whereas this attitude toward mortality is more muted among the more developed (MD) countries. The majority of LLD countries have in the last decade sought to lower their fertility, while 44% of the MD countries now consider raising their fertility as beneficial. Policies that support directly or indirectly access to contraceptive methods are approved by 95% of the LLD countries by 2004, a marked increase over the last 25 years, and by 86% of the MD countries, despite their public preference for higher fertility. The timing of fertility is also of growing concern to governments. The high level of adolescent fertility is a worry in more than 60% of the LLD countries by 2004, and in a third of the MD countries. A final aspect of population policy involves international migration, though it is not pursued in this chapter. From the UN survey it appears that a growing share of countries at all levels of development, constituting a quarter by 2004, favor policies that reduce emigration and immigration, and only 2% of MD countries favor policies to raise emigration and immigration.

Although a consensus has been reached among LLD countries in the last quarter century in favor of government support of contraceptives and efforts to reduce fertility, MD countries with low or negative rates of population growth have begun to favor policies to raise fertility. The UN survey makes no attempt to assess the willingness of the MD countries to underwrite pronatalist incentives to avoid population decline or mitigate the previously noted fiscal consequences of population aging. However, the inclination to favor higher fertility in MD countries has not reduced public support for voluntary contraception, or the preference for lower adolescent fertility, or increased support of immigration. Improved health and reduced mortality is valued universally, though the financial priority assigned to improved health appears to differ by levels of development. Fertility control in terms of contraception is widely valued as a public good. The social externalities of reducing fertility in low-income countries may be valued positively, whereas the positive social externalities of increasing fertility in MD countries are beginning to be appreciated. There is no consensus among economists on how to evaluate the magnitude of population externalities, or how much people are privately willing to pay to reduce mortality, or change fertility, or modify future rates of population growth through their provision of services and design of related tax-transfer policies. These should be active areas of research.

3. STYLIZED FACTS RELATED TO HEALTH, FERTILITY, AND DEVELOPMENT

When the registration of deaths by age and sex is relatively complete, and the underlying population is accurately enumerated by periodic censuses, demographers can assemble a “life table” to describe the pattern of mortality. This table is often summarized in terms of the average duration of life for a cohort born today that is assumed to experience this year’s age-specific death rates over their hypothetical lifetime. It is based on the concept of a “synthetic” cohort for whom the current age-specific death rates persist indefinitely into the future, not the outcome experienced by any real cohort of births living out their lives. Gradually after the Second World War an increasing number of such life tables have been estimated for various populations and periods. At about the same time a consensus developed on how to best calculate national income. This led to the discovery that the current era of “modern economic growth” had sustained growth in *per capita* real output of 1-3% per year over a century or longer, which qualified as a unique historical period (Kuznets, 1966). Demographic evidence in more developed countries, as noted above, suggested that the expectation of life at birth was also increasing over time after the start of the nineteenth century by a quarter of a year per year (Oeppen & Vaupel, 2002). Both indicators of *per capita* national income and of life span are measured with error and may not represent the ideal concepts of economic well-being or health status. The long-run advancement in both income and health at the aggregate level of the nation has nonetheless become an accepted empirical regularity. Although income and consumption measures from household sample surveys provide an auxiliary microestimate of *per capita* income and consumption to compare to national income estimates, there is no analogous measure of health status at the individual, family, or even community level to compare with life expectancy calculated for a nation. Nor, as I argue later, is it clear that life expectancy at birth is conceptually a good summary measure of the health objective of society or a satisfactory indicator of the productive returns associated with secular improvements in health.

For many countries with incomplete death registrations, methods have been developed for estimating indirectly life tables. First, the age composition of the population from a census provides some information on historic trends in birth rates and age-specific death rates, and repeated censuses set limits on the likely range of vital rates consistent with intercensus

population growth rates and age compositions (United Nations, 1967; Coale, 1972). Second, from the collection of life tables from higher-income countries, for which the population and mortality data are thought to be relatively accurate, similar life tables are clustered into regional model classifications, possibly influenced by distinctive diseases and resulting regimes of mortality prevalent in these geographically defined areas. Once a country is assigned to a regional class of life tables, the level of mortality within that class can be approximated in that country on the basis of estimated child mortality rates, derived generally from the responses of women to a census or representative survey question regarding the number of children they have born and number of their children surviving (Brass, 1975; Coale & Demeny, 1966). Imputations of life tables for less developed countries based on these indirect methods have been promulgated by the Population Division of the United Nations and the World Bank, and they are essentially extrapolations from countries with more reliable data sources.

National income accounts and household surveys periodically tell inconsistent tales regarding the occurrence of economic growth, but at least for income and the prevalence of poverty there are generally two empirical approaches to compare (Deaton, 2003). Health status, morbidity, and mortality are not yet subject to systematic comparisons based on macro- and microindicators, although height and weight are increasingly considered as providing critical information on nutrition, health status, and well-being at various levels of aggregation over time. Finally, to facilitate comparisons across countries, these national income estimates measured in terms of local currencies are revalued at common international prices. Early studies relied on official foreign exchange (FX) rates to convert local currency income into common dollar units of purchasing power, but this FX approach excluded cross-country variation in the prices for untraded goods and services and their share in income. Purchasing power parity (PPP) price indices were therefore constructed for a few countries at various levels of development where coordinated price surveys were conducted, and then these implied domestic price exchange rates for traded and untraded goods and services were extrapolated to the majority of countries where such comparable price surveys did not yet exist (Summers & Heston, 1991).

Figure 1 illustrates the problems in interpreting the relationship between health and development. The association between health status (H) and income per adult (Y) is positive. This may happen because increased income leads people to (1) demand directly better health as a consumer good adding utility per year lived and possibly increasing longevity, or (2) people's

increased standard of living shifts consumption patterns which on balance contribute indirectly to health improvements, for example, suppose decreased malnutrition and reduced crowding of housing improved health more than increased smoking and drinking reduced health.

Alternatively, improved health status can raise the productivity of people that adds to their market income, which sums into increased national income, unless (1) their market labor supply decreases by a larger percentage than (2) their wage opportunities increase over their lifetime.¹

These national estimates of income and mortality, summarized generally by variations across countries in terms of market income (GDP) and life expectancy, are analyzed in most macroeconomic studies discussed in [Section 5](#). Household surveys provide the data for microeconomic studies on how, on the one hand, exogenous variation in income (due to changes in Z in [Figure 1](#)) increases inputs for the production of health (H), and how on the other hand, the exogenous improvements in health status (due to changes in X_2) enhance the economic output of workers (W) and probably boost market income and certainly increase “full” income. These empirical relationships may tell us how an increase in income from an exogenous improvement in a country’s terms of trade (Z) contributes to the purchase of more food or better living conditions, to the adoption of healthier behaviors over the life cycle, or to the improved access to medical care and more effective health technologies, which in combination produce a longer life span. It may also tell us how global improvements in shared health technology, X_2 ([Acemoglu & Johnson, 2006](#)), or public health expenditures on HIV drugs provided by a philanthropic foundation reduce the consequences of disease and disability, on worker earnings, and aggregate income. Both possible causal relationships are thought to be positive, and may operate therefore to reinforce each other. The observed gross relationship between life span and income might overstate the causal effect operating in either direction. The choice of information that could identify the causal effects operating in one direction, or the other, is controversial. Without the assessment of these causal effects that describe specific pathways, however, it is difficult to evaluate whether health-related inputs foster development as well as health, or how population policies are designed to internalize social externalities associated with the production of health or the modification of fertility.

4. FRAMEWORK FOR STUDYING THE DETERMINANTS AND CONSEQUENCES OF HEALTH

One of the first descriptions of the empirical pattern between health and development is by Preston (1975), who represented health by the expectation of life at birth, and development by *per capita* national income in 1962 dollars (compared by foreign exchange rates). He reports estimates from life tables and national income for 10 countries in the 1900s, and for 38 countries in the 1930s, and for 57 countries in the 1960s.² His observations are plotted in Figure 2. If income is expressed in logarithmic (proportional) terms, the semi-log-linear fit or R^2 is relatively high, 0.885 in 1930s and 0.880 in 1960s. His paper explores the upward shift in the relationship over time in the twentieth century. Had the fitted function remained as it was in the 1930s, the gain in life expectancy would have been substantially smaller than was actually observed in 1960s, suggesting some other factors have also changed, whose inclusion would help to explain the increase in life expectancy, such as advances in the technical knowledge of how to improve health. The residual gains between 1930 and 1960 are disproportionately larger for lower-income countries. Countries with incomes between \$100 and \$500 “experienced” increases in life expectancy of 10-12 years between 1930 and 1960, whereas high-income countries achieved more modest increases, and the upper asymptote approached at very high-income levels increased by less than 5 years.³

Strauss and Thomas (2008) update these patterns by adding World Bank estimates from 2004 in Figure 3. The fitted lines are nonparametric flexible approximations with income now expressed in logarithmic terms.⁴ The increases in life expectancy controlling for income from 1930 to 1960 among countries with \$200 to \$500 dollars of income *per capita* (1995 dollars) is evident, but the gains from 1960 to 2004 are largest for the middle income countries, between \$500 and \$2000 dollars *per capita*. Even without forcing an upper asymptote to the life expectancy pattern as implied by Preston’s exponential function, it is clear that most countries with incomes greater than \$1000 have life expectancy in 2004 in a relatively narrow range between 70 and 80 years. The outliers in 2004 are those with exceptionally low life expectancy, which are named in Figure 3 (Strauss & Thomas, 2008), and are those countries where HIV/AIDS has raised adult mortality to exceptional levels in sub-Saharan Africa, disrupting the past empirical pattern between health and development.⁵

4.1 Estimating without bias health production functions with endogenous inputs

What type of information would help to disentangle from the simple association between health and development the underlying causal links and specific pathways between these variables that could be relevant to the choice of policies that contributes effectively to improvements in health or economic development or both? To identify these causal effects, a variable must be known that affects either health (H) or income (Y) in Figure 1, but does not directly affect the other outcome. The causal effect of private behavior and public institutions and conditions on the production of health can only be evaluated without bias under special circumstances. Typical measures of the association between the use of health inputs (e.g., consumption of drugs) and health outcomes (e.g., less disabled or living longer) have little information value to the policy maker, because the choice of using the health input may be associated with other unobserved health factors and other intercorrelated omitted personal choices whose health effects are likely to otherwise be attributed to the observed health inputs.

To establish a framework for discussing this problem, assume individuals value good health (H) and other consumption (C):

$$U = U(H, C). \quad (1)$$

An individual's current stock of health human capital is assumed to be produced by a function of the prior period's health endowment (e), current health-related private inputs (I), and exogenous environmental disease conditions (D). Over time D might be modified by cooperative or public health investments and infrastructure, such as water, sanitation, and community disease control programs, but is initially taken as exogenous by the individual or household:

$$H_{ijct} = H(e_{it-1}, I_{ijct}, D_{ct}), \quad (2)$$

where the subscripts i denote the individual, j the household or family, c the community, and t the time period. Because the health-related inputs are expected to respond to prior health endowments (e) that are difficult for researchers to measure comprehensively or introduce into the analysis as exogenous control variables, or local health conditions (D), identifying variables or exclusion restrictions must be observed that cause variation in the demand for health-related

inputs that can be assumed unrelated to variation in e or D . The health input demand functions could be portrayed in the following form:

$$I_{ijct} = I(e_{it-1}, X_{ct}, Y_{jt}, D_{ct}; S_{jt}, T_t), \quad (3)$$

where X_{ct} denotes access to and the prices and quality of health inputs that vary by community, Y_{jt} the household's income or lifetime resources, S_{jt} the schooling of those in the household who manage the production of health, such as a mother for her child, and T_t the prevailing medical technology or knowledge of public health that could change with time and customs. These final four variables might be imagined to vary independently of the initial health endowment, e , and public health environment, D , and provide identifying exogenous variation in health input use, required for estimating the causal effect of health input use on the production of health (Eq. 2). For example, suppose a public health program subsidizes the price of a health input at time t in a random subset of the communities c^* , and the subsidy increases the local use of health inputs in these treated communities. Alternatively, a seemingly random exposure across farm communities to crop pests in period t might reduce current household income, Y_{jt} , and thereby impact the use of health-related inputs, and not otherwise affect health conditions.

As discussed later in Section 5, parameters of the health production function might be recovered from panel observations on population aggregates, such as countries. Factors external to a country may presumably affect the value of a nation's resource endowments, which impacts the income of an average household, but does not otherwise affect the inducements or capacities to invest in health (Pritchett & Summers, 1996). Finally, shifts in locally effective medical and public health technology (T) may also occur from time to time due to research and development, affecting health differentially in different locations depending on the initial prevalence of the specific diseases controlled by the technological change, disseminated under a variety of arrangements involving trade, licensing, shared property rights, or international grant assistance (e.g., Acemoglu & Johnson, 2007).

It should be clear that these types of identifying exclusion restrictions to recover the health production function parameters are likely to be controversial. This is why randomized trials for medical interventions are often supported by the public, despite their complex ethical ramifications, to confirm the effectiveness of promising health inputs and associated

technologies when health delivery systems and living conditions differ greatly (e.g., [Thomas et al., 2006](#)).

4.2 Estimating the marginal product of health human capital

To allocate efficiently health and social resources, a policy maker must understand a second health relationship: How do improvements in reproducible aspects of health influence the potential productivity of individuals and improve their well-being? One approach to evaluating how health outcomes alter the productive potential of society is to estimate an earnings function conditional on measures of human capital including population health. Although this may be undertaken at the aggregate level of a country or health administrative region, most work in this area builds on the empirical study of earnings function estimated at the individual level, following the research on schooling by [Card \(1999\)](#), [Griliches \(1977, 1979\)](#), and [Mincer \(1974\)](#), in which an approximation for health stocks is included with schooling as a determinant of wages or labor productivity ([Schultz, 2003, 2005](#)).

The logarithm of the productivity of labor or the wage rate ($\ln W$) is expected to be greater for a healthier worker (H), as well as for one with more schooling (S), and ability (a), and the supply of other productive factors, such as land and capital per worker (K) that may complement labor and increase its marginal product. Productive technology (T) may shift over time, and it has been hypothesized that recent global improvements in technology have disproportionately increased the productivity of better educated workers, and technological change could also differentially affect the productivity of workers according to their health status or disabilities:

$$\ln W = W(H, S, a; K, T), \quad (4)$$

where local prices of health inputs, X , or the community health environment, D , could possibly identify estimates of the effect of exogenous variation in H on the wage, just as the distance to and quality of local schools could serve as instruments (prices) for schooling, S . The positive correlation between ability (a) and schooling (S) is expected to bias up (positively) the estimated effect of schooling on wages, if ability is omitted from the estimated wage function, and both ability and schooling add to the productivity of workers ([Card, 1999](#); [Duflo, 2001](#); [Griliches, 1977, 1979](#); [Schultz, 1988a](#)). Empirically, however, this source of bias does not appear to be a severe problem for policy inferences, which focus on the measurement of the private wage

returns to years of schooling, such as estimates based on educational reform instruments for schooling or estimates based on between twin differences in schooling and earnings. This lack of systematic or apparently substantial omitted ability bias may be due to the measurement error associated with schooling, which would in the classical case of random errors introduce an offsetting downward bias in the ordinary least squares (OLS) estimate of the wage return to schooling in the estimation of the wage function (Eq. 4).

There is less empirical research on the returns to aspects of health in a wage function than to schooling, and this issue is complicated by the multifaceted form of health and the greater difficulty of measuring health than schooling for individuals in a general survey. Among the studies that have explored the issue of health consequences on productivity, health is first approximated by body mass index (BMI) and local food prices were used as instruments (Deolalikar, 1988; Strauss, 1986) followed by studies also using disability days, height, menarche (Savedoff & Schultz, 2001; Schultz, 2002, 2003; Schultz & Tansel, 1997; Strauss & Thomas, 2008).⁶ Among the studies I know that estimate wage returns to indicators of individual health, there are few which find the OLS return estimates are positively larger than the instrumental variable (IV) estimates that treat health as endogenous or measured with error, identified by health conditions and services at birthplace or in their family. Thus, the endogeneity of these adult health indicators do not appear to introduce a dominant upward bias in the OLS estimated wage returns to these health outcomes.

4.3 Parent investment in children in response to their children's initial endowments

If parents' investment in their children's human capital responds to the child's initial endowments at birth, either in the form of health, e , or ability, a , the OLS estimates of the child's wage returns to adult health or schooling would be biased by the unobserved investment behavior of parents. For example, assume that parents invest more in their less healthy and less able children, perhaps because they are motivated to equalize their children's life-cycle consumption opportunities or compensate those children who initially exhibit low endowments (Griliches, 1979, Table 1, p. S61). Then, the omission of these early endowments in estimating the wage function (Eq. 4) leads OLS to underestimate the wage returns to the observed health

inputs and indicators of schooling attainment. Griliches in his early review of the literature estimating wage returns to schooling within families and between twins concludes returns to schooling tend to be lower when estimated based on the variation between siblings, than when estimated across families. The coefficients on IQ, approximating ability, are also reduced when wage functions are estimated within families (i.e., with family fixed effects) rather than across families. Griliches interprets this empirical pattern to suggest that family allocation of schooling investments among offspring and twins is reducing inequality within families, consistent with compensatory allocation of human capital by parents across their children according to their initial endowments.

Becker and Tomes (1976) suggest that wage returns to schooling are greater for more able children. If parents then maximize the earnings of the sum of all dynasty members, they would concentrate their human capital investments on their most able children. If it were costless to then reallocate the dynasty's total earnings among members, parents could subsequently accomplish the distribution of consumption among family members they wanted, without sacrificing their initial objective of efficiently allocating their human capital investment. Given Becker-Tomes assumptions regarding parent reinforcing investments in the better endowed children, the OLS estimated wage function (Eq. 4) would tend to overstate the returns to human capital, S and H , unless the researcher could also control for the child's initial endowments. Estimating returns within families, by including family fixed effects, or by analyzing differences between twins. These within family return estimates would be purged of bias due to other confounding factors that do not vary within families or twins, respectively. The direction of the bias due to parent child investment response will thus depends on whether parents compensate or reinforce the exogenous initial endowments they observe in their offspring. In this regard, a natural initial endowment of the child to study is birth weight.

Health endowments at birth, such as birth weight and gestation, may be influenced by parents, however, by their prenatal behavior, such as maternal nutrition, the timing and form of prenatal care, smoking, and the number, timing and spacing of births (Mwabu, 2009; Rosenzweig & Schultz, 1983), all of which could be affected by parent heterogeneous preferences toward child health and might persistently influence later family resource allocations among children, making these measures of endowments at birth endogenous to the family's reproduction, life-cycle behavior, and subsequent child investments. Behrman, Rosenzweig, and

Taubman (1994) find evidence that family allocation of schooling reinforces the preschool endowments of children, and add to the final inequality in their earnings, through their analysis of fraternal and identical twins from the Minnesota Twin Registry. One way to identify exogenous variation in endowments birth, approximated by fetal growth rate, is to examine differences in birth weight between identical (homozygotic) twins that cannot be affected by parents (Behrman & Rosenzweig, 2004; cf. Figure 4). Identical female twins from the Minnesota Twin Registry ($n = 804$) are analyzed, and the effect of birth weight per week of gestation (i.e., fetal growth rate) is estimated on the child's later outcomes, such as adult height, completed years of schooling, and adult wages. This within twin estimate holds constant any effect of different genetics on ability (a), sex, gestation, maternal health, and family fixed effects from the time of birth. These within twin estimates of the effect of fetal growth rate on later child schooling are 70% larger than OLS estimates across twins, and within twin effects of fetal growth on wages are nearly five times as large as the OLS estimates, whereas the estimated effect of fetal growth rate on adult height remains essentially unchanged from the OLS estimate, and that on adult BMI ceases to be significant within twins. The increase in the effect of birth weight on schooling and wages within identical twins suggests parents are reinforcing the exogenous variation in fetal growth rate at the time of birth by their subsequent resource allocations that enhance schooling and adult productivity. The stability of the fetal growth effect on height within and across twins suggests that parents do not affect adult height through their child investment response to their child's initial endowment. Alternatively, adult height may be largely determined by genetics and conditions *in utero* and is not strongly affected by parent child investments between twins, possibly due to postnatal nutrition and exposure to early childhood diseases. Behrman and Rosenzweig adjust their estimates of birth endowments on schooling and productivity for the diminishing returns to fetal growth rate (nonlinear health-productivity relationship), incorporating the fact that the distribution of fetal growth rates for twins are lower than for singleton births. Consequently, the impact of raising the average birth weight for all births is likely to have a smaller effect than they estimated within the sample of twins who are on average low birth weight.

The objective of Rosenzweig and Zhang (2009) is to estimate from a sample of Chinese twins, the consequences of changing exogenously family size on child quality. To recreate the effect of increased family size for parents who have predominantly singleton births (99% of the

Chinese births), they control for the fact that twins have on average a lower birth endowment (i.e., birth weight) than do singleton births, and the (closer) spacing of twins may also affect the relative cost to parents of investments in the human capital of their children, because the time parents have for child investments is limited, and parent credit constraints could raise the cost of borrowing to invest in both twins at the same time. They show that the birth order of the twins allows them to identify within bounds the tradeoff between increasing family size due to the exogenous occurrence of twins increasing family size, and the effect of twins reducing child quality compared with other family births. Their analysis is based on a sample of twins and children of similar ages drawn from the 2000 Census of China from the Kunming district of Yunnan Province ($n = 1169$). They formulate a model for the biological and behavioral components involving parent response to twins that could bias direct estimates of the effect of child quantity on child quality associated with the occurrence of twins, assuming as [Becker and Lewis \(1974\)](#) do that child quantity and quality are substitutes for parents.⁷ Child quality might be represented by either schooling or health. Rosenzweig and Zhang (2009) consult several indicators of schooling—expected college enrollment, years completed, and standardized math and language exams—and health—subjective good health, weight, height, and BMI.

Evaluating the magnitude of this cross-effect of family size due to twinning on child quality is potentially important for population policies regarding fertility and health, because if family planning or reproductive health and education programs encourage woman to reduce voluntarily their fertility, the consequence of these program-induced declines in fertility would raise child quality and this could be viewed by a policy maker as a positive social externality of the program, potentially justifying public subsidies and the burden of taxes needed to finance publically such subsidies ([Schultz, 2008a](#)). First, twins are biologically lighter at birth, have shorter gestations, and lower APGAR scores. There is also evidence that twins experience lower survival rates, fewer years of school attainment, and at least for males, lower cognitive achievements and wages in later life ([Almond, Chay, & Lee, 2005](#); [Black et al., 2005](#); [Black, Devereux, & Salvanes, 2007](#); [Royer, 2009](#); [Cesur & Rashad, 2008](#)). But these subsequent measures of child “quality” could be due to both the lower endowments of twins at birth, and the effects of differential investments by parents after birth, which could reinforce (or compensate) for twin lower birth endowments, and thereby strengthen the negative quantity-quality negative effects of twins on child quality (or if compensating, reduce their magnitude).

Assume that parents reinforce the birth endowment effects between twins, as [Behrman and Rosenzweig \(2004\)](#) and [Behrman et al. \(1994\)](#) found in the US, and [Rosenzweig and Zhang \(2009\)](#) find in China. The effect of twinning on later child quality outcomes allows for twins to have lower endowments at birth than singleton births. If the twinning occurs at the second pregnancy the estimated effect of the twin on second birth children's quality provides an upper bound (more negative) estimate of the average negative effect of increasing family size. If the twinning effect occurs on the first pregnancy, the effects on later child quality provides a lower bound (less negative) estimate of the effect of family size on child quality. When the child quality equation is also conditioned on birth weight, as an approximation for initial birth endowment that parents observe, the range of these upper and lower bound estimates of the quantity-quality tradeoff should be narrowed, with reinforcing parent behavior according to their model ([Rosenzweig & Zhang, 2009](#)).⁸

A panel study of births from the US and UK of child-specific postnatal human capital investments by parents suggests that observed child inputs, for example, breastfeeding, respond inversely to the child's initial endowment measured by birth weight, holding constant for fixed mother effects (i.e., within families), but recognizing the endogeneity of birth weight and identifying its independent variation by the effects on birth weight of the mother's prenatal smoking and working in the labor force until less than 2 months before the birth ([Del Bono, Ermisch, & Francesconi, 2008](#)). This finding is consistent with [Griliches' \(1979\)](#) hypothesis that parent exhibit compensating behavior that reduces sibling inequality. But the smoking and labor supply behavior are also endogenous, and the birth weight outcome of earlier births might affect subsequent maternal behavior and thereby impact the difference in prenatal behavior between births. Further empirical study of the direction and magnitude of parent allocation of child investments in response to initial child endowment is a priority area for further research on the family and population policy, especially in low-income and high child mortality settings (cf. [Data, Gosh, & Sood, 2007](#)). Decisive studies will probably require panel data where the endogeneity of prenatal maternal behavior on initial child endowments are reasonably identified, and important postnatal investments in children, such as breastfeeding and the timing of immunizations, are observed by gender.

Complementarities between health and education inputs are also potentially an important feature of the process of child development ([Glewwe, 2005](#); [Glewwe & Jacoby, 1995](#); [Glewwe](#)

and Miguel, 2008). There may be different private as well as social externalities in the timing of these child investments that could be significant for setting priorities for social welfare and population policies. Diminishing returns to child investments in each time period seem plausible, and intertemporal specificity of the formation of different socioemotional skills and abilities suggests that social returns to compensatory interventions for some low endowments groups may be more effective at an early age (Cunha & Heckman, 2007). Economic returns to child investments may also differ significantly across socioeconomic strata, advantaged and disadvantaged classes, ethnic groups, geographic populations, and thus these programs could be strengthened and their overall effectiveness increased by appropriate targeting of the program to specific groups.

Disabilities among children are another endowment widely observed by parents at an early age that appear to occur at all economic levels in society, but are less often studied as a factor in parent investment behavior. In low-income countries, children identified with disabilities in household surveys are less likely to enroll in school, and complete fewer years of schooling when they do enroll. In 8 out of 12 recent DHS conducted in low-income countries, adults with disabilities are significantly more likely to reside in poorer households, with imputed poverty in the lowest two fifths of the population. But controlling for the lower educational attainment of the disabled member of the household, only 3 out of 12 surveys continue to find a significant partial association between disability and this measure of poverty (Filmer, 2008). This study suggests that disabilities among children increase their poverty as adults, primarily through reducing their schooling. The economic burdens associated with disabilities might be reduced if opportunities to improve schooling opportunities for the disabled were pursued. There are limitations in available data from developing countries on disability, verbal and nonverbal ability, noncognitive and social skills, and geographic lifetime migration and how these characteristics of children are themselves influenced by parents, families, schools, and other social institutions and policy. Research on these topics could have implications for population policies, but it will require both improved data and better analyses of the economic returns to improving the health, schooling, cognitive ability, noncognitive skills of youth and their determinants.

4.4 Spillovers from health human capital beyond the individual and family

The health human capital stock of workers could also raise the returns on capital, by reducing absenteeism, which could reduce the need for firms to have a redundant work force, and could even reduce household size if hiring casual replacement labor for sick family workers is less productive than relying on workers with more specialized experience in the respective firms and families. These externalities of health in the functioning of the economy are hypothesized to be important in those countries in which the HIV/AIDS epidemic is severe, but estimates of their magnitude in the aggregate economy are elusive and even estimates of the microeconomic consequences of health on worker productivity are rare and generally treat health as an exogenous shock and not as an accumulated family and individual investment (cf. [Thirumurthy, Zivin, & Goldstein, 2008](#)).

As already noted, there are few forcing variables that theoretically provide identification of the unanticipated effect of income on health, or the effect of exogenous shocks to health on labor productivity and income. This line of research in health economics does not have a comparable natural experiment as with twins affecting exogenously fertility and thereby family size. It is attractive, therefore, to find settings where social experiments can be implemented in which access to specific promising health treatments or subsidies for health-related inputs can be randomly offered to separated populations who are then followed over time. Survey data on these treatment and control populations should be considered as a basis for assessing the health consequences of welfare expenditures, and the short-run and long-run productive consequences of these public programs on individuals, families, and communities. Social experiments assessing health interventions are ethically complex. But neglecting the task of evaluating health program effectiveness is also difficult to defend, particularly when returns to health appear to be promising, but the uncertainty of these returns leads policy makers to discount the contribution of health to economic growth and treat health as merely a valuable consumer good ([World Bank, 2008](#)). Controlled field experiments have proven useful for testing medical procedures and drugs in order to maximize their benefits to society, while protecting the privacy of individuals and providing participants with an understandable basis on which they can decide whether they want

to participate in the social experiment and grant their informed consent (Duflo, Glennerster, & Kremer, 2008).

Health status reflects the accumulation over a lifetime of three types of factors. First, there is a genetic health endowment, which is assumed fixed after conception and is generally not affected by policy. Taking genetic variation into account may increase the effectiveness of policy interventions and influence the personal distribution of benefits from a given intervention. Second, after conception the individual's stock of health is modified by investment choices. The health care system as well as the family and individual makes choices that affect exposure to environmental risks, modify health-related behavior, and provide inputs relevant for the production of health, and possibly influence the formation of preferences, such as individual discount rates. These choices are called here investments in health human capital or health inputs, although they may satisfy other possibly conflicting consumption goals, such as smoking, drinking, and risky behavior. The third health component includes unpredictable stochastic factors associated with incidence of disease, climate variability, economic conditions, and epigenetic biological developments that are outside of the control of the individual or family or community institutions. Whatever indicators of health are studied, it is likely that many inputs to health will be omitted from an empirical analysis of health, and omitted variable bias is a serious limitation in estimating health production functions, if the omitted variables are correlated with those health inputs that are being empirically assessed (Mwabu, 2008; Rosenzweig & Schultz, 1983).

Measures of health human capital are, moreover, likely to be heterogeneous, by which I mean some part of variation in health indicators is innate or produced by genetics at the time of conception and affected by subsequent unpredictable shocks. Some part of health is produced systematically by the private and social investment choices, and I call this second component *reproducible health human capital*. The uncontrolled and reproducible components of health may be indistinguishable in a survey, because surveys tend to rely on indirect proxies for latent health status and conditions. But variation in these proxies for health may have different effects on worker productivity and well-being, depending on the source of variation in the proxy. Therefore, to evaluate the effects of health status for policy purposes, the productive effects of changing the *reproducible* component of health is relevant to most social policy choices. For evaluations of health policies, we are primarily interested in the production of health and

specifically the productive returns to the reproducible health human capital component of measured health that can be effectively increased by policies, and possibly targeted to disadvantaged individuals. To estimate the impact of reproducible health human capital on worker productivity, the effect of such heterogeneous indicators of health is estimated as though they are measured with error and are potentially endogenous. Consequently, instruments are needed for the observed stock of health that identify the health effects of health changes due to environmental sources of variation as would be affected by household investments in nutrition and health care and community health investments.

4.5 Phases of the mortality transition and implications for health inequality

The decline in mortality in high-income countries until about 1950, and in low-income countries from about 1940 to 2000, are attributable mainly to the first phase of control of infectious, communicable diseases. The related medical and public health technologies which accomplished this mortality revolution were relatively cheap to disseminate and were available to most basic community health care systems in the low-income world. Reduced malnutrition, combined with these medical interventions, achieved a sharp decline in infant and child mortality. The second phase of medical interventions is designed to deal with degenerative, noncommunicable, and chronic diseases which claim the lives mainly of prime aged adults and the elderly. It remains unclear whether effective low cost therapies for chronic diseases of the heart, lungs, cancer, arthritis, diabetes, etc., can be implemented in low-income countries or even provided to the poor in high-income countries without increases in public and private expenditures on health, or how effective they will be, if the fetal and childhood health conditions were disadvantaged. Drug therapies often involve large research and development costs, but low marginal costs due to large scale manufacturing. Some of these drugs have reached low-income countries because, as they lost patent protection, their prices as generic drugs have declined substantially. Diseases which are not prevalent in high-income countries do not receive much attention from researchers, because there is no high-income community of potential users willing to pay for the initial research and development costs. The diffusion of medical technology to reduce mortality from degenerative diseases among adults and the elderly may not equalize life span or health status

across countries in the future, as occurred dramatically in the first phase of the mortality transition when infectious diseases were controlled in childhood (Becker, Philipson, & Soares, 2005). Declines in adult mortality since 1955, increasingly due to control of chronic and degenerative diseases, such as heart, lung, cancers, or diabetes, have not noticeably reduced health inequality as measured by life expectancy at age 10, at least not among 21 high-income countries, or within these countries as can be measured between distinguishable race and ethnic groups (White, 2002).

Another approach to measure health inequality does not seek to distinguish differences between socioeconomic status groups, but relies on the unconditional frequency distribution of cohort survival, as reported in a national life table. The variance in expected lifetimes for the synthetic cohort is calculated in addition to the mean or median (Edwards & Tuljapurkar, 2005). The variance in life span for the cohort of survivors to age 10 has not shown a tendency to decline within high-income countries since 1960, though it does when the variances are calculated from birth. With less reliable adult age-specific registrations of death by cause in low-income countries, there are few studies of the causes of adult mortality declines in the low-income world, or who in the population has benefited most from the actual declines. And when health objectives are medically defined in terms of the incidence of specific categories disease, such as Malaria, TB, or HIV/AIDS, it is not surprising that knowledge about the determinants of death by cause is uncertain. Many deaths are due to multiple causes, making it even more difficult to infer how deaths from specific causes respond to policy interventions.⁹ Presumably many developing countries are reaching the stage today when further mortality reductions will depend on preventing adults from dying of chronic and degenerative illnesses and a handful of drug-resistant infectious diseases.¹⁰

5. MACROECONOMICS EVIDENCE ON HEALTH DETERMINANTS AND ECONOMIC GROWTH

The primary causes of death before the mid-twentieth century in more developed countries, and in most less developed countries until the end of the twentieth century, were infectious and parasitic diseases. Today deaths due to these diseases are geographically concentrated in a few tropical regions of the world with poor health infrastructure, extreme poverty, and other

conditions unfavorable to health. These geographic patterns of health and the prevalence of particular diseases, such as malaria, TB, and HIV/AIDS, have been used to account for economic development in a variety of ways. Poor health, short life span, and specific endemic diseases have been treated by economists as if they were exogenous determinants of the income levels and growth of income. Based on cross-country regressions, growth in income since 1960 is greater in countries for which life expectancy is greater. These partial correlations, however, may or may not remain significant statistically after controlling for other economic inputs to growth, such as the *per capita* supply of labor, human capital, and physical capital (e.g., Barro, 1997; Barro & Sala-I-Martin, 1995; Bloom, Canning, & Sevilla, 2004; Bloom & Sachs, 1998; Gallup & Sachs, 2001; WHO, 2001). But many of these economic inputs to growth are themselves endogenous to households and countries. If these intercountry comparisons are extended to interpret changes over time within countries (i.e., allowing for fixed country effects), the impact of invariant geographic and climatic indicators are, of course, then not identified. It is only reasonable to expect unobserved factors which are correlated with levels and changes in measured health conditions to also be related to income levels and income growth, for reasons not directly related to health, biasing upwards cross-country regression estimates of health effects on income and economic growth (Acemoglu & Johnson, 2007; Weil, 2007). It is widely concluded, therefore, that the prevalence of disease and indicators of health status used in cross-country studies are themselves produced by a combination of environmental and institutional conditions, as well as social and private behavioral responses to the conditions in these countries. The social scientist must understand and explain these health determinants if they are to clarify how these general health outcomes causally contribute to the economic development of countries, as illustrated earlier in the stylized Figure 1. Several studies have used country-level data to identify what are interpreted as causal estimates linking health and development.

Acemoglu, Johnson, and Robinson (2001, 2003) propose a dynamic framework involving a two-stage explanation for the contemporary relationship between development and disease environments. In the first stage, the health-disease environment impacts how colonial governments settle areas which in turn facilitate or impede the development of social institutions that favor long-term economic growth. European colonialists were more likely to settle permanently in temperate climates where their native agricultural crops and production practices could be transferred with the least investment in local adaptation, and where local diseases were

least lethal to the European immigrants, for example, North America, Argentina, Chile, and South Africa (Diamond, 1997). Agricultural settlers brought with them concepts of property rights and institutions to protect those rights and sought to replicate institutions of governance that corresponded roughly to those they had known in Europe. Conversely, where regimes confronted tropical agricultural climates and endemic diseases that were especially lethal for the European immigrants, the colonial regimes were more likely to resort to extractive industries, which exported precious metals, natural resources, or slaves, and depended frequently on restrictive governing arrangements to assure a reliable supply of labor in the export industries. These “extractive colonies” adopted institutions which did not promote property rights or evolve rules of governance which called forth investments that would increase total factor productivity and sustain modern economic growth. Acemoglu and his colleagues hypothesize that the distinctive institutions evolved by colonial regimes in response in part to their health conditions offer one explanation for the subsequent poor economic performance of colonial sub-Saharan Africa and some tropical areas of Latin America and Asia, if not the later collapse of Argentina. The contemporary efforts to control tropical diseases and improve health in areas such as Africa are not likely to achieve by themselves the economic benefits correlated today with better health outcomes (WHO, 2001), because the institutions, which are beneficial for sustained economic development, such as protection of private property and good governance, might be lacking for some time.

To quantify how health affects development, Acemoglu and Johnson (2007) assess how exogenous improvements in health directly affect growth in low-income countries since World War II. First, they approximate the time when new effective public health technologies were introduced in the world against seven major infectious diseases or causes of death. Second, they consult League of Nations estimates of country-specific cause-of-death rates. The approximated discontinuities over time in these disease-specific health technologies are then interacted with the initial prevalence of each disease in each low-income country for which they have data. These disease-specific health technology changes weighted by country-specific initial prevalence of the disease become their instruments to account for life expectancy in the first stage of their analysis. The instruments account for a significant share of the increase in life expectancy (and in population growth) in their sample of countries from 1940 to 1980. But the increases in life expectancy predicted by these instruments do *not* account for the level or growth in *per capita*

income in this period. They cast “doubt on claims that unfavorable health conditions are the root cause of the poverty.” As already noted, life expectancy is not a perfect indicator of the contemporaneous productive benefits of health, and although they introduce discrete lags for income growth of 5, 10, or 20 years, to allow the gains in life expectation due to reduced child mortality to affect income growth, this method may still not capture the dynamic life course biological process by which healthier surviving children become more productive adults and thereby enable their countries to grow more rapidly in the long run. African countries are also notably absent from their sample, because of the scarcity of reliable mortality data in these countries for this time period.

Lorentzen et al. (2008) consider growth regressions across countries from 1960 to 2000 and concludes adult (i.e., age 15-60) mortality is more significantly associated with growth than is infant mortality. They assume first that 12 environmentally fixed indicators of malaria ecology, climate, and geography are valid instruments for predicting infant and adult mortality rates at the country level, and second that these age-specific measures of mortality affect three inputs to growth, namely, the investment rates (physical capital), secondary school enrollment rates (proxy for human capital), and total fertility rates (proxy for cohort fertility). Their two- and three-stage structural estimates of growth depend on the exclusion restrictions they impose on the two intermediate levels of their model: (1) the 12 fixed environment instruments affect growth inputs and growth *only* through their correlations with the age-specific measures of mortality and (2) that mortality affects growth *only* through their correlations with the distinguished inputs to growth of investment, enrollment, and fertility. Both restrictions appear tenuous. For example, why should being a landlocked nation affect economic growth only by means of variation in age-specific mortality, and not by traditional economic routes, such as access to international trading opportunities. Why should fertility be specified as a negative input to growth, except conveniently because it happens in this period to be negatively correlated with income and growth (cf. Barro & Sala-I-Martin, 1995; Kuznets, 1967). Moreover, for some parents fertility is a choice, which appears to respond as might be expected in a simple economic model to the wages of men, women and children, wealth, production technology and technical change, women’s education, and access to birth control, among other factors (Schultz, 1997, 2008a; Section 8). It has also been hypothesized that exogenous variation in mortality could affect the demand of parents for births. Yet most empirical models of economic growth fit to cross-country

data continue to assume health, mortality, and fertility are all determined outside of the model or exogenous, and this methodology conceals whatever role there may be for population policy to affect development.

Conditional on the assumptions underlying the model of [Lorentzen et al. \(2008\)](#), the estimated standardized association between adult mortality and growth is larger, as expected, than the standardized association between infant mortality and growth, if for no other reason than that children do not immediately contribute to national income, whereas adult often do. For example, a one standard deviation decrease in adult mortality is associated with an increase in annual economic growth of between 0.8% and 1.1% points, when the average growth in their sample is 1.8% per year. A standard deviation decline in infant mortality is associated with a smaller 0.27% point increase in growth, and their estimated association with infant mortality is not significantly different from zero (Table 10). Thus, the specification choice of Acemoglu and Johnson (2007) and most other studies of growth in national income to rely on life expectancy at birth as their measure of health assigns a larger weight to the variation in infant mortality in this phase of the demographic transition than is justified for understanding short-run variation in production. [Lorentzen et al. \(2008\)](#) also find their reduced-form association between adult mortality and growth is attributable in their recursive model to an association between lower adult mortality and increased capital investments and lower fertility. However, the association between adult mortality and secondary school enrollment rates, or enrollment and growth are both insignificant, which may signal a limitation of this specification, or a shortcoming of the [Barro and Lee \(2000\)](#) school attainment series used in many cross-country studies ([Topel, 1999](#)).¹¹ It is interesting, however, that changes in adult mortality and infant mortality are empirically distinguishable in this period, suggesting that future studies should at least decompose changes in life expectancy at birth into variation in adult and in child mortality as potentially distinct determinants of growth, even if the pathways by which mortality today affects growth inputs and outcomes in the future are not yet understood (cf. [Weil, 2007](#)).

5.1 Macroeconomic evidence of the impact of income on health

The second causal relationship to assess in the field of health and economic development is the effect of personal income and other features of economic development that may influence mortality, morbidity, and health status. To estimate the effect of income on health at the country level, Pritchett and Summers (1996) examine changes between 5-year intervals in income and health in low-income countries from 1965 to 1985. Estimating by ordinary least squares (OLS) the linear association between changes in national income *per capita* and changes in the infant mortality rate, controlling for time-period effects and adult schooling levels, they find a 10% increase in income is associated with a 1.9% decrease in infant mortality. Because other factors could affect both income and mortality, they then propose several possible instrumental variables (IV) accounting for income changes that are assumed to affect health only through their impact on income, or in other words instruments correlated with income but uncorrelated with the errors in mortality or unexplained variation in health. Among their proposed instruments, the most plausible is a country's "terms of trade" (Z in Figure 1). Presumably an increase in the price of a country's exports relative to the price of its imports is an exogenous "shock" promoting domestic income growth, but because export prices are determined outside of the country in the global economy, this shock should not affect the health of the domestic population except through income. This IV estimate of income's effect on infant mortality is not significantly different from zero (i.e., $t = 1.28$). Among their other four proposed IV estimates, only the "investment to income ratio" yields a second-stage estimate which is statistically significantly different from zero, implying a 10% increase in income is associated with a 3.5% decline in the infant mortality rate over a 5-year period. However, the "investment ratio" is not a valid instrument, because it could be affected by many factors in the domestic economy such as weather, civil conflict, or development policies, all of which could arguably also affect health and thereby violate the assumption for IV methods. When Pritchett and Summers consider 5-year differences in life expectancy as their dependent health variable, the direct OLS association implies a 10% increase in income is associated with a smaller 0.15% increase in life expectancy, and this association is not significant. Moreover, none of the IV estimates for income's effect on life expectancy differ significantly from zero. Nonetheless, Pritchett and Summers conclude that "gains from rapid economic growth flow into health gains," whereas the actual evidence they report suggests the contrary that direct or IV linkages in the cross section of countries are not significant *within* low-income countries. When a credible instrument, such as terms of trade, is used to predict

exogenous variation in national income, which is sufficiently powerful in the first-stage equation, there is no significant income effect on health according to IV methods, even when health is proxied by infant mortality, a relatively sensitive short run measure of health.

Pritchett and Summers (1996) do not consider a suitable instrument which might allow them to vary exogenously adult mortality without directly affecting national income, and thereby estimate the influence of health on the productivity of workers and assess empirically the strength of causation flowing in the opposite direction from population health to national income. These macroeconomic studies do not offer a convincing basis as yet for identifying causal relationships between health/mortality and development in either direction. The historical relationships Acemoglu et al. (2001, 2003) describe between disease conditions and colonial settlement strategies and resulting institutional developments are plausible, but they are not relevant for assessing the contemporary consequences for income of health policies or measures designed to control specific diseases. Pritchett and Summers (1996) report no significant evidence of the impact of income on infant or life-span mortality. These cross-country studies do not succeed in disentangling satisfactorily the causal relationships that might account for the provocative association between health and development.

5.2 Alternative indicators of health status and their economic consequences

Contemporary epidemiological and historical studies have documented that bad early life health conditions experienced by a pregnant woman, her fetus, and young child are significantly associated with the child's reduced longevity after age 50 (e.g., Alters & Oris, 2006; Barker, 1994, 2001; Bengtsson & Brostrom, 2006; Bengtsson, 2009). The unstated reason for focusing on late life mortality is probably because too few deaths occur between age 15 and 50 to analyze in the moderate sized historical samples. A growing number of econometric studies are also finding that bad early life shocks to health are associated with decreased cognitive test scores, schooling attainment, occupational status and earnings, and with increased adult morbidity, chronic health problems, nonparticipation in the labor force, and disability even before the age of 50, and more notably thereafter (e.g., Almond, 2006; Bleakley, 2007; Costa, 1996; Glewwe, Jacoby, & King, 2000; Maccini & Yang, 2006; Maluccio et al., 2006; Miguel & Kremer, 2004).

If these early bad health shocks increase morbidity and disability among working aged adults, one might then expect four to five decades after an improvement occurs in maternal and child health, adult health and labor productive potential per working aged population should increase, while the decline in late mortality would raise slightly the adult share of the population and add further to growth in income *per capita*. The challenge for research is to identify exogenous variations in fetal growth and early child health that can be convincingly linked to long-gestating adult health, worker productivity, and economic growth per adult. These lines of research are discussed further in [Sections 6](#) and [7](#).¹²

Many studies analyze the birth weight of a child, which is highly positively correlated with child survival, and child's cognitive performance, school attainment, adult earnings, and late life survival and health status ([Alderman & Behrman, 2006](#)). Cross-sectional association between birth weight and subsequent outcomes, however, could be biased from causal relationships, because unobserved preferences and behaviors of parents, and their access to medical care and health-related inputs could not only influence the child birth weight, but also affect subsequent health inputs and behaviors, contributing to later welfare outcomes ([Rosenzweig & Schultz, 1983](#)). Evidence is more confidently interpreted when differences within twins are followed, for whom within twin differences in birth weight measure exogenous differences in rates of fetal growth, and can then be causally related to later parent health inputs and the child's development, holding constant for such commonly omitted variables as gestation, mother's health, family fixed effects, and even genetic potential affecting ability or initial health endowments ([Behrman & Rosenzweig, 2004](#); [Black et al., 2007](#); [Rosenzweig & Zhang, 2009](#)).

This literature is reviewed later in the chapter and suggests that the productive benefits of the decline in child mortality, which is responsible for most of the increase in life expectancy in low-income countries in the twentieth century, might be expected to impact adult economic productivity after a lag of several decades. Moreover, declines in child mortality are frequently associated with declines in fertility within a decade or two. And voluntary reductions in fertility may facilitate economic growth, if they are associated with women increasing their participation in the market labor force as they reallocate time from child care and allied home production, and potentially increase life-cycle savings and investments for retirement, and lead to a substitution of more human capital per child for having fewer children (discussed in [Section 8](#)). If change in life expectancy at birth is a poorly designed indicator of change in the productive stock of health

of the current working age population that might drive economic growth, how can the latent productive stock of health human capital be better measured at both the aggregate level and the individual level?

The present discounted value of the expected future productive lifetime of an individual is another approach to value the economic gains from reducing mortality for individuals at each age. This present value calculation would assign a larger productive value to saving a life of a 25-year-old adult compared with saving the life of an infant who would not work for more than a decade. Alternatively, the standard summary measure of life expectancy at birth implicitly weights the saving of an infant's life more heavily than that of an adult, because this synthetic cohort measure of mortality does not (1) accumulate the burden of investment costs of child rearing, (2) discount these costs and eventual adult earnings back to the time when an intervention might have avoided a premature death, and (3) allow an individual's future productivity to depend on the intervening accumulation of capital, technical change, and inputs that could complement labor. Life expectancy attaches the greater value to improvements in the survival of a cohort when the improvement occurs at the earlier age. If the valuation of life is based on a discounted value of the profile of consumption for a synthetic cohort, rather than on the profile of productive potential, the social weight assigned to saving the infant's life would increase relative to that of a working aged adult (Usher, 1973). On the other hand, national income statistics do not currently treat health or even schooling as an investment which can add to future productivity and growth. If national income statistics treated human capital as they do physical capital, then the present discounted value of lifetime total productivity (in market or nonmarket activities) could provide an analogous "production basis" for imputing a value to health improvements that would also account for future growth in national income. Valuing lives among the elderly may still require an extended human capital conceptual framework involving a social utility function, or the elderly consumer's willingness to pay for extending their life. Public health services may in addition be viewed as producing a public good or a positive externality, if your welfare improves when your neighbor's health improves, even though the neighbor is too poor to pay for life-extending public health care (e.g., Murphy & Topel, 2006; Tolley, Kenkel, & Fabian, 1994; Usher, 1973). Observed wages may also be an increasingly unrepresentative indicator of the average potential productivity of the elderly cohort, because those who continue to work for a wage are influenced in their participation decision by their

health status, public pensions, and by the tax treatment of earnings and savings (Gruber & Wise, 1995, 2004).

As emphasized earlier, the pattern of mortality change by age has evolved, possibly due to changes in health technologies, the economic benefits and health burdens of urbanization, and the diffusion of new diseases in the world. Consequently, a year increase in life expectancy at birth is associated at different times and in different countries with changes in different causes of mortality, affecting persons of different ages, with different implications for *per capita* productivity and desired fertility.

The rising costs of treatments for degenerative diseases is one factor underlying the growing share of national income expended on health-related goods and services in rich countries, and studies suggest the income elasticity of health expenditures exceeds unity, that is, consumers treat health expenditures as a luxury (see Table 1). Unless these health-related expenditures in high-income countries spill over and reduce substantially the opportunity cost of health therapies available to low-income countries, the convergence by income levels in health status across countries may not continue. However, in the case of anti-retroviral drug therapies for those living with HIV/AIDS the cost for low-income countries has decreased sharply in the last 5 years, suggesting it is possible for modern medical technological advances to spill over as a public good and benefit the world's poor. Nonetheless, to sustain the convergence in health across countries and across socioeconomic status groups within countries, new policies may be needed. Increased population assistance may be necessary, a social redistribution of the costs of effective preventive and even curative health care may be required, and intellectual property rights associated with new drugs and medical technologies may have to be evolved that benefit more widely poor countries or peoples (see Table 2).

6. MICROECONOMICS OF HEALTH AND DEVELOPMENT: INDIVIDUALS AND HOUSEHOLDS

Critical periods of early human development during which conditions can encourage or deter the healthy development of humans and influence not only their early survival and extended life span, but also affect in a complementary fashion their cognitive performance, schooling, and their productivity as adults, as measured by wages and earnings. Research on this topic is

reported by epidemiologists, medical researchers, demographers, anthropologists, economic historians, and finally most recently, by economists studying development in low-income countries. These disparate literatures are only beginning to be integrated, and the policy implications of the research are not always clear, though it is expected that more social attention will be directed in the future to care of pregnant women and to the health environment of children in the first few years of life in order to achieve long-term improvements in the health and economic potential of populations (Gluckman & Hanson, 2005; Koenig et al. 1991; Frankenberg, 1995; Frankenberg, et al. 2005). Only a few segments of this emergent field are summarized here to show how the measurement of health has evolved to rely on anthropometric indicators of health and physical development, how these indicators are used to link background conditions to economic behavior, the use of health inputs, and health outcomes, and motivate the use of two-stage estimation methods for quantifying key relationships and testing causal hypotheses.

McKeown (1976, 1979) concluded from an analysis of cause-specific death rates in the UK that the gradual increase in life span from 1750 to 1900 was due to rising standards of living, because the decline in death rates could not be explained by effective medical interventions or public health initiatives. Without finding any change in medical or public health technology or practices to explain adequately the actual reduction in deaths by cause, he hypothesized that the decline in mortality until the end of the nineteenth century was related to the improvement in standards of living and specifically nutrition, and perhaps also the reduced crowding of poor populations in urban housing.

Fogel (1986, 1994, 2004) extends this line of thinking by drawing on the nutrition literature to show how the available supply of calories to a population is initially consumed to maintain basic metabolism and physical maintenance activities, as well as used to fight off infections and disease (Scrimshaw, Taylor, & Gordon, 1968). Only calories in excess to those required for sustaining life are then available to support productive “work” and leisure activities. This nutritional accounting scheme suggests that increases in the *per capita* supply of calories yields first a phase of increasing productive potential, followed by decreasing productive returns to calories as individuals approach a level of nutrition where the cost of additional calories exceeds the value of their marginal contribution to health and productive work. Increases in the consumption of calories may also be transformed into increases in weight, represented by BMI,¹³

which is associated with improved health and increased physical capacity for work, until BMI reaches a value of about 25, illustrated by the relative risk of mortality in [Figure 4](#), based on Norwegian data from the 1970s ([Waller, 1984](#)) and the Union Army Veteran sample from the 1880s ([Fogel, 2004](#)). Obesity occurs as BMI exceeds a threshold of about 30, above which adult risks of mortality increase and productivity is expected to fall. [Fogel \(2004\)](#) extends this form of calorie requirement accounting to his historical estimates of the personal distribution of calories available to the populations of England and France from 1700 to 2000. He thereby offers his explanation for France's initially higher mortality and lower labor productivity in terms of France's lower *per capita* supply of calories compared to England's in the eighteenth century. He thus accounts for much of the increase in European labor productivity since the industrial revolution in terms of increased adult height and improvements in the distribution of BMI ([Komlos, 1994](#); [Steckel, 1995, 2008](#)).

In other words, increases in the current flow of nutrition in calories in excess of the requirements for work and illness should reduce the fraction of the population with very low BMI, and shift the lower tail of the distribution of BMI to the right. This shift is interpreted as an accumulation of the population's health human capital stock, which tends to be associated with both declines in mortality and increases in labor productivity. By approximating stocks of health human capital in these objective terms of the physical stature (i.e., height and weight) of populations, Fogel has oriented researchers to measure time series variations in these anthropometric indicators of well-being or health status within relatively closed populations. Researchers have also documented how adult chronic health conditions improve for those who have experienced less nutritional deprivation and less exposure to infectious disease causing inflammation or micronutrient deficiency, especially as a child ([Crimmins & Finch, 2006a](#); [Finch, 2007](#); [Fields, et al. 2007](#)). The challenge is to quantify inflows of nutrition, various claims on these calories, such as diseases, that could together affect stature and imprint on the epigenetic process, and work through these hard to observe pathways from measurable indicators of health status to biological processes shaping physical and mental development, and economic productive capacity, as well as life expectancy.

Substitution and complementarity between nutrition, health inputs, preschool stimulation and training, as well as intertemporal tradeoffs, and diminishing returns to scale suggest child development should be modeled to include health, cognitive and noncognitive capacities. Health

status or the stock of health human capital can in addition be assessed in household representative surveys by the functional capacity of persons to perform activities of daily living (ADLs) which seem to discriminate health status among the elderly. Finally, the effect on productivity and labor supply of “days ill and unable to work” during a specified interval, such as the month before a survey, can also be treated as an endogenous indicator of health status and evaluated as a determinant of wages and labor supply using instruments related to the individual’s birthplace and socioeconomic origins (Schultz & Tansel, 1997).

6.1 Height, weight, body mass index, and birth weight as indicators of health stocks

Anthropometric indicators of height and weight and birth weight are being increasingly used in comparative studies of the nutritional and health status of historical and contemporary populations. Height as an adult includes the long-run effect of fetal and childhood nutritional limitations and disease environment and is referred to as “stunting” when height is two standard deviations below the average in a reasonably well-fed reference population, whereas weight for height (BMI) responds to the shorter-run nutritional balance among food, disease, and work and is referred to as “wasting” when BMI values are two standard deviations below average (Fogel, 1994, 2004; Waaler, 1984; World Health Organization, 1995, 2006).

Adult height is thought to be largely determined by an individual’s very early development, and to be more or less fixed by the age of four, conditional on genetic height potential (Floud, Wachter, & Gregory, 1990; Martorell & Habicht, 1986; Eveleth, 1986). Elo and Preston (1992) conclude their review of the mortality literature succinctly: “Height is probably the single best indicator of nutritional conditions and disease environment of childhood. Like date and place of birth, it is a summary measure of many health-related circumstances and events, but it has the advantage of reflecting the experiences of an individual child.” Because adult height is more or less stable over the life course from age 25 to 55, the age “profile” of heights estimated from a cross-sectional survey or census of a population with negligible migration should portray time trends in childhood nutrition and exposure to infections (i.e., scarring). But these historic trends of early biological development is revealed among survivors, and may be altered by swings over time in past mortality that cull from the survivors of a cohort (i.e., selection) less healthy

individuals at more vulnerable ages, who might be shorter, on average, as in the Chinese famine of 1959-1961 (Almond, Edlund, Li, & Zhang, 2007; Gorgens, et al., 2007; Schultz, 2004a). Fertility is also to some degree a choice variable, and some groups in society reduce their birth rate when economic and health conditions are uncertain or threatening, introducing the possibility that the socioeconomic composition of birth cohorts could change in anticipation of hard times that could modify the apparent effect of scarring and selection, and could affect as well the composition of births by season (Buckles & Hungerman, 2008; Lokshin & Radyakin, 2009; Thomas, 2008). Composition, scarring, and selection could therefore all potentially leave their mark on the stature and health of a series of birth cohorts observed in surveys or censuses, though the scarring effect is thought to be the dominant factor in the last century. It is ultimately important to consult panel data for a large population in which individual stature is measured and subsequent mortality is recorded reliably. Waaler (1984) documented that the risk of mortality relative to others in an age and sex group declines for taller Norwegians over the age of 50 in the 1970s, and the regularity of declining relative mortality risk for those of greater height is approximately linear (Figure 4), and is especially salient for death due to cardiovascular disease, tuberculosis, and chronic obstructive lung disease.

In low-income countries the relationships between height and BMI, as indicators of health human capital, and health outcomes, such as mortality, morbidity, and labor productivity, are difficult to document among adults because there are few large panel surveys that record stature, health and economic outcomes and health-related inputs, and register death relatively completely by cause. However, height can be measured both for aggregate populations and for individuals from a single survey, although it should be emphasized that the information “signal” conveyed about prior nutrition, disease, and latent health by adult height is relatively small, unless averaged over a large population, because of the relative importance of genetic variability in this characteristic. However, height appears to be more objective than many self-assessed health indicators, and therefore may be less biased by subjective considerations or cultural conditioning, and may discriminate between the latent health status of younger adults, before chronic diseases and disabilities emerge in late middle age, after which ADLs may be a reliable survey indicator of health status.¹⁴ Panel data on the evolution of health outcomes for individuals over a lifetime and responses to potentially exogenous health, economic, and weather shocks promise to reveal how these shocks affect health stocks and welfare, holding individual initial health endowments

constant. But even correlations between changes over time in health and productivity may still not capture causal effects, if the earlier outcomes are themselves endogenous or responding to prior unobserved constraints and preferences of the parents and children, and to their family's available health care.¹⁵

Where death registration is incomplete and unreliable, data on adult height can still be collected in a representative survey to provide a historical window on the evolution of health human capital in the population, and even time trends may be estimated within closed ethnic, racial, or caste groups, who do not change their assignment by group over their lifetime (Schultz, 2003). Figures 5 and 6 report the pattern of average adult female height for rural/urban/total populations by 5-year moving averages of year of birth in Ghana (1987-1989), a country experiencing little economic growth in the three decades after its independence in 1957, and Brazil (1989), a country growing relatively rapidly from 1935 to 1980. *Per capita* national income increased in this period more rapidly in Brazil than in Ghana, and this is one possible explanation for the more marked increase in height among younger women in Brazil compared with those in Ghana, even though both countries should have had access to similar international improvements in medical technology (Schultz, 2005).

Attributing the secular increase in adult height to specific causes in high-income countries remains controversial, and estimating the longer-run cumulative and even short-run responses of height to increases in income or decreases in infectious diseases are debated, especially in less developed countries. Yet the magnitude of differences across birth cohorts cannot be readily attributed to genetic changes in the high-income populations in such a short period of time. The share of the variance in individual height attributed to environmental risks and socioeconomic conditions (in contrast to genetic factors) is thought to be on the order of 20% in high-income countries today, such as the United States (Stunkard, Foch, & Hrubec, 1986) and Finland (Silventoinen, Kaprio, Lahelma, & Koskenvuo, 2000) based on twin studies of trait heritability. But the environmental share of height variation is believed to be larger in low-income settings where malnutrition is much more common and disease imposes a heavy burden on available proteins and calories needed for satisfactory physical development (Silventoinen, 2003). Gene-environment interactions can also be important, and if these factors are correlated and contribute nonlinearly to height and socioeconomic outcomes, the task of decomposing the effects of genes and environment on adult height is still more complex. Genetic studies of five ethnically and

geographically dispersed populations suggest that 39-56% of the variance in individual adult height is correlated with a few major gene effects (Ginsburg, Livshits, Yakovenko, & Kobylansky, 1998). Socioeconomic characteristics of parents and birthplace also account for a significant amount of the variation in individual height, but for a substantially smaller fraction of the variance than is believed to be determined by a few genomic regions of chromosomes that have been scanned (Liu et al., 2004). Length at birth and height as a child and adult are, of course, persistently highly correlated over the life cycle of an individual, although the timing of the adolescent growth spurt occurs earlier as does puberty in the better nourished population. Both child and adult height are significantly associated with cognitive ability, schooling attainment and adult wages or productivity, in both high- and low-income countries (Schultz, 2003; Strauss & Thomas, 1998). The causal interpretation of these associations between height, and health, ability, and productivity remains open to debate, though some view height as simply one indicator of cognitive ability, and when these IQ measures are appropriately controlled, height loses much of its power to predict productivity in a recursive framework (Case & Paxson, 2008).

Data on adult height has only recently been collected from representative household surveys in low-income countries, such as for women of childbearing age in the DHS (downloadable at <http://www.measuredhs.com>). Because these surveys do not provide much information on economic or disease conditions at birth or even at the time of the survey, analysis of these height data examine time trends in averages for birth cohorts. Deaton (2007) has matched the height of surviving female birth cohorts age 25-55 to national income (log GDP *per capita* in purchasing power parity from Penn World Tables) at the time of the women's birth, and the infant mortality rates (interpolated to the birth year from World Bank estimates every 10 years). In a pooled sample of low-income country survey cohorts from the DHS, he reports few stable relationships between height and income and infant mortality, interpreted as measures of nutrition and the burden of disease at the time of birth, respectively. However, there are distinct regional differences in height. On average, the 28 African surveys and that from Haiti (whose population is 95% of African descent) report women are on average taller, despite incomes being lower and infant mortality being higher than in the rest the sample. Conversely, female heights are distinctly shorter in the South Asian countries (i.e., India, Bangladesh, and Nepal). These robust regional regularities in height suggest a genetic component may differ among these regions that

accounts for some of the height differences, and Deaton concludes it is not possible to infer disease or development effects on height outside of the OECD developed countries, because of the likely bias introduced by omitted variables, such as genetic differences, which might be correlated as well as with income and infant mortality. Deaton and coauthors also propose that in low-income countries such as Africa (but not in South Asia?) the force of mortality selecting out shorter individuals dominates the force of scarring which reduces individuals achieving her genetic potential height (Bozzoli & Deaton, 2008). Anthropologists have long noted regional variation in height, but they tend to hypothesize that within a few generations nutrition improvements and decreases in disease lead to convergence in height, as documented in Japan and East Asia, and noted among recent immigrants to the UK from South Asia (e.g., [Eveleth & Tanner, 1990](#); [Floud et al., 1990](#); [Komlos, 1994](#); [Tanner, 1981](#); [Tarozzi, 2009](#); [Van Wieringen, 1986](#)).

Akachi and Canning (2006) focus on the DHS female height data within sub-Saharan African countries born from 1965 to 1985. Average height by birth cohort for 19 African countries provides 438 observations and the simple linear time trends by birth date in height across women age 20-49 are mixed: positive in two countries, negative in two, and statistically insignificant in the remaining 15 countries. Then they add linear controls for GDP *per capita* and infant mortality, following [Deaton \(2007\)](#), as well as protein and calorie availability, as estimated by FAO.¹⁶ All of their explanatory variables are initially included not only in the year of birth, but also lagged 5, 10, and 15 years. The health and nutrition literature suggests that when a cohort is growing most rapidly, in the first 3 years and during the adolescent spurt, physical development is most vulnerable to nutritional stress and disease. The final regression specification they prefer is based on retaining only those variables that are statistically significant when country and 5 year of birth time dummies are also included. Infant mortality is significant only in the birth year, which is negatively related to the cohort's height; income is significant only when lagged 15 years and is positive; protein availability is positively associated with height in the year of birth and 15 years later; calorie availability is not significant jointly or independently at any lag ([Table 3](#), column 4). In the final regression, the coefficients estimated from the birth cohort time dummies decline, especially for cohorts born from 1968 to 1977 ([Figure 3](#)). To account for this residual negative time trend in height, the authors speculate that infant mortality may not capture entirely the deteriorating effect of the disease environment on

height, and possible increases in inequality in nutrition and disease might account for the declining trend in height and be important omitted variables in these aggregate regressions based only on population means. However, their reliance on “stepwise regression” to specify their preferred model is a shortcoming, though it does confirm Deaton’s observation that younger women in Africa do not appear to be taller than older ones. Nonetheless, other data sources than the DHS suggest positive time trends in height for male and female birth cohorts, as in the LSMS surveys (<http://www.worldbank.org/LSMS/>) from Cote d’Ivoire, Ghana, Viet Nam, a monitoring survey of Kenya, as well as health and nutrition surveys from Brazil, Colombia, and China (Svedoff & Schultz, 2001; Schultz, 2003, 2005). Deaton (2008) subsequently reports modest gains in height across male and female birth cohorts in India, based on the 2005/2006 NFHS (DHS), but the time trends in height are smaller in rural than in urban areas, especially for women. Most of these surveys, unfortunately, do not include health information on birthplace, or sufficient information on migration histories on which to impute health and nutritional conditions at the time and place of birth.

6.2 Heterogeneity of height as a measure of the stock of health human capital

Height as an indicator of an individual’s stock of health human capital is a proxy for latent health and can be thought of as having at least two components: one which is a form of “reproducible human capital” created by families, individuals and society by nutrition, control of disease, and early health care, and a second component which is not readily explained by observed socioeconomic variables, or greatly affected by measures of environmental risk. Only about a tenth of the variation in individual adult height across a population tends to be accounted for by parent socioeconomic characteristics (e.g., education, wealth, and socioeconomic status) and location of birth and childhood, which may be related to exposure to diseases and availability of health care. The remainder of the variation in height might be attributed to characteristics which may be genetic in origin or conditions affecting unpredictably biological development, and of course errors in survey measurement (Ginsburg et al., 1998; Gluckman & Hanson, 2006).

Schooling is another form of reproducible human capital created by investments of student time and educational resources. A fifth to a half of the variance in years of schooling attained

across adults can be typically explained by family and student exogenous characteristics along with the family's local access to schooling, and a smaller fraction of the variation in schooling is generally linked to intrinsic ability of the student, measured by such variables as IQ. The omission of ability as a determinant of the wage rate in estimating a wage function is expected to bias upward the estimate of the private wage return to schooling, as discussed in [Section 4.2](#) although this has not been widely confirmed perhaps because of errors in measurement of schooling. However, other studies find upward bias in OLS estimated schooling returns, in the sense that if they also condition wages on school quality, treating it as exogenous, the estimated coefficients on years of schooling is likely to fall because quality and quantity are commonly positively correlated ([Behrman & Birdsall, 1983](#)).

BMI could affect wage productivity as a physical attribute of workers or as a latent indicator of health, but the association could also capture reverse causation, because more productive workers have more income to spend on food and health care that would add to worker BMI in the short run. To deal with the potential reverse causality, BMI has been treated as endogenous in the wage function and local prices of food have been used as an instrument for BMI ([Strauss, 1986](#); [Strauss & Thomas, 1998](#)). Generally, the IV estimates of the effect of BMI on wages are significant and positive, though subject to diminishing returns and even counterproductive if the individual is overweight.

Adult height, because it is thought to be primarily determined by an early age, is not expected to be subject to reverse causation with wages. But height could be heterogeneous, in the sense that the majority of variation in height potential is genetic and is not explained by socioeconomic conditions at an early age. Therefore the socioeconomic and genetic variation in height need not capture equally the latent health human capital stock. The two components of height could enhance wage productivity by different amounts. Studies have used instruments from family background at time of birth, food availability, local health services, exposure to diseases, or weather shocks to predict adult height in estimating the determinants of the wage rate of individuals. They find an added centimeter in height is associated according to OLS with a significantly larger proportionate effect on wages in low-income countries than in high-income countries, such as the United States, presumably because of wage returns to height diminish at higher levels of nutrition, health care, and income ([Schultz, 2002](#); [Strauss & Thomas, 1998](#)). However, IV estimates of the log wage returns to height tend to be several times larger than the

direct (OLS) estimates of height on log wages, holding constant for schooling, postschooling potential experience, and sex (Maccini & Yang, 2006; Savedoff & Schultz, 2001; Schultz, 2002, 2003, 2005).

Because the IV estimates of the effect of adult height on adult productivity tend to be significantly larger than the OLS estimates, the Hausman specification test for the exogeneity of height in the wage function is generally rejected, implying that the IV estimates are preferred if the instruments are valid (Schultz, 2002). There are two possible explanations for the difference between the OLS and IV estimates of the wage returns to height: (1) the survey measurement error in height in household sample surveys is relatively large, leading to a downward bias when height is treated as measured without error in OLS estimates and (2) measured height is heterogeneous and the health human capital component of height identified by variation in instruments such as parent socioeconomic characteristics and local health conditions at birthplace contributes to health human capital and increases health and wage productivity more than does the residual (presumably genetic) unexplained variation in height.¹⁷

First, measurement error in anthropometric indicators from surveys may not be negligible. Indeed, multiyear averages of measures of adult height from a panel survey in Cote d'Ivoire yield significant estimates of the proportion of measurement error in height measured in any single round of a survey, and this proportionate error appears to be larger for height than the proportionate measurement error in responses on years of schooling or year of birth. But measurement error in height is still not of a sufficient magnitude to explain a several fold increase in IV compared to OLS estimates for height's effect on log wages (Schultz, 2003). The second hypothesis is the more plausible: height is a latent indicator for health human capital and includes the modest effect of early biological development processes that also contributes to the individual's persisting health endowment, adult productive capacities, and longevity. The genetically induced variation in height, on the other hand, that accounts for the bulk of the observed variation in height across individuals is less closely related to productivity than is the reproducible component of height predicted on the basis of socioeconomic characteristics of birth environment and family endowments.

Investments in health human capital are thought to occur throughout life, but as with determinants of height, they appear to be critical in biological developments during specific periods in fetal development *in utero* and during very early childhood (Crimmins & Finch,

2006b; Finch, 2007; Gluckman & Hanson, 2005, 2006; Martorell & Habicht, 1986).¹⁸ Height is likely to be affected by household resources and the environment of the mother during her pregnancy and during her child's first few years of life, and her education may increase the effectiveness of these health inputs and facilitate her adoption of best health practices for her child (Barker, 1992, 2005; Rosenzweig & Schultz, 1983; Strauss & Thomas, 2008). Childhood height also forecasts reasonably well adult height, and it is generally correlated with lifetime health and life span, mental or cognitive capacity, learning of skills, and productive outcomes for the individual over the life cycle (e.g., Almond, 2006; Cunha & Heckman, 2007; Elo & Preston, 1992; Floud et al., 1990; Fogel, 2004; Heckman, 2008; Komlos, 1994; Maluccio et al., 2006; Martorell & Habicht, 1986; Schultz, 2003, 2005). Much remains to be understood regarding this dynamic process and the socially and privately optimal allocation of health and development inputs over the course of a lifetime.

Cross-tabulations of surveys and censuses since the 1970s document the significant positive correlation between child survival and the schooling of the mother, even after controlling for the schooling of the father and many other household and community exogenous factors (Caldwell, 1979; Cochrane, O'Hara, & Leslie, 1980; Mensch, Lentzner, & Preston, 1985; Schultz, 1980, 1997). One year more schooling is associated with a 5-15% decline in early child mortality. It is an open question whether this empirical regularity reflects the knowledge of child health care obtained by better educated women, or women's empowerment that stems from their schooling relative to their partner, which allows them to allocate more household resources to food and child health in response to women's greater altruism toward their children (Schultz, 1990; Thomas, 1990). The impact of mother's schooling also generally impacts more positively than that of the father on other child investments, such as schooling, and this has led many to conclude that public transfers to women are more effective in raising the welfare of children than transfers to fathers. Hence, the structure of the influential Progres program started in 1997 in Mexico mandated that the mother should receive the income transfer, conditional on her children attending school and receiving regular preventive health care (Gertler, 2004; Parker, Rubalcava, & Teruel, 2008). The schooling of the mother is also generally associated with the favorable physical development of the child, such as birth weight, height for age, and weight for height.

Weight for sex and age, as already noted, is generally transformed into a BMI, defined as the weight in kilograms divided by height in meters squared, and its impact on health is distinctly

not monotonic, in contrast to that with height. Waaler (1984) in his study of the Norwegian population (1963-1979) demonstrated in Figure 7 that the relative risk of late mortality varied by BMI, controlling for age, sex, following a U-shaped pattern with respect to BMI for a variety of causes of death or diseases, with above average relative risks of death associated with BMIs less than 21 and over 29. Reporting these patterns for specific causes of mortality among persons over the age of 50, Waaler linked the effects of BMI on health to the functioning of particular organs and the resistance of individuals to specific degenerative diseases. The analogous calculation is performed for the Union Army Veteran's sample of Fogel (2004) from about 1865 to 1900, resulting in a similar pattern (Figure 7) (Costa & Steckel, 1997).

It is hypothesized that a malnourished fetal environment will imprint on genes a metabolic adjustment which will compensate to some degree for the expected lifetime shortages of nutrients facing the child, by converting more dietary calories into weight gains and stores of fat to sustain regular development in lean periods. Then, in the event the child's family experiences an increase in income or improved access to nutrition, the child as an adult will be more likely to become obese or exhibit an unhealthy elevated BMI. As obesity has become more common in high-income countries, its prevalence has increased even more rapidly in a number of relatively low-income countries, such as Mexico (Strauss & Thomas, 2008). It should be recalled, however, that individuals with very low BMI are still the most likely to die in many parts of the developing world, including South Asia and sub-Saharan Africa. Few other large panel studies of risk factors for mortality and morbidity are found in the literature to replicate Waaler's association between height and BMI and older adult mortality in low-income countries. Further replications of Waaler's findings would help to evaluate these anthropometric measures of health outcomes, and how variations in these measures are reproduced by the individual's origins and environment.¹⁹

Su (2005) provides such a comparison over time of the Waaler curve between BMI and mortality for US white males age 50-59. He relates the overall mortality risk to BMI based first on the Union Army Veteran Survival sample followed from about 1860 to 1900, and then for a representative US survival sample from the 1971-1974 NHANES-I to the epidemiological vital status follow up survey in 1993. Su reports, as did Waaler, a U-shaped variation in the relative risk of death with increasing BMI, measured over an 18-year period (Figure 8). Using a variety of methods to fit quintiles in BMI to relative mortality rates starting in about 1860 and 1972, Su

shows that the healthier BMI values appear to have shifted to the right or to higher values of BMI, between these two observations of the US population separated by roughly a century. The optimal BMI, namely that with the lowest relative mortality risk, increases from about 21 in 1870s to nearly 26 in 1972, although it may be noted that the lowest mortality segments of the Waaler curve are quite flat in this middle range of BMI.²⁰ Many factors could have contributed to this shift over time in the Waaler mortality curve with respect to BMI in the US, and to the difference between Norway and the US in the 1970s. I do not know of other research accounting for changes in this association between BMI and mortality or morbidity and disability.²¹

In addition to height and BMI, birth weight or fetal growth rate or rate of uterine development are related to later life health status and economic performance. The relationship between birth weight and developmental consequences for child survival, health status, schooling attainment, and economic performance of the mature child is interpreted by some as the causal effects of birth weight (Case et al., 2005; see footnote 15). But this inference depends on the assumption that parents have no effect on their child's birth weight and on other prenatal and postnatal health and developmental inputs for their children that might be correlated with those observed, that is, birth weight. More realistically, parents do influence their children's birth weight by their prenatal care and most obviously by their smoking behavior and nutrition, spacing of births, and the timing of prenatal care (Rosenzweig & Schultz, 1983). It is, therefore, likely that birth weight will be correlated positively with other unobserved child health and developmental inputs, because all of these parent health behaviors may be affected in the same direction by unobserved parent preferences regarding investments in child health and development and intergenerational altruism. Other unobserved constraints affecting the family and its children may also impact both birth weight and subsequent health inputs for the children. To avoid this "heterogeneity bias" in estimating the determinants of birth weight, Rosenzweig and Schultz (1983) use arguably exogenous local prices of health inputs and the local availability of maternal health services as instruments to predict variation in parent provision of prenatal health inputs, smoking, age, and parity, and these predicted inputs are then used to explain birth weight in a consistently estimated child health production function using two-stage least squares or instrumental variable methods.

Another basis for identifying exogenous variations in birth weight that is not affected by parent preferences or unobserved constraints on the family involve comparisons between the

birth weight of identical twins, as introduced in [Section 4.3](#). [Behrman and Rosenzweig \(2004\)](#) conclude that fetal growth and presumably uterine nutrition is a significant pathway for lifetime effects on health and economic outcomes of the adult female, but this research does not shed light on how health inputs or behavior of parents might be modified to improve birth weight, or how policies could increase birth weight.²² [Black et al. \(2007\)](#) examine all Norwegian births from 1967 to 1997 to estimate the effect of within twin birth weight differences on infant health indicators (e.g., APGAR scores), completing high school, and labor market success. They also can assess IQ for males who were required to take a military exam at age 18-20, and find within twin 10% differences in birth weight are associated with 1/20 stanine difference in IQ.²³ With the large magnitudes of causal effects of birth weight on human development and adult productivity, the economic case can be made for policies that reduce the incidence of low birth weight children in low-income countries ([Alderman & Behrman, 2006](#)). However, the precise policies that would achieve this objective of reducing the proportion of low birth weight children in the world have not been clarified or the cost of such policies estimated.

These indicators of prenatal physical growth and early health endowments may embody not only the effects of parent preferences in and between children, but also the response of parents to independent variation in child initial health status, including as well their gender, and thereby compensate or reinforce the children's prior health endowments. Consequently, indicators of the physical growth of children cannot be viewed as exogenous to their family, except for prenatal growth in the restricted sample of twins, where fixed effects are allowed for each pair of twins. In standard samples of all births, birth weight and subsequent development of the child are likely to reflect parental choices informed by parent understanding of the child health production technology, their preferences for different health and welfare outcomes for their children, to their child's health which can clearly also influence postnatal health investments in their different children.²⁴

Studies of differences between twins, reviewed earlier, also shed light on the parental tradeoff of quality and quantity of children. They suggest the adjustment in quality to an exogenous increase in fertility is generally negative, but it is absolutely smaller and more complex than association across all births or twins. [Behrman and Rosenzweig \(2004\)](#) show that the exogenous differences in fetal growth rate between identical female US twins are associated positively with twin differences in schooling and wage rates, confirming that this initial

endowment of a child is reinforced by later investments in child quality. Rosenzweig and Zhang (2009) estimate from a sample of Chinese twins the consequences of exogenously increasing family size due to twins on child quality. In a high-income, low-fertility Norwegian population, Black (2005) finds the occurrence of twins leads to increased family size, but does not significantly lower child quality as measured by schooling of the earlier born siblings. The Norwegian parents appear to compensate for the lower birth weight of their twin children, as Griliches' (1979) concluded from his early survey of US sibling studies.

The task of estimating how height, BMI and birth weight affects the child's later adult's life requires that the researcher first understand what exogenous factors affect variation in these early health indicators, and more specifically the variables that affect these health indicators which can be influenced by policy. In other words, how is health human capital produced? For example, favorable rainfall in a location in the year before an individual is born into an agricultural society may be associated with an increase in height of this adult. In Indonesia local rainfall deviations predict the height of females born locally in that year, and also increase the probability that these women marry a taller husband, who has higher expected earnings, and she will enjoy higher *per capita* consumption in her household (Maccini & Yang, 2006). Estimates based on the actual (all of the variation in observed) height, and not just the component of height related to the rainfall instrumental variable, would mix together genetic factors permutating height in the population which are hypothesized to be less closely related to lifetime productivity than the local rainfall-induced component of height, which could be thought of as simulating a quasiexperimental effect of variation in reproducible health human capital. In several country studies that use as instruments for the offspring's adult height and BMI the socioeconomic background characteristics of the parents, the local health infrastructure, and disease environment at birthplace. These IV estimates of the effect of height and BMI on the offspring's log wage rates tend to be a larger positive value than if the association of height and BMI on log wages are estimated directly by OLS (Svedoff & Schultz, 2001; Schultz, 2003, 2005). This tendency for instrumental variable estimates of the apparent productive returns to anthropometric health human capital indicators to be larger in absolute values than the simple partial correlations is consistent with the previously advanced hypothesis that the reproducible health human capital component of height, identified by their association with socioeconomic instruments or time series deviations in weather operating on income and possibly disease vectors, has a relatively

larger effect on the child than does the variation in height unexplained by these socioeconomic variables, which is more likely to be predominantly genetic variation.²⁵

Table 3 illustrates these empirical patterns for adult height and wages for three countries, Ghana in 1987-1989, Brazil, 1989, and the United States in 1989-1993 (Schultz, 2005). The coefficient on height in centimeters in a standard log wage equation, separately for men and women, age 25-54 who report hours and earnings.²⁶ The first column (a) reports the direct OLS coefficient which implies a male 1 cm taller receives a wage in Ghana that is 1.5% higher, and females 1.7% higher. In Brazil the association is 1.4% and 1.7% higher wages associated with being 1 cm taller for males and females, respectively. In the United States where nutrition is better and people are taller on average (10 cm taller than Brazil) a gain of 1 cm is associated with only 0.6% higher wage for males and 0.4% for females. Columns (b) reports the instrumental variable estimate for height predicted on the basis of regional and household characteristics at birthplace, and these IV estimated effects of height on log wage increase three- to fivefold. The variation in height systematically associated with socioeconomic background and regional birthplace is more steeply associated with wage variation than is the entire distribution of the variation in height including presumably genetic components. If years of schooling of the mother and father are added to the list of instruments in Ghana and the United States, the IV estimates in column (c) of the effect of height remains roughly the same but tends to become more statistically significant as the power of the first-stage regression increases and these overidentification restrictions are accepted by conventional tests (Schultz, 2002; Wooldridge, 2002). The IV estimates based on birthplace and family socioeconomic status are believed to identify the effect of variation in height primarily due to reproducible health human capital, and therefore down weights the genetic components of height. The IV estimates of height on wages may represent the causal effect of height changes that might be induced by nutrition and health interventions, whereas the OLS estimates portray to a greater extent the association between genetic and other sources of variation in height for which social welfare policy probably has little leverage.

Table 4 reports the joint estimation in Ghana (1987-1989) of the wage effects of four forms of human capital: years of education, migration from birthplace, adult current BMI, and adult height. First it is assumed that all four forms of human capital are homogeneous, exogenous, and measured without error. Under these working assumptions, OLS estimates as reported in

columns (1)–(4) have desirable properties. Because the four forms of productive human capital tend to be positively associated across people, the conventional approach to estimating of wage functions which tends to omit several of these human capital factors would be inclined to attribute too large a productivity effect to the included form(s) of human capital, typically only schooling. The conventional returns to schooling that exclude these additional human capital variables are only moderately diminished by the inclusion of the other three forms of mobility and health human capital, declining 15% for males from 0.052 to 0.044. But if these forms of human capital are endogenous, heterogeneous, or measured with error, column (5) provides consistent IV estimates, based on the individual’s birthplace health and schooling environment, and parent education and agricultural occupation. Although this IV approach to estimating the returns to the reproducible human capital component of these four forms of human capital does not change significantly the estimated wage returns to schooling or to migration, it often increases the coefficient on BMI significantly, and increases the coefficient on height several fold. Hausman tests of the exogeneity of these forms of human capital imply education and migration appear to be exogenous, whereas the health variables—height and BMI—are generally endogenous (Schultz, 2003).²⁷ The interpretation proposed earlier is that these forms of health human capital are heterogeneous and the reproducible IV component of height and BMI are significantly more productive than the remaining variation in these anthropometric indicators of adult health associated with genetic variability in the physical characteristics of the worker.

Table 5 illustrates that adult height and years of schooling have increased rapidly in some countries, such as Brazil, Vietnam, and Cote d’Ivoire, by nearly a centimeter per decade for height and a year of schooling or more per decade, but not in stagnating Ghana. Wage functions are estimated for Brazil in 1989 that includes years of completed schooling and height, where height is estimated by instrumental variables as in Table 3. Had these proportional gains in Brazilian wages associated with schooling and height in 1989 been invariant over time from 1960 to 1989, the observed advances across cohorts born from 1940 to 1960 in height would account for a 4.1% increase per decade in male wages, and 5.8% increase per decade in female wages.²⁸ The advance in schooling in Brazil is associated with an even larger increase in wages of males of 16% per decade, while for females the schooling gained across birth cohorts would account for a 22% growth. Although the probable wage effects of secular growth in both forms of human capital are large, the impact of increased schooling on wage growth, according to these

estimates for Brazil from adults born from 1940 to 1960, is able to account for two to four times more growth than that associated with the increase in height health human capital (Schultz, 2005).

Do instrumental variables for local public services, health input prices or subsidies, and parent socioeconomic status summarize variation in health human capital embodied in height or achieved through improving fetal growth? They may also capture secular improvements in fetal and early child development that widely parallels economic growth, improved nutrition, and increased access to preventive child health care. Research on the productive contribution of health needs to describe explicitly geographic variation in child health interventions, relative prices of food, and gains in household real income opportunities for female and male workers that are expected to affect adult height. If these local health policies, income and relative price instruments suggest pathways for policy to affect height, more focused IV estimates are now needed to better approximate local average treatment effects of actual policy interventions that could be scaled up by governments (Imbens & Angrist, 1994). Health policy measures will eventually need to be assessed by allocating randomly across localities the prescribed policy interventions, and then relate these policies to the regions where individuals were born and grew up in order to obtain more credible policy-relevant estimates of how public and private programs affect child and adult health and thus impact adult productivity.

6.3 Pathways from fetal and early child development to adult productive life span

Research primarily in epidemiology and demography is being extended in economics which links the early health environment of individuals to their longevity, health status, and economic performance, and relates the consequences specifically of health for welfare outcomes and productivity over an individual's lifetime. This is a brief and very selective summary of a few studies from this large multidisciplinary literature. More detail is found in Alderman, Hoddinott, and Kinsey (2006) and Strauss and Thomas (2008) from an economic perspective, and Finch (2007), Gluckman and Hanson (2006), and Finch & Crimmins (2004) from an epidemiological and medical perspective, though it should be noted that approaches to statistical modeling differ between fields making some comparisons tenuous.

The “fetal origins hypothesis” of [Barker \(1992, 1994, 2001, 2005\)](#) postulates that some chronic health conditions, such as cardiovascular and lung diseases, are caused by the environment *in utero* which affects fetal growth and development at various critical periods in gestation. The initial evidence for the hypothesis was the correlation between place of birth in the UK and risks of mortality as older adults, where it was reported that being born in a high mortality period and location was positively associated with the level of late adult mortality, disproportionately due to the failure of specific systems of organs, such as the heart and lungs ([Barker, 1992](#)). However, many other factors could be related to birthplace health conditions and to later adult chronic health problems, reducing the credibility of these early epidemiological studies as tests of causal hypotheses. More discriminating tests of these hypotheses have sought to specify a “narrower” pathway for a “treatment” of the fetus, and differential effects on the treated, say by gender or economic status, and construction of matched control populations, followed often in a representative sample survey or census. Discontinuity designs are also being used to target the statistical tests to changes occurring at a particular time, given existing time trends. Because chronic degenerative health problems begin to cause substantial numbers of deaths only after middle age, these studies require matching birth and early childhood conditions with the cohort’s health and economic outcomes many years later. With such long lags between treatments and the consequences on health and productivity, the problem of sample selection bias arises, simply due to mortality removing from the birth cohort the more frail individuals, or due to other forms of sample selective attrition, such as due to selective migration or survey nonresponse.²⁹

[Doblhammer \(2004\)](#) examines mortality variation among the elderly by month of their birth to refine our understanding of the fetal origins hypothesis. She finds seasonal patterns among those in the Northern hemisphere, such as Denmark, where those who are born in the spring have below average life expectancy at later ages.³⁰ With the rate of growth of the fetus reaching a maximum in the third trimester, it is believed that if the mother experiences nutritional stress in this critical period as is more common with severe winters and late spring seasons, healthy development of the child’s heart and lung systems tend to be jeopardized ([Barker, 2005](#)). The seasonal pattern among natives in the Southern Hemisphere, such as Australia, is roughly reversed. Moreover, migrants born in the North and residing the South exhibit month of birth effects on their elderly life span similar to nonmigrant natives in the North. The regional effect of

birth month on elderly longevity thus appears imprinted through fetal growth and early development, and cannot be attributed to the differences in the disease environment or health infrastructure that could affect mortality among the elderly differently in the two hemispheres. An active area of research explores what specific diseases, health conditions, and institutional arrangements are associated with the higher elderly mortality due to slower fetal growth rates or particular environmental risks. How are these limits to growth translated into higher mortality among the elderly, what chronic and acute health problems are observed with advancing age due to less adequate fetal development, how do these observable health conditions affect the economic productivity of adult workers and impact their health investments, and modify health inequalities by initial socioeconomic status?³¹ There are significant season-of-birth effects on height and weight for age Z scores of Indian children under age 3 in the three rounds of the National Family Health Survey collected in 1992, 1998, and 2005 (Lokshin & Radyakin, 2009). Births in the monsoon season exhibit lower height and weight, presumably due to greater malnutrition in this lean season and greater disease. These season-of-birth effects also interact with maternal education and a crude household wealth index, suggesting that socioeconomic behavior is a factor modifying the impact of season of birth on early child health and expresses itself in birth weight.

If seasonal variations in these conditions are important, then additional insults to fetal growth may be caused as well by unpredictable drought, floods, weather, pollution, epidemics, or radiation accidents. Narrowly focused analyses of birth cohort data may identify the physical and economic consequences of these “quasinatural experiments” on aspects of the fetal development, impacts on the mortality of older populations, and on other life-cycle events such as schooling, the formation of cognitive skills, earnings capacities, migration and consumption opportunities. Do private and social insurance schemes, or food relief programs, buffer pregnant women and young children from these economic and biological shocks, or are these resources disseminated beyond vulnerable target groups perhaps due to corruption or political economy, and lose their effectiveness to raise child health?

A study of the 1918 flu pandemic by Almond (2006) illustrates how this literature combines a variety of data sources to describe connections between environmental shocks at fetal origin and delayed measures of outcomes among mature members of a birth cohort. The severity and timing of the flu infections varied by states in the United States, providing

instruments to identify the likelihood a child born before, during, and after the pandemic had been affected *in utero* by the flu infection that her mother might have experienced. The cohorts born from January to September 1919, in the wake of the pandemic, exhibit “deficiencies” which can be documented in the US Population Censuses of 1960 and 1980 and can be attributed to the epidemic. For example, the male children of mothers who were likely to be infected by the flu were about 15% less likely to complete high school, and report wages which are 5-9% lower than those born earlier or later.³²

Historical studies have frequently found that periods of economic and health crises are associated with birth cohorts that experience greater late life mortality than do birth cohorts somewhat older and younger, and these extra deaths are often linked to cardiovascular and lung diseases and type 2 diabetes (Elo & Preston, 1992). Alters and Oris (2006) report in several preindustrial Belgium communities (1750-1830) that being born in years of relatively high mortality is a significant predictor of high mortality in middle and late ages for the survivors of these cohorts, even after controlling for a variety of individual socioeconomic characteristics and community effects. These periods of health crises are often associated with high food prices, and are more likely to be associated with higher late adult mortality among those originally born into poorer, more vulnerable, households in crises years. Bengtsson and Brostrom (2006) find in Southern Sweden from 1829 to 1894 the disease load that children are exposed to in the year of their birth is significantly related to their higher mortality after age 55, even after controlling for their landed/landless state (i.e., an indicator of economic status) at birth and at the end of their working career. Additional studies are collected by Bengtsson (2009).

Preston, Hill, and Drevenstedt (1998) note evidence among African Americans that being born and raised as children in farm families is associated with lower mortality as an adult in the early twentieth century United States. They hypothesize that this is due to the childhood disease conditions being more favorable at the start of the century in the rural South than in urban areas of the United States in either the North or the South. Bleakley (2007) finds that the eradication of hookworms after 1910 in the American South contributed in those areas where the infection was most common before the eradication campaigns to increases in school enrollment, attendance and literacy compared to neighboring areas which were subject initially to lower levels of hookworm infection. In the longer run, the eradication was associated with gains in personal income and increased private wage returns to schooling. He argues this one disease eradication

campaign was responsible for closing one-half of the gap between the average years of schooling in the South and North in the United States, and to closing one-fifth of the income gap between these regions. With the evidence of an increase in returns to schooling, the hypothesis that child quality and quantity are substitutes is confirmed by the steep decline in fertility following the eradication of hookworm (Bleakley & Lange, 2009). Crimmins and Finch (2006b) suggest that the reduction in childhood infections with their burden of inflammation is responsible for secular trends in adult height and longevity of birth cohorts over the twentieth century in the United States, recognizing that height is an external anthropometric indicator (observable proxy) for the internal (unobserved) health human capital that impacts mortality (Fogel, 2004; Ginsburg et al., 1998).

Vitamin D deficiency is also biologically linked to bone development and height (e.g., rickets in children and osteomalacia in adults) as well as a variety of chronic diseases affected by fetal development, including cardiovascular, autoimmune, cancers and type 1 diabetes (Holick, 2004; Raiten & Picciano, 2004). Because vitamin D production is stimulated predominantly by sunlight (namely, UVB radiation) and is affected by skin pigmentation or race, variation in height has been studied as a function of latitude, location, occupation, and race (Carson, 2008), and its recent reemergence in the US population may be related to increased breastfeeding without vitamin D supplementation or the use of sunscreen which reduces UVB absorption.³³

Van den Berg, Lindenbroom, and Portrait (2006) examine the business cycle conditions in the year of birth in the Netherlands from 1812 to 1920s, and find significant effects of the business cycle, presumably operating through early life conditions, which are negatively related to end of life mortality rates. They conclude that especially among the poor, food, housing, and health care available at the time of birth affect later health and disability of the population. Although there is growing confirmation of the link from various sources of fetal stress to diminished schooling, cognitive capacity, and earnings for the resulting birth cohort, this literature has made less progress in describing how specific policies can effectively protect individuals during this vulnerable stage of fetal and early child development and mitigate the longer-run consequences of poverty and health shocks on lifetime health status, productivity, and longevity.³⁴

7. MACROECONOMICS OF FERTILITY AND DEVELOPMENT: DIMINISHING RETURNS TO LABOR, HUMAN CAPITAL AND SAVINGS, AND CHANGING AGE COMPOSITION

Several lines of macroeconomic reasoning are used to infer how declines in fertility during the demographic transition could affect economic growth and development. First, there was Malthus (1789), who relied on the classical economic idea of diminishing returns to labor when workers are employed with a fixed supply of complementary resources, such as agricultural land. The high fertility in low-income countries in the first two decades following the Second World War was viewed in a Malthusian framework as an impediment to economic development. Population growth increased in these poor countries from 0.5% per year in 1900, to 1.2% by 1940, and doubled again to 2.5% by 1960 (Kuznets, 1966; United Nations, 2003). Although due to reduced mortality rather than increased fertility, this “population explosion” appeared to overwhelm the capacity to accumulate capital and employ productively such rapidly growing populations. A demographic poverty trap could arise as Malthus had hypothesized, and slow economic development in these poor countries unless fertility declined quickly (Coale & Hoover, 1958).

However, the Malthusian link between rapid population growth and slower economic development was not evident to Kuznets (1967) from his analysis of historical data, nor did Malthus’ forecasts materialize in subsequent decades, as savings rates increased in many parts of the developing world, and human capital formation and technical change increased total factor productivity and achieved unprecedented growth in output per worker (Johnson, 1999; National Research Council, 1986). Technical change and possibly behavioral responses to the decline in mortality may have outweighed the diminishing returns to labor foreseen by Malthus, allowing growth in *per capita* income in Latin America and Asia. Even the crude associations across countries between population growth rates and growth rates in availability of calories *per capita* are insignificant until 1985, and only thereafter from 1985 to 1995 is the overall association negative. The growing scarcity of food supplies recently in countries with rapid population growth appears to be explained by declining net imports of food, whereas domestic crop production has continued to outpace population growth (Kravdal, 2001).³⁵

The second macroeconomic framework used by economists to assess the implications of the fertility decline focused on a life-cycle pattern of consumption and savings. It was expected that high fertility would depress aggregate rates of savings and thereby discourage economic growth, because the proportion of the population in their most productive ages would not increase until fertility began to fall secularly. In a classic formulation of the issue by [Modigliani and Brumberg \(1954\)](#), they assumed that the marginal utility from consumption diminishes as the level of consumption increases, lifetime utility is time separable, and the age profile of adult productivity rises and falls over the life cycle. Adults in this setting would be motivated to accumulate savings in their most productive periods to sustain their consumption in old age when their productivity declines. These widely accepted assumptions led to the hypothesis that savings rates would rise and fall for a cohort over its life cycle. Holding factor productivity constant, the decline in fertility would contribute over time to an increase in the proportion of the population between the ages of 35 and 55, and thus to an increase in the average national savings rates, all else equal. Some studies have reported within East Asian countries a relationship over time between age composition changes and savings rates changes that could have contributed to the Asian miracle. But this relationship is fragile and evaporates when lagged savings is not included as an exogenous regressor or it is treated as endogenous, or country-specific time trends in savings rates are included in the estimated model ([Higgins & Williamson, 1997](#); [Schultz, 2004a](#)). Counterexamples are also notable. Savings rates have stagnated in other regions, such as Latin America, even though the region experienced a relatively early demographic transition which generated the changes in age compositions as observed in East Asia. In a country such as India, in which the demographic transition has been more gradual and the changes in age composition more modest, savings rates have nonetheless increased ([Schultz, 2004a](#)). There are also inconsistencies between micro- and macroevidence. Microeconomic studies of household surveys do not find the pronounced life-cycle variation in savings rates in either high- or low-income countries as postulated by Modigliani ([Deaton & Paxson, 1997](#)). Although the life-cycle model of consumption and savings behavior remains a plausible conceptual framework for studying many macroeconomic issues, it does not provide a satisfactory explanation for savings and growth in terms of changes in the age composition of low-income countries.

Labor force participation rates for males also rise and fall with an individual's age in many populations, leading to the expectation that labor supply *per capita* would tend to rise as fertility

falls, for at least three decades into the demographic transition. In the longer run, 40 or 50 years after fertility begins to decline, the share of the male population in the labor force is expected to decline, as the fraction of the population over age 50 increases more than the fraction of children falls, as illustrated in the rapid aging of contemporary Japan. This expectation is reinforced as the age-specific death rates among the elderly continue to decline.

However, most of the increase in labor supply per adult following the demographic transition in East Asia and Latin America is due to the increased market labor force participation of *women*, whose participation profiles by age differ across countries and changes over time. The female participation rates outside of the family in the wage labor force shows a general tendency to increase with women's education and the level of development in a country (Durand, 1975; Schultz, 1990). It is also likely that the women who enter the market labor force are also those who bear fewer children, and who are thus able to engage more readily in economic activities outside of their household. The underlying factors changing fertility may thus be responsible for changing a variety of other family coordinated productive behaviors, including most centrally women's market labor supply. Therefore, changes in fertility and age composition cannot be treated as exogenous causes for change in labor inputs to the market. Both fertility and female labor supply are jointly determined in response to male and female market wage opportunities and nonearned income, among other factors (Mincer, 1963).

Schooling and vocational training are concentrated among youth, and thus if enrollment rates are held constant, these private and social investments in the human capital of youth will increase on a *per capita* basis for a decade or two after fertility starts to decline. Moreover, if the decline in family size encourages parents to invest in more years of schooling on average for their children or in higher quality education, this increase in child "quality" may be an indirect consequence of fertility decline that could favor long-run economic growth. In other words, if parents behave as if the schooling of their children is a *substitute* for the number of children they have, population policies which facilitate the decline in fertility by reducing the cost of birth control can contribute to the formation of more human capital as explored in more detail at the microlevel in the next section. A model is required to extrapolate from population policies that reduce fertility to assess how they modify other lifetime family resource allocations, notably the reallocation of the time of family members, and the investment in human capital and various forms of physical capital. The country-level correlations between age composition and economic

growth do not clarify the causal pathways from the resources and opportunities of families, to their health outcomes and fertility, which drive changes in the age composition of the population. There is no reason to assume that age profiles of endogenous household behavior, such as savings or female market labor force participation, will remain unchanged when fertility declines during the demographic transition. Depending on whether the behavior complements or substitutes for fertility, the behavior may decrease or increase, respectively, and these evolving forms of life-cycle behavior may contribute to economic development or stagnation. The decline in fertility is also generally associated with increased schooling and in particular women's postschooling investment in career skills that will modify systematically the age profile of market earnings for women and men, and potentially spill over to affect savings and consumption behavior of their families.

An alternative research agenda to the study of patterns across and within countries seeks to understand how *individual* household behavior responds to arguably exogenous changes in the individual and family environment, such as changes in relative prices, wages of women, men, and children, returns to schooling and other human capital and other asset, and technical change, which may be biased toward better educated labor (Mincer, 1963; Schultz, 1981). For example, it might be hypothesized that the decline in fertility is caused by technical change, which raises the returns on human capital, and thereby encourages parents to invest more in their children's human capital. Parents consequently have fewer children, and women increase the share of their adult lives working in activities other than child care (Foster & Rosenzweig, 2007; Galor & Weil, 1999, 2000; Rosenzweig, 1990). The intercorrelations between fertility and these family lifetime time allocations, investments, and outcomes do not describe the causal impact of fertility nor can these intercorrelations be treated as consequences of fertility and used as a reason to promote fertility reducing population policies (Birdsall, Kelley, & Sinding, 2001; Bloom, Canning, & Sevilla, 2002; Behrman, 2001). Much empirical evidence substantiates the view that fertility is subject to choice in many settings and is determined simultaneously with these other family behaviors (Rosenzweig & Wolpin, 1980a,b; Schultz, 1981, 1997). Cross-country regressions are notoriously difficult to interpret as representing causal relationships, because in the case of mortality and fertility they tend to be dominated empirically by poorly understood secular trends, and because mortality and fertility are jointly determined by unobservables at the family level. This suggests that empirical evidence be analyzed at the household level and

organized to identify plausible structure in models by exploiting variation over time as well as across individuals (Moffitt, 2005; Schultz, 2004b).

8. MICROEVIDENCE OF DETERMINANTS OF FERTILITY AND CONSEQUENCES OF POLICY ON FERTILITY

There are few social experiments where individuals or communities are randomly assigned to population programs or policies, such as family planning programs that subsidize the adoption and use of birth control. The consequences of a population policy on fertility may generate long-term indirect or “cross-effects,” in addition to their direct effect on lifetime fertility and the timing of births. These long-term cross-effects of fertility reduction may constitute important social “externalities” affecting the net social cost of these welfare programs (Schultz, 2008a). The concluding part of this section reviews the case of Matlab, Bangladesh, where a social experiment reducing the cost of birth control has been implemented, and the long consequences have been estimated. But to otherwise evaluate these consequences of policy-induced fertility change, an economic model of fertility as a constrained choice may be useful to help specify an instrument for exogenous variation in fertility as a determinant of family cross-effects. For an instrumental variable estimation strategy to be valid, the instrument must be correlated with fertility, but be arguably uncorrelated with the long-run family behavioral outcomes that are hypothesized to be affected by exogenous changes in fertility over the life cycle. The list of likely family consequences of fertility variation includes the health of women, their labor supply, women’s investment in their own human capital relevant to their productivity in the labor market and home, for which the return may change as she gains more technical control over the timing and number of her births. In other words, the simple association (OLS) between the number of births and related family outcomes cannot be interpreted as an estimate of a causal effect, whereas well-specified two-stage (IV) estimate of these long-run cross-effects of fertility may inform policy.

8.1 Income and price effects affecting the quantity and quality of children

Economists have considered a variety of factors potentially responsible for fertility change and in particular the secular decline in fertility since about 1870 in high-income countries, and since about 1965-1985 in a number of low-income countries. Malthus (1798-1830) hypothesized that increasing real wages would encourage youth to marry earlier, and this would lead them to have more children. In other words, as Adam Smith has postulated, marriage and children were normal goods whose demand would increase with income in the absence of price changes.

Becker (1981) proposed the idea that because children were more “time-intensive” to produce by parents than other forms of their consumption, rising real wages would increase the opportunity cost of children and this price effect would offset the income-driven demand for more children.

The idea that parents substitute offspring “quality” or the resource intensity of each of their offspring for the number of offspring is consistent with the influential ideas of Darwin (1859), and is reflected in the work of various social sciences and evolutionary biology (e.g., Dawkins, 1989; Wilson, 1975). In the formulation of Becker and Lewis (1974), the quality and quantity of children are viewed as substitutes in producing a combination of “services” of children that parents value. But Becker and Lewis make the additional assumption that the income elasticity of demand for child quality is positive and larger in magnitude than the income elasticity of demand for quantity of children. Thus, their framework implies that increases in income may be sufficient by themselves to account for the shift of parents toward demanding higher quality children which could, other things equal, reduce their fertility. It offers an explanation for the demographic transition that does not require an exogenous increase in the relative price of children.

Galor and Weil (2000) conclude that increases in income levels alone are insufficient to explain the timing of the decline in fertility or the demographic transition across high-income countries. They also postulate that human capital complementing technical change occurred after 1870, and liberated European countries from Malthusian stagnation. But few empirical indicators are described that govern the timing or character of this “second industrial revolution,” and their theory does not help to forecast the timing of this technical change in low-income countries or how to foster its advancement. Lucas (2002) hypothesizes that social externalities of human capital were especially strong in urban areas of Europe, and urbanization could account for the increase in labor productivity as an increasing share of the population moved to the European cities during the industrial revolution. However, the elevated mortality in urban areas until the

end of the nineteenth century must have also dampened private and social returns to investment in human capital in these rapidly industrializing, but unhealthy, cities, especially in England and Scotland (Tanner, 1982). The unified growth theory that incorporates the outlines of the demographic transition and the human capital revolution remains a challenging field of economics (Galor, 2005). This integrated description of the industrial revolution and demographic transition requires, however, a stronger empirical foundation with more interdependent predictions for empirical study.³⁶

If the fertility decision is made by utility maximizing parents who are altruistic toward their children's consumption, Becker and Barro (1988) show in their intergenerational growth framework that fertility is an increasing function of the long-term real rate of interest and the probability that children survive. More rapid technical change (biased toward human capital) increases child rearing costs, and public transfers to the elderly of pensions and health care reduce the value of surviving children to parents as a means to support their consumption in old age. This dynastic family growth model draws attention to new constraints on fertility and the role of the welfare state, but these constraints may not all be exogenous from the point of view of families or societies.

What leads the state to provide transfers to the elderly? Why might the majority of citizens prefer to receive old-age support from the state, rather than to invest in rearing more of their own children? Is the increase in cost of child rearing for parents a response to the increasing returns to child human capital? What conditions then stimulate this increase in returns to human capital other than a black box of technical change, and why does technical change become a complement to educated labor in the twentieth century, when it appears to have been a complement to unskilled labor in the nineteenth century (Goldin & Katz, 1998, 2008)? How much of the decrease in child mortality is linked to differences in preferences for, or capacity to produce, child health, associated possibly with the increased schooling of mothers?

Many questions are skirted by Becker and Barro (1988) when they assume a priori technical change complements human capital, child mortality declines independently, and the state provide old-age pensions, while their aggregate model depends on importing all of these critical developments from outside of their intergenerational growth model. Mincer's (1963) observation that the gender gap in schooling and wages could be an important factor driving up the opportunity cost of children is lost in Becker-Barro's dynastic model, where there is no

distinction between the schooling, productivity, or productive roles of men and women. The nonunitary bargaining model of household decision making as proposed by Chiappori (1992) may provide a more realistic framework to study the determinants of such behavior as fertility and family labor supply, and to assess their consequences.

More widely accepted is the specific notion that the returns to human capital, or more specifically schooling, began to increase at the end of the nineteenth century, which induced parents to increase their demand for schooling of their children, and led them to substitute away from having a large family. Reliable empirical evidence of the returns to schooling is scarce before the 1940 US Census, with the exception of a census of Iowa conducted in 1915, which asked for information on both income and schooling (Goldin & Katz, 2000). The substantial wage returns to secondary school and higher education observed in Iowa in 1915 have gradually declined according to the subsequent census samples from Iowa for 1940, 1950, and 1960. Earlier insights into returns to skills or schooling relies on US wage data not by schooling but for blue and white collar workers (Goldin & Katz, 1998, 2008). It is reasonable that technological change in the early twentieth century complemented skills or schooling, and raised returns to schooling, especially for women, until the aggregate supply of education in the population caught up to the new aggregate derived demands for these skills. A second wave of increasing returns to higher education emerges in the 1970s, and may again be attributed to a skill bias in global technical change at the end of the twentieth century, adding to the constraints decreasing fertility (Acemoglu, 2002).

Mincer (1963) emphasized that children were produced primarily by women, and therefore any increase in women's wages relative to men's wages would raise the relative cost of child rearing, and encourage women's work outside of the home and discourage fertility, other things being equal. If women's education increased their productivity only in the market labor force, as assumed by Willis (1974), the decline in fertility and increase in women labor force participation could be attributed to an exogenous increase in women's schooling. Although women's schooling has been gradually catching up to men in many parts of the world (Schultz, 1981, 1990, 1995), there is little agreement on the underlying causes for this trend. Technological change may be attributed a role in increasing the share of services in national income, which favors derived demands for traditional female occupations, such as nursing, teaching, and clerical work, and thus to increase women's wage returns to schooling. In a growing number of

countries female wage returns to schooling exceed male returns at the secondary and higher education levels (Schultz, 1995). As the overall gender gap in wages closes, and the schooling of women catches up to that of men, the private opportunity cost of childbearing should increase further, and the decline in fertility may continue, unless the private costs of child care are absorbed by the public sector of the welfare state (e.g., Sweden).

8.2 Family planning policies and fertility: Cross-country relationships

For couples who want fewer children than they would otherwise have, a reduction in the monetary and psychic cost of birth control due to a family planning program is expected to reduce fertility and yield private gains and possible social externalities (Rosenzweig & Schultz, 1985). One approach to measuring variation in fertility that is attributable to policy initiatives that subsidize birth control is to examine information couples provide on surveys on their reproductive goals and behavior. This research strategy might distinguish between the behavioral demand for births and their biological supply through an analysis of what couples say they want and what they have. Pritchett (1974) provocatively explores survey responses regarding desired fertility (DF) and the number of wanted births (WB) to evaluate how responsive actual fertility is to population policies, such as family planning effort (FPE) as measured by Lapham and Mauldin (1984), where these policies are designed to reduce the cost of knowledge of birth control methods, their adoption, and their continued use. He shows that most of the variation across countries in their total fertility rate (TFR) can be accounted for by the DF ($R^2 = 0.91$, $n = 66$) or WB derived from some of the same representative surveys ($R^2 = 0.85$, $n = 47$). Conversely, unwanted fertility, or the fraction of births, which at the time of conception are reported as unwanted, does not explain much of the cross-country variation in TFR. About the same proportion of births are unwanted in high-income countries where contraceptive prevalence is high and family planning services are widely available, as in low-income countries, where in some cases these services are limited and their absence is expected to lead to more unwanted conceptions. These stylized facts lead Pritchett to conclude that family planning services and policies do not account for much of the variation in fertility, and if FPE were to increase in low-income countries from its indexed value of zero to the average level in his sample of countries

(i.e., 31.4), holding desired fertility fixed, this major policy revolution would reduce TFR by only 0.22 to 0.37 births according to his model regression estimates.

Pritchett dismisses the idea that DF or WB could themselves be affected by family planning services, because the survey questions are phrased to focus on an “ideal” family size, as if there were no costs associated with future birth control. However, if some women viewed abortion as a likely means to control their unwanted fertility, DF and WB might be higher than in an environment where effective contraception and sterilization were not widely available or understood as a reliable and safe remedy to avoid excess or ill timed childbearing. In addition, DF and WB are also undoubtedly measured with error in a survey as responses to a hypothetical situation. Therefore, WB and DF should be treated as endogenous choice variables that are potentially measured with error in a model of fertility determination. To estimate their effects on fertility without bias some exclusion restriction is required, or an instrument must be specified that is correlated with WB or DF, but is not correlated with the residual error in the equation predicting TFR. What might this instrument be?

Culture, customs, or religion could lead some women to report wanting more children than they would actually demand because it fit their norms or morals, but these variables would also likely impact actual fertility or TFR. Education of women might strengthen the willingness of women to report wanting fewer children than they had, or admitting more of their conceptions were “unwanted.” But such a variable that is likely to influence DF or WB is expected to explain some of the error in the TFR equation as well. In other words, it is difficult to imagine an identifying variable that affects DF or WB, but does not also perturb TFR, because all three variables are closely interrelated at a subjective level of the couple and may be affected by complex economic constraints and psychological traits. Pritchett’s additional working assumption that DF and WB are unaffected by FPE could also be challenged by specifying an independent variable shocking FPE that does not affect DF and WB. When instruments of this form cannot be proposed to identify the causal channels between constraints and behavioral variables, social scientists might forego estimating structural relationships and estimate instead a reduced-form equation for fertility. How large an effect does the independent shock to FPE actually exert on TFR?

This is the objective of [Schultz \(1997\)](#) based on a cross section of low-income countries in the 1970s and 1980s in the period when FPE scores are periodically reported by the Population

Council (Lapham & Mauldin, 1984). Family planning program subsidies by the state or nonprofit organizations tend to increase, however, when a substantial proportion of local women demand these program services to control their “excess” fertility, or where $(TFR-DF) > 0$ (Rosenzweig & Schultz, 1985). One way to begin to deal with the endogeneity of FPE in a country is to construct data in the form of a panel of cross sections, and regress differences over time in TFR on differences over time in FPE within countries and other conditions affecting reproductive demands over time, which sweeps out the effects of unobserved time-invariant characteristics of a country that might be associated with excess fertility and result in response in terms of population policy. This first-differenced specification of the fertility model also reduces the problem of measuring fertility in a cross section of age groups of women, summarized by TFR, rather than by measuring the more appropriate lifetime fertility in the form of children ever born for a birth cohort of women at the end of their childbearing years. When this first-differenced model is estimated from a panel including observations for 1972, 1982, and 1988, the estimated effect of change in FPE on change in TFR is not statistically significant (Schultz, 1994, 1997, Tables 8 and 9).

Alternatively, if FPE is treated as an endogenous policy variable, as it should be, the supply of international donor funding for domestic family planning in each low-income country (planned parenthood federation expenditures per woman of childbearing age) can be treated as an exogenous subsidy for FPE in the first-stage estimates, and the second-stage least squares estimate of this source of variation in FPE on TFR is also insignificant, though the donor subsidy is significantly related to FPE in the first-stage equation. In 1 year, 1988, the local price of contraceptive pills is available for 58 low-income countries as one objective indicator of FPE, and the estimated reduced-form price elasticity of TFR is 0.3, but it is not statistically significant ($t = 1.51$). However, when the child mortality rate is also included as an endogenous determinant of TFR, identified by the available *per capita* supply of food calories within a country, the contraceptive price elasticity of fertility decreases to 0.05, but is now measured somewhat more precisely ($t = 1.70$). These estimates (Schultz, 1997, Table 7) are consistent with Pritchett’s conclusion that the effects of FPE on fertility are probably small across all available countries. But using DF or WB, as if they were exogenous parent demand-driven variables in a cross-country regression does not recover the causal relationship between FPE and TFR as claimed by Pritchett. Nor does it provide a satisfactory basis for inferring the consequences of independent

variation in population policies on fertility in an average country. Using outside donor assistance for family planning programs or the prices of contraception suggests that the impact of FPE on TFR is modest, if the policy variable, FPE, is treated appropriately as endogenous and identified by the international population assistance. Unfortunately, cross-country regressions are a blunt tool for deriving policy insights in this case, except under exceptional circumstances. More promising are analyses at the level of the family or community based on the increasing number of public use samples from censuses and surveys.

8.3 Estimating the cross-effects of fertility variation on family lifetime outcomes

At the individual level the two instruments used most commonly to represent exogenous fertility variation are twins and the sex composition of initial births (Schultz, 2008a). Twins are reasonably interpreted as an exogenous shock to fertility that may be more or less uncorrelated with many sources of economic and preference variations affecting other family outcomes. The main limitation of interpreting twins as simply one “unwanted” birth is that twins impose on their parents two additional burdens. First, there is no spacing of the two births which is not likely to be optimal for most couples and may thus impact negatively other family outcomes. Second, and twins tend to be of lower birth weight, health and cognitive performance compared to singleton births, or in other words, twins are on average lower quality births, at least initially. Subject to these limitations on the validity of twins as an instrument for “undemanded” singleton births, one finds that OLS estimates of the partial association of fertility on various family outcomes, for example, women’s labor supply, her health, or the human capital of her other children, etc., tends to be an absolutely larger magnitude than the unbiased IV estimate of fertility’s effect on the same family outcomes, where the IV “treatment” is a twin on first birth, or the ratio of twins per completed pregnancy. If the IV estimate is smaller in absolute magnitude than the OLS estimate, it suggests that a preference for quality tends to be inversely correlated with fertility, and omitting this family control variable from the OLS estimate of fertility’s effect on family outcomes contributes to an omitted variable upward bias (in absolute magnitude) (Schultz, 2008a). Part of the difference between the IV and OLS estimate of the effect of fertility

on family outcomes could also be due to the intrinsic differences between twins and one extra singleton birth.

As described earlier in [Section 4.3](#), Rosenzweig and Zhang (2009) analyze twins and singleton births surveyed in China to estimate the effects of exogenous variation in family size due to a twin on their child quality, measured by various indicators of schooling and health. According to their model, they first adjust for the lower initial endowments of twins, given the estimated propensity of their Chinese parents to reinforce the initial endowments of their children. They then estimate the quantity-quality tradeoff by contrasting the twin instrumented family size effect, depending on whether the twin occurred on the first or second birth. The sacrificed child quality is larger, as suggested by their model, when the unwanted fertility arises because of a twin on the second birth providing the parents with fewer opportunities in the future to compensate for the additional child.

A less satisfactory instrumental variable for fertility is the sex composition of initial births as proposed first in the study of third- and higher-order births in the United States ([Angrist & Evans, 1998](#)). They report that couples with a boy and girl are significantly less likely to have a third birth, and using this information on sex composition of the first two births of parents in the United States as their IV for later fertility, they obtain IV estimated effects of fertility on family outcomes that are smaller in absolute magnitude than the OLS estimates of the partial association. Although this instrument is determined for the most part randomly, the fertility response to the IV varies by the strength of parent preferences for having both a boy and a girl. In some low-income countries parent preferences for the sex of their offspring may differ notably across cultures and could change over time with development. In some countries where daughters are provided a dowry to marry and sons are paid a dowry, family lifetime wealth is directly affected by the sex composition of births ([Rose, 2000](#)). The sex composition of births used as an instrument for exogenous variation in fertility is then no longer uncorrelated with other family lifetime constraints or preferences. These IV estimates based on sex composition of initial births are, therefore, invalid estimates of the effect of exogenous variation in fertility as would arise from the implementation of most population policies, such as sex education, family planning, or reproductive health programs. Additional studies using different instruments for fertility and its consequences on family outcomes are reviewed elsewhere ([Schultz, 2008a](#)).

Recent technological developments have also begun to undermine the validity of both twins and sex composition of initial births as instruments for exogenous variation in fertility or population policies, although analyses of birth and family histories from earlier periods are not affected. Drug therapies to assist women in controlling ovulation and conception have had the side effect of increasing the incidence of multiple births in such countries as the United States. Therefore, the occurrence of twins has become correlated with parent demands for births and couple fecundity in complex ways, and may also be associated with family wealth since infertility treatments maybe relatively costly and not reimbursed under health insurance. Thus, twins born after about 1980 in high-income countries are a less satisfactory basis for estimating the effects of fertility.

Techniques to test for the sex of the fetus at an early stage in a pregnancy by means of ultrasound, amniocentesis, or chorionic villus sampling have allowed a growing share of couples who have sufficiently strong preferences for the gender of their child to abort a fetus of the unwanted sex. As this practice occurs more widely, the sex composition of children becomes correlated with the couples' reproductive preferences and other family choice outcomes, and the sex of initial births ceases to be a valid instrument for estimating the effects of exogenous variation in fertility. The increasing ratio of males to female births at each successively higher parity in countries such as China, Korea, and portions of India reflects this pattern (Schultz, 1997, 2008a).

Another way to distinguish exogenous variation in fertility is to recover a measure of biological heterogeneity in terms of couple fecundity, by assuming more structure for the reproductive process, and then predicting the choice of contraceptive practice during each reproductive cycle. The couple's latent fecundity is inferred by comparing the couples conception rate and the conception rate expected based on their predicted contraceptive practice and consistent estimates of the reproduction function (Rosenzweig & Schultz, 1987). This "residual" variation in fertility, which includes biological fecundity, is associated with having borne more (or less) children, leads to adopting more effective contraceptive methods presumably to compensate for higher fecundity, having lower birth weight children, and providing them with less schooling. These structurally estimated effects of exogenous residual fertility on child quality outcomes are again smaller in absolute value than if they are estimated from the partial association (OLS) effect of fertility on child quality.

8.4 A social experiment in family planning and reproductive health: Bangladesh

One family planning, maternal and child health program was designed as a social experiment in a remote rural area of Bangladesh, in the Matlab Thana. It was initiated in half of 141 villages for which there was already in place a reliable demographic surveillance system of the population, registering all births, deaths, marriages and population movements. The family planning program outreach effort was started in October 1977, which contacted in their homes all married women of childbearing age every 2 weeks, offering them various methods of birth control. The populations were periodically censused and then randomly sampled in a comprehensive socioeconomic survey in 1996. A census in 1974 confirms that the program treatment and comparison villages did not differ significantly 3 years before the program started in terms of their surviving fertility, approximated by the village ratio of children age 0-4 to women age 15-49. A difference-in-difference change between the program and comparison villages preprogram and postprogram indicates that by 1982 surviving fertility is 17% lower in the program areas, and remained 16% lower in the 1996 survey after the program was in operation for nearly two decades. Fertility is lower in the program areas only for women less than age 55, presumably because women over 55 were over 37 in 1977 when the program started, and these older women had essentially completed their childbearing at that time and hence their fertility did not respond to the program treatment (Joshi & Schultz, 2007).

With this social experiment, it is possible to estimate that women age 25-55 in 1996 had about one child less in the program villages compared with the comparison villages. These women in program villages were healthier measured by their BMI being 1.0-1.5 units higher than in the comparison villages, and their children experienced a death rate by their fifth birthday which was 25% lower in the program villages. The girls age 9-14 and 15-29 had obtained about one-third of a standard deviation more years of schooling for their age and sex in the program areas, whereas the boys had obtained about half a standard deviation more schooling. The estimated program effect on the boy's schooling was statistically significant at the 5% level, whereas this schooling effect was not significant for girls. On the other hand, girls age 1-14 were reported to have a significantly higher BMI in the program villages, normalized for age, whereas there was no significant difference in BMI for boys (Joshi & Schultz, 2007). Women age 25-54

in 1996 report log monthly earnings a third higher in the program villages compared to the other villages, and the households in which women reside have proportionately more financial, agricultural, nonagricultural, and housing assets, more consumer durables and jewelry and household tube wells in the program villages (Schultz, 2008b). The prediction of Malthusian diminishing returns to labor is not supported by the Matlab social experiment, in the sense that the wages of young men and women, age 15-24, are not higher in the program villages despite the tendency for there to be fewer children in these villages and they were more likely to attend school. Finally, the wage rates for adult males age 25-54 are no higher in program areas than in the higher fertility comparison villages, whereas in contrast the wages of adult women were at least one-third higher. Moreover these wage patterns are not affected when the selection of who participates among youth and adults into the wage labor force is jointly estimated with the wage function, allowing for the heterogeneity of workers. These findings suggest that women in the program villages who are likely to have had fewer unwanted births due to the efforts of the outreach program have also enjoyed disproportionate gains in their health, market earning opportunities, and household assets compared with comparable women in the comparison villages.

This single case study of the long-run consequences of policy-induced voluntary reductions in fertility suggest that in this poor rural South Asian region, a concerted outreach program achieved a significant decline in fertility and sustained lower levels of fertility for two decades, during which fertility has declined substantially in both groups of villages. This policy-induced reduction in fertility and allied improvement in child and maternal health is associated with women and children being in better health, sons receiving more schooling, and women earning proportionately more in the paid labor market, and living in households with proportionately greater assets. Whether these estimates of population policy effects would exist in other parts of the world remain an unexplored issue, because social experiments or even matching evaluation studies of family planning, reproductive health, and child health programs are rare. Multivariate regression studies of individual and community data suggest that where family planning programs have been expanded more rapidly, birth rates, especially among older women, have declined more rapidly. Evidence from such countries as Taiwan, Thailand, Indonesia, and Colombia are reviewed elsewhere (Frankenberg, et al. 2003; Schultz, 2008a). There are, nonetheless, indications that the effectiveness of national programs to reduce fertility diminish as

they expand to serve all parts of a country, and birth control technology becomes widely understood and birth control supplies are competitively available in the private and public sectors.

9. INTERNAL MIGRATION, DEMOGRAPHIC TRANSITION, AND EVALUATION OF POLICY

Internal migration is a major force redistributing the population during development as sectoral composition of the economy and the geographic distribution of employment change (Kuznets, 1966). This has a variety of implications for the evaluation of population policies. Assessing the impact of programs and policies are often based on interregional differences in program availability, generosity, or eligibility (in fact or in law) which may depend on residence. Implementing policy evaluations therefore frequently requires the joint analysis of migration and outcomes targeted by the program (Rosenzweig & Wolpin, 1986; Schultz, 1988b).

The regional allocation of programs, the intensity of those programs, and their effectiveness in achieving their objectives are likely to be nonrandom, due to many features of policy planning and political economy. Thus, if local program treatments are not administered as a social experiment such that they are allocated independently of the population's endowments and conditions, direct inferences from cross-sectional data of the effect of the program treatment on behavior and outcomes of the treated may mismeasure the local area treatment effect (LATE) on the average member of the population (Imbens & Angrist, 1994; Rosenzweig & Wolpin, 1986). Programs may be allocated to population groups which are thought to be most in need of the program's services, and therefore failure to control for these preprogram conditions could lead to an underestimate of the program's impact based only on postprogram variation across regions. Alternatively, the allocation of the program may favor those groups who are most likely to change their behavior due to the program, in order to maximize the program's demand for services. This scheme of program allocation would tend to lead to an overestimate of the program's effect on the average population. When controls for preprogram conditions and behavior are included in the analysis of program impacts, or the analysis focuses on difference-in-differences between the changes in behavior between the program and control areas over time, these nonexperimental program evaluations are likely to be more reliable.

Interregional migration changes the composition of regional populations, adding another form of potential bias to conventional cross-sectional estimates of the response of a representative individual to regional variation in program treatment. There are many reasons in different environments for the regional access to population programs to differ. Most social welfare programs are better funded and more accessible in urban than in rural areas, and one reason for migration from rural to urban areas is to improve individual and family access to such social services, such as health, schooling, and family planning, and in some settings personal security and protection of private property. If populations are heterogeneous in their preferences for these population and social service programs, as well as other features of the local economy and its amenities, it is expected that those individuals who decide to migrate from rural to urban areas will on average attach greater value to these improved urban social services than would the average of potential migrants. This positive correlation between the probability of migration and the preferences for urban (destination) services will lead to a disproportionate number of rural-urban migrants in urban areas who demand the services provided in their destination areas. This correlation can bias up the regionally estimated “effect” of the program treatment on the demand for, and use of, program services. The problem is likely to be more serious where the average fraction of migrants in the population is larger, as in Latin America after the 1970s, or where lifetime mobility is greater, or mobility is not impeded across regions by languages and ethnically diverse cultures. One diagnostic check on evaluation assessments is to estimate program effect on outcomes for the sample of only natives (i.e., nonmigrants living in their region of birth), and separately for in-migrants. The native estimates that exclude the migrants provides under certain assumptions a lower bound on the local average treatment effect (Duflo et al., 2008; Imbens & Angrist, 1994). Even when the design of regional variation in program treatments is randomized, comparisons over time for a panel sample may still be subject to selective migration, and also involve panel attrition in part due to migration and nonresponse (Fitzgerald, Gottschalk, & Moffitt, 1998).

Regional differences in the relative price of different types of food may be persistent, due to variation in local climates and agricultural potential, which engenders regionally heterogeneous patterns of food consumption. In this case, these patterns of taste for locally plentiful and lower cost foods would deter certain paths of interregional migration. Without recognizing these origin-determined tastes the apparent gains in migrant real income would be overstated, at least

until migrants adopted the tastes for food favored at their destination. [Atkin \(2009\)](#) estimates the magnitude of this regional taste effect on Indian migrant consumption and welfare.

Because education of individuals is widely observed to be positively correlated with the probability of migration between regions, or the likelihood of having moved from one's birthplace ([Schultz, 1982, 2003](#); [Schwartz, 1976](#)), a family's investment in the schooling of children increases the likelihood that the child will migrate, other things being equal. A dynamic structural model is required to disentangle the consequences of education and migration. It is difficult to assess how migrant and native differences arise, though they tend to be notable in terms of adult health, labor market productivity, income, fertility, and even investments in the health and schooling of the children of immigrants and natives at destination.

The composition of internal migration in different parts of the world differs in terms of the sex composition. In North America men were initially the first to migrate toward the Western frontier territories and women followed. In the twentieth century with the frontier closed in the United States, women were more likely to migrate from the rural to urban areas and find employment outside of agriculture. Similar gender patterns in migration are later evident in Latin America, with the exception of indigenous populations where women's migration is restricted by their lack of education, limited knowledge of Spanish, and possibly other cultural constraints. For example, in Colombia and Brazil men dominated in the stream of migration toward rural frontiers, such as the Amazon basin, whereas the probability for women to migrate from the agricultural sector to towns and cities was larger than for men.³⁷ The reversal in the gender ratio of migration from rural to urban areas between the Americas and South Asia and Africa may be explained in large part by the greater levels of schooling of women relative to men in the Americas compared with South Asia, Africa (except for Southern Africa), the Middle East, and West Asia. The culture and family systems that provided girls and boys with similar levels of education in the Americas also facilitated the movement of women to the cities where they could earn more income and provide their children access to better educational opportunities than in the rural areas ([Schultz, 1988b](#)). Evaluation of the effects of social and population programs, especially those targeted to the needs of women and children, require a model of migration which can simultaneously account for the contribution of the programs to female and male productivity, fertility and family health, as well as the migration.

In many South Asian settings where women's education and mobility are more limited than men's, women marry at a relatively young age, and they can migrate to urban areas only in the company of their husbands. The effect of migration or its limitations on women's economic mobility has profound effects on gender differences in economic and demographic behavior and their persistence in different regions. In Latin America migration of women and men from rural areas to towns and on to cities occurs when their schooling is completed, and marriage often occurred after migration, if at all, with many women establishing themselves first in the urban labor force, working in some instances as a domestic servant. Although it is not well understood how policies influence these sources of gender inequality and migration, a few examples are cited in the literature. Bangladesh implemented a secondary school scholarship program for girls, which appears to have contributed to the closure of the initially large gender gap in primary and secondary schooling (Arends-Kuenning & Amin, 2004). This promotion of educational opportunities for girls, especially in rural areas of Bangladesh, has been more recently reinforced by the expansion of urban employment of young women in industries exporting textiles and apparel. Basic education of women prepared them for migrating from their rural villages to work in urban factories, living in dormitories, and remitting much of their earnings to their parents until they marry, at a much later age than did their mothers.

In South East and East Asia, women also migrate to the cities to work in export-oriented industries, but the gender gap in education was smaller than in most South Asian countries and the overall level of schooling was higher. In Taiwan, for example, older married women in rural areas found work in rural-based industries. They thus contributed to their family's income, without having to migrate and incur the higher costs of living in urban areas (Brinton, Lee, & Parish, 1995). Circular migration in other areas of SE Asia, such as Malaysia, allowed men in rural areas to migrate to find temporary urban employment, while returning to help with family farm work during the peak agricultural seasons of planting and harvesting. Their families thus avoid the high urban costs of housing and moving their entire family to coreside with their urban jobs. Many development policies, restrictions on international trade, and factor subsidies combine to create the incentives for different migration patterns which affect the reallocation of the labor of family members from rural household production activities to urban jobs and off farm employments. In the Middle Eastern region women have made progress in obtaining more schooling, reducing their child mortality and fertility, but not finding employment in the formal

labor market. The public sector in some settings provides women with more favorable employment opportunities (Panizza, 2003). Yet as globalization has increased privatization and reduced the restrictions on international trade, growth of the public sector has slowed. Reciprocal expansion of private exporting firms has provided in some countries more jobs for women, as is documented in Turkey (Ozler, 2000; Wood, 1995). But even in the period since 2000 when employment has grown rapidly in the Middle East and North Africa, unemployment rates have remained relatively high for women compared with that for men, and this “underutilized” supply of women’s labor is greater for better educated women (Nabli, Silva Fauregui, & De Silva, 2007).

10. CONCLUSIONS AND POLICY DIRECTIONS

Population policies seek to reduce mortality, morbidity, to produce a longer disability free life, to reduce the inequality in health, and to provide couples with cost-effective techniques for birth control. Internal migration within countries is viewed here as another form of individual behavior which is coordinated with fertility, health status, and the balance of geographically distributed resources and population. Migration complicates the task of evaluating the effects of population policies on family behavior and lifetime outcomes, because migration is another form of human capital investment that responds to similar observed and unobserved determinants of health and control of fertility, and probably complements both.

Many lines of research indicate that environmental conditions existing at the time and location of the conception and birth of a child are related to the child’s length of gestation, height and weight at birth, and these initial endowments of the infant help to explain early child health, and remain significantly associated with the child’s health status as an adult, adult height, IQ, achievements in school, labor market productivity, reduced late age mortality, and delayed onset of chronic health limitations, which permit labor force participation at later ages, and improve well-being. These often delayed consequences of the latent health capability arising from ample fetal development and birth weight for gestational age (i.e., fetal growth rate) suggest the difficulties researchers face if they are to understand precisely how health human capital is produced efficiently and is accumulated equitably over a generation.

Then the question arises how health is potentially transmitted through initial health endowments, both genetic and reproducible, and the direct transfer of economic resources and care to the next generation? Understanding how parents respond to the initial endowments of their children and health shocks they experience over their life is only beginning to be explored by comparisons of siblings and twins. Are parents compensating and thus reducing the final inequality in health or productive outcomes among their offspring, or do they reinforce these initial allocations? What are appropriate fiscal and institutional roles of the state to encourage efficiently these early forms of investment in child health and development? How does this health human capital generate productive and consumption returns to the individual, and where are the most valuable spillovers which improve the lives of other family members and society more generally? In sum, how does policy-induced variation in health human capital and fertility control enhance total factor productivity? This opens an extensive field for research where the existing stock of survey and census data can support some explorations, while data collection efforts and social experiments may be designed to shed light on unresolved questions.

Cross-country macroeconomic comparisons do not seem well designed to add to our understanding of the specific pathways through which technological change, medical inputs, consumption patterns, and health-related behaviors might be channeled by health insurance, or suitably subsidized by governments and NGOs to improve health outcomes, and enhance in a cost-effective manner the productive potential and well-being of populations. Such changes in economic potential related to health improvements parallel closely economic development, but because of the long lags between fetal growth and child development and adult productivity, the connections are only beginning to be persuasively documented, and then only at the microeconomic level of the individual and intergenerational family. One potential reason to pursue aggregate comparisons is that they could in the future be better focused to shed light on the productive spillovers of health status and externalities of fertility declines that are not captured in individual output and wages. These externalities might otherwise be overlooked in conventional microeconomic studies of households, yet could be captured in aggregate data for communities, health administrative areas, service areas for clinics and hospitals, or subnational states. Some preventive health measures and efforts to control infectious diseases may constitute an important public good with significant social externalities, but empirical measurements of these externalities are rare for either health or fertility. Policy-induced declines in fertility

attributable to subsidies for birth control and reproductive health services may also be linked at the individual level to improvements in women's lifetime productivity and intergenerational gains in the health, nutrition, schooling and migration of their children. Convincing empirical studies estimating the magnitude of these spillovers of population policies on health and fertility would be valuable for setting priorities among programs in different localities, and assessing the effectiveness of alternative program designs.

A tentative review of the empirical evidence suggests that higher priority should be assigned to prenatal and reproductive health care for women, and to programs that prevent childhood and adult infectious diseases and control their spread. Some of these health programs can be relatively effective for their cost and yet are underutilized by the uneducated, poor, rural segments of the world's population, where related health problems are most severe. Childhood immunizations and those provided to pregnant women, such as tetanus toxoid, are often neglected. For example, in rural India the prevalence of childhood immunizations has been roughly stable for the last 7 years, a period when incomes have grown rapidly and public and private expenditures on curative health care have increased markedly.³⁸ BMIs of children age 0-3 are low in rural India and have not improved as rapidly for girls as they have for boys, or in rural areas as in urban areas (Tarozzi & Mahajan, 2007). As noted earlier, according to the 2005/2006 National Family Health Survey (NFHS), adult female heights in rural India, an indicator of early childhood health and nutrition, have advanced at a third the rate as those of adult males born from the 1960s to the 1980s (Deaton, 2008). These indicators of initial health conditions in India do not indicate a recent convergence in basic health outcomes. Modest progress is observed in a region such as Latin America, which is otherwise known for its entrenched economic inequality (Schultz, 2005).

The convergence over the last 50 years in life expectation at birth across countries has been interpreted as welfare equalizing (Becker et al., 2005), but the trend has slowed if not reversed since 1990. This is largely due to the AIDS epidemic raising adult mortality in many low-income countries. Is this a special exception to the prevailing pattern of technological diffusion of medical knowledge? Will increasing access to anti retroviral drug therapy reestablish convergence in life expectancy? The cost of curative care to treat noninfectious and degenerative diseases is increasing as the epidemiological transition progresses from high- to low-income countries. Even within high-income countries it is notable that the variance in life expectancy

after age ten, by which time most infectious childhood illnesses have been confronted, has not decreased since 1960 in the OECD, indicating this measure of adult health inequality has not diminished within or between countries (Edwards & Tuljapurkar, 2005). The failure to reduce this measure of the variance in adult mortality may signal that further improvements in health and reduced mortality requires a larger redistribution of income to extend health gains to all socioeconomic groups. Providing low-income countries with cost-effective *curative* care for noninfectious and degenerative diseases may thus be a long-run fiscal and administrative challenge. This fiscal challenge could divert attention from delivering the core preventive child and maternal health measures which may be more cost effective, though apparently not adequately understood or used by a substantial share of the world's poorest populations.

Prevention involves more than the provision of public health services at the community level. It also involves the education of women who manage the household's production of health and nutrition. Women also appear to assign a higher priority to the medical care and feeding of children than do men, given the same household budget constraint (Schultz, 2001). Raising the level of women's schooling may be an efficient means to increase the fetal growth and development of their children, reduce family deaths, and mitigate childhood infections. Are there better ways to improve hygiene, properly use of water, sanitation, and select preventive and curative medical care, and monitor the growth and health status of children (Christiaensen & Alderman, 2004)? The potential links from a mother's schooling to her health and that of her family are not thoroughly assessed and used to coordinate public health strategies. This may be because the schooling of females does not fall strictly in the bailiwick of health ministries, medical systems, programs of public health, water and sanitation engineering, or the disease-specific interventions around which international agencies, such as WHO or the World Bank, organize their programs and marshal their resources.³⁹

The production of health in the household and the diffusion of knowledge to decision makers in the household that could improve the effectiveness of preventive and curative health care are rarely studied. This may be because household behavior relevant to producing health is not integrated into the planning of public medical systems. National plans tend to prescribe the allocation of public expenditures across tiers of institutions in the public sector, from hospitals to clinics, down to local "health posts." But these medical plans often neglect the compensating behavior of the private medical care sector, and the role of privately determined demands of

households for health-related inputs, and for modification in household behavior, including unhealthy behavior such as smoking, alcohol, and substance abuse.

There are many promising avenues for research on the connections between health and development, and fertility and development that would improve our understanding of economic and demographic behavior and the design of policy institutions. This research is conducted through analyses of cross-sectional and panel surveys and censuses for individuals, households and communities, on a country by country basis. These investigations increasingly include the collection of biological markers and health information and migration histories, to better define the causal pathways from the policy instruments to fetal growth to the lifetime adult outcomes of interest to economists and health scientists. Pragmatic and replicable policy and program interventions in the public and private health sectors may thus be documented in the regions where individuals are born, matured, and currently reside. As new programs are implemented experimentally, or at least in a well-documented scaling up of pilot programs by service areas, it should become possible to evaluate the consequences over time of such programs. These assessments will be difficult to perform. Only when the records from the underlying demographic surveillance systems are open to public evaluation by independent researchers, will confidence build as to the strengths and limitations of the data and the validity of evaluation studies they support.

Where the levels of health are lowest in the world, infectious diseases are still a burdensome problem, and reproductive and child health interventions may be most cost effective. Yet these are also the areas where today panel survey data focused on health and economics development are least adequate to the task, and what information is collected is not likely to be open to the research community, outside of the governmental agency who have a weakly defined interest in evaluating the success of their own health programs. In addition, the evaluation and design of health programs requires merging accurate administrative data on program operations, variation in performance incentives, and independent monitoring of local program staff. Finally, a political commitment is required to engage in such organizational variations in the delivery of public health and educational services, the long-run-independent collection of high-quality household sample surveys, and the provision of these merged administrative records and survey data to independent researchers to undertake the difficult task of program evaluation. These conditions are not common, but may be increasing. Evaluation of social programs within development

assistance agencies appears to be receiving a higher priority today, and health and population programs would be a good place to start.

References

Acemoglu, D. (2002). Technical change, inequality and the labor market. *Journal of Economic Literature*, 40(1), 7-72.

Acemoglu, D., & Johnson, S. (2007). Disease and development: The effect of life expectancy on economic growth. *Journal of Political Economy*, 115(6), 925-985.

Acemoglu, D., Johnson, S., & Robinson, J. (2001). The colonial origins of comparative development: An empirical investigation. *American Economic Review*, 91(5), 1369-1401.

Acemoglu, D., Johnson, S., & Robinson, J. A. (2003). Disease and development in historical perspective. *Journal of the European Economic Association*, 1(2-3), 397-405.

Akachi, Y., & D. Canning, 2006, The height of women in Sub Saharan Africa: The role of health, nutrition and Income in childhood, PGDA working paper no. 2207, Harvard School of Public Health, Boston MA.

Alderman, H., & Behrman, J. R. (2006). Reducing the incidence of low birth weight in low income countries has substantial economic benefits. *World Bank Research Observer*, 21(1), 25-48.

Alderman, H., Hoddinott, J., & Kinsey, W. (2006). Long term consequences of early childhood malnutrition. *Oxford Economic Papers*, 58(3), 450-474.

Almond, D. (2006). Is the 1918 Influenza pandemic over? Long term effects of in utero exposure in the post-1940 US population. *Journal of Political Economy*, 114(4), 672-712.

Almond, D., Chay, K., & Lee, D. S. (2005). The cost of low birth weight. *Quarterly Journal of Economics*, 120, 1031-1084.

Almond, D., Edlund, L., Li, H., & Zhang, J. (2007). Long-term effects of the 1959-61 Chinese famine: Mainland China and Hong Kong. Working Paper No. 13384. Cambridge, MA: National Bureau of Economic Research.

Almond, D., Edlund, L., & Palme, M. (2007). Chernobyl's subclinical legacy: Prenatal exposure to radioactive fallout and school outcomes in Sweden. Working Paper No. 13347. Cambridge, MA: National Bureau of Economic Research.

Alters, G., & Oris, M. (2006). Environment before and after birth and mortality in old age. In Annual meetings of the population association of America, Los Angeles, 30 March-1 April, 2006 (also in *Social Biology*, 52(3-4), 178-191).

Angrist, J., & Evans, W. (1998). Children and their parents' labor supply: Evidence from exogenous variation in family size. *American Economic Review*, 88(3), 450-477.

Angrist, J., & Krueger, A. B. (1991). Does compulsory school attendance affect schooling and earnings. *Quarterly Journal of Economics*, 106, 979-1014.

Arends-Kuenning, M., & Amin, S. (2004). School incentive programs and children's activities: The case of Bangladesh. *Comparative Education Review*, 48(3), 295-317.

Atkin, D. (2009). Trade, tastes, and nutrition in India. Unpublished Ph.D. chapter, Princeton University, Princeton, NJ.

Banerjee, A. B., & Duflo, E. (2007). Aging and death under a dollar a day. Working Paper No. 13683. Cambridge, MA: National Bureau of Economic Research.

Barker, D. J. P. (Ed.). (1992). *Fetal and infant origins of adult disease*. London: British Medical Journal.

Barker, D. J. P. (1994). *Mothers, babies, and disease in later life*. London: BMJ Publishing Group.

Barker, D. J. P. (Ed.). (2001). *Fetal origins of cardiovascular and lung disease*. New York, NY: Marcel Dekker.

Barker, D. J. P. (2005). Infant growth and income 50 years later. *Archives of Disease in Childhood*, 90, 272-273.

Barro, R. J. (1997). *Determinants of economic growth: A cross-country empirical study*. Cambridge, MA: MIT Press.

Barro, R. J., & Lee, J. W. (2000). *International data on educational attainment: Updates and implications*. Working Paper No. 42. Cambridge, MA: Harvard University Press.

Barro, R. J., & Sala-I-Martin, X. (1995). *Economic growth*. New York: McGraw-Hill.

Becker, G. S. (1981). *A treatise on the family*. Cambridge, MA: Harvard University Press.

Becker, G. S., & Barro, R. J. (1988). A reformulation of the economic theory of fertility. *Quarterly Journal of Economics*, 103(1), 1-25.

Becker, G. S., & Lewis, H. G. (1974). Interaction between quantity and quality of children. In T. W. Schultz (Ed.), *The economics of the family*. Chicago: University of Chicago Press.

- Becker, G. S., Philipson, T. J., & Soares, R. R. (2005). The quantity and quality of life and the evolution of world inequality. *American Economic Review*, 95(1), 277-291.
- Becker, G.S., & N. Tomes, (1976) Child endowment and the quality and quantity of children, *Journal of Political Economy*, 84, 4, part 2, 159-179.
- Behrman, J. R. (2001). Why micro-matters. In N. Birdsall, A. C. Kelley, & S. W. Sinding (Eds.), *Population matters*. New York: Oxford University Press.
- Behrman, J. R., & Birdsall, N. (1983). The quality of schooling. *American Economic Review*, 73, 928-946.
- Behrman, J. R., & Rosenzweig, M. R. (2004). The returns to birthweight. *Review of Economics and Statistics*, 86(2), 586-601.
- Behrman, J. R., Rosenzweig, M. R., & Taubman, P. (1994). Endowments and the allocation of schooling in the family and in the marriage market: The twin experiment. *Journal of Political Economy*, 102(6), 1131-1174.
- Ben-Porath, Y. (1967). The production of human capital and the life cycle of earnings. *Journal of Political Economy*, 75(4, Part 1), 352-365.
- Bengtsson, T. ,editor, (2009), Early Life effects on socioeconomic performance and mortality in later life, *Social Science & Medicine*, 68, 1561-1564 (entire issue on topics)
- Bengtsson, T., & Brostrom, G. (2006). Old age mortality in a life course perspective: Southern Sweden, 1829-1894. In *Annual meetings of the population association of America*, Los Angeles, 30 March-1 April, 2006.
- Birdsall, N., Kelley, A. C., & Sinding, S. W. (2001). *Population matters: Demographic change, economic growth and poverty in the developing world*. New York: Oxford University Press.
- Black, S.E., P.J. Devereux, & K.G. Salvanes, (2005) The more the merrier? The effect of family composition on children's education, *quarterly Journal of Economics*, 122,1, 409-439.
- Black, S. E., Devereux, P. J., & Salvanes, K. G. (2007). From cradle to labor market? The effect of birth weight on adult outcomes. *Quarterly Journal of Economics*, 122(1), 409-439.
- Bleakley, H. (2007). Disease and development: Evidence from hookworm eradication in the American South. *Quarterly Journal of Economics*, 122(1), 72-117.
- Bleakley, H., & Lange, F. (2009). Chronic disease burden and the interaction of education, fertility and growth. *Review of Economics and Statistics*, 91, 52-65.

- Bloom, D. E., Canning, D., & Sevilla, J. (2002). The demographic dividend: A new perspective on the economic consequences of population change. Santa Monica, CA: The Rand Corporation (MR-12274).
- Bloom, D. E., Canning, D., & Sevilla, J. (2004). The effects of health on economic growth: A production function approach. *World Development*, 32(1), 1-13.
- Bloom, D. E., & Sachs, J. D. (1998). Geography, demography, and economic growth in Africa. *Brookings Papers in Economic Activity*, 2, 207-295.
- Bozzoli, C., & Deaton, A. (2008). Adult height and childhood disease. Princeton, NJ: Center for Health and Wellbeing, Princeton University.
- Brass, W. (1975). Methods for estimating fertility and mortality from limited and defective data. Chapel Hill, NC: Carolina Population Center.
- Brinton, M. C., Lee, Y. J., & Parish, W. L. (1995). Married women, employment in rapidly industrializing societies. *American Journal of Sociology*, 100(5), 1099-1130.
- Buckles, K., & Hungerman, D. M. (2008). Season of birth and later outcomes: Old questions, new answers. Working Paper No. 14573. Cambridge, MA: National Bureau of Economic Research.
- Caldwell, J. C. (1979). Education as a factor in mortality decline. *Population Studies*, 33, 395-413.
- Card, D. (1999). The causal effect of education on earnings. In O. Ashenfelter & D. Card (Eds.), *Handbook of labor economics* (Vol. 3A, pp. 1801-1963). Amsterdam: Elsevier.
- Carson, S. A. (2008). The effect of geography and vitamin D on African American stature in the 19th century: Evidence from prison records. *Journal of Economic History*, 68(3), 812-831.
- Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics*, 24, 365-389.
- Case, A., & Paxson, C. (2008). Height, health, and cognitive function at older ages. *American Economic Review*, 98(2), 463-467.
- Cesur, R., & Rashad, I. (2008). High birth weight and cognitive outcomes. Working Paper No. 14524. Cambridge, MA: National Bureau of Economic Research.
- Chiappori, P. A. (1992). Collective labor supply and welfare. *Journal of Political Economy*, 100(3), 437-467.

- Christiaensen, L., & Alderman, H. (2004). Child malnutrition in Ethiopia: Can maternal knowledge augment the role of income. *Economic Development and Cultural Change*, 52(2), 287-312.
- Coale, A. J. (1972). *The growth and structure of human populations: A mathematical investigation*. Princeton, NJ: Princeton University Press.
- Coale, A. J., & Demeny, P. (1966). *Regional model life tables and stable populations*. Princeton, NJ: Princeton University Press.
- Coale, A., & Hoover, E. (1958). *Population growth and economic development in low income countries*. Princeton, NJ: Princeton University Press.
- Cochrane, S. H., O'Hara, D. J., & Leslie, J. (1980). The effects of education on health. World Bank Staff Working Paper No. 405. Washington, DC: World Bank.
- Costa, D. (1996). Health and labor force participation of older men, 1900-1991. *Journal of Economic History*, 56(1), 62-89.
- Costa, D. L., & Steckel, R. H. (1997). Long-term trends in health, welfare, and economic growth in the United States. In R. H. Steckel & R. Floud (Eds.), *Health and welfare during industrialization*. Chicago: University of Chicago Press.
- Crimmins, E., & Finch, C. (2006a). Infection, inflammation, height and longevity. *Proceedings of the National Academy of Sciences of the United States of America*, 103(2), 498-503.
- Crimmins, E. M., & Finch, C. E. (2006b). Commentary: Do older men and women gain equally from improving childhood conditions? *International Journal of Epidemiology*, 35, 1270-1271.
- Cunha, F., & Heckman, J. J. (2007). *The technology of skill formation*. IZA Discussion Paper No. 2350. Bonn, Germany: Institute for the study of labor (IZA).
- Currie, J., & Hyson, R. (1999). Is the impact of health shocks cushioned by socioeconomic status? *American Economic Review*, 89(2), 245-250.
- Currie, J., & Stabile, M. (2003). Socioeconomic status and child health: Why is the relationship stronger for older children? *American Economic Review*, 93(5), 1813-1823.
- Darwin, C. R. (1859). *The origin of species*. London: John Murray.
- Data, A., Gosh, A., & Sood, N. (2007). *Mortality risk, health endowments, and parental investments in infancy: Evidence from rural India*. Working Paper No. 13647. Cambridge, MA: National Bureau of Economic Research.
- Dawkins, R. (1989). *The selfish gene*. Oxford: Oxford University Press.

- Deaton, A. (2003). Health, inequality, and economic development. *Journal of Economic Literature*, 49(1), 113-158.
- Deaton, A. (2007). Height, health, and development. *Proceedings of the National Academy of Sciences of the United States of America*, 104(33), 13232-13237.
- Deaton, A. (2008). Height, health and inequality: Distribution of adult heights in India. *American Economic Review*, 98(2), 468-474.
- Deaton, A., & Paxson, C. H. (1997). The effects of economic and population growth on national savings and inequality. *Demography*, 34(1), 97-114.
- Del Bono, E., Ermisch, J., & Francesconi, M. (2008). Intrafamily resource allocations: A dynamic model of birth weight. Unpublished paper, University of Essex, UK.
- Deolalikar, A. B. (1988). Nutrition and labor productivity in agriculture: Estimates for rural South India. *Review of Economics and Statistics*, 70(3), 406-413.
- Diamond, J. (1997). *Guns, germs and steel: The fates of human societies*. New York: W.W. Norton & Co.
- Doblhammer, G. (2004). *The late life legacy of very early life*. Berlin: Springer-Verlag.
- Doblhammer, G., & Vaupel, J. (2001). Lifespan depends on month of birth. *Proceedings of the National Academy of Sciences of the United States of America*, 98(5), 2934.
- Duflo, E. (2001). Schooling and labor market consequences of school construction in Indonesia. *American Economic Review*, 91(3), 795-813.
- Duflo, E., Glennerster, R., & Kremer, M. (2008). Using randomization in development economics research: A toolkit. In T. P. Schultz & J. Strauss (Eds.), *Handbook of development economics* (Vol. 4). Amsterdam: Elsevier.
- Durand, J. D. (1975). *The labor force in economic development*. Princeton, NJ: Princeton University Press.
- Edwards, R. D., & Tuljapurkar, S. (2005). Inequality in life spans and a new perspective on mortality convergence across industrialized countries. *Population and Development Review*, 31(4), 645-674.
- Eckstein, Z, T. P. Schultz and K. I. Wolpin (1984). Short run fluctuations in fertility and mortality in preindustrial Sweden. *European Economic Review*, 26(3), 295-317.
- Elo, I. T., & Preston, S. H. (1992). Effects of early life conditions on adult mortality: A review. *Population Index*, 58(2), 186-212.

- Eveleth, P. B. (1986). Population differences in growth, environmental and genetic factors. In F. Falkner & J. M. Tanner (Eds.), *Human growth: A comprehensive treatise* (2nd ed., Vol. 3). New York: Plenum Press.
- Eveleth, P. B., & Tanner, J. M. (1990). *World wide variation in human growth* (2nd ed.). Cambridge, UK: Cambridge University Press.
- Field, E., Robles, O., & Torero, M. (2007). *Iodine deficiency and schooling attainment in Tanzania*. Cambridge, MA: Harvard University Press.
- Filmer, D. (2008). Disability, poverty and schooling in developing countries: Results from 14 household surveys. *World Bank Economic Review*, 22(1), 141-163.
- Finch, C.E., (2007), *The Biology of human longevity: inflammation, nutrition and aging in the evolution of lifespan*, Amsterdam: Elsevier
- Finch, C. E., & Crimmins, E. (2004). Inflammatory exposure and historical changes in human life-spans. *Science*, 305, 1736-1739.
- Fitzgerald, J., Gottschalk, P., & Moffitt, R. (1998). Attrition in longitudinal surveys. *Journal of Human Resources*, 33(2), 251-299.
- Floud, R., Wachter, K., & Gregory, A. (1990). *Height, health and history: Nutritional status in the United Kingdom, 1750-1980*. New York: Cambridge University Press.
- Fogel, R. W. (1986). Physical growth as a measure of the economic well-being of populations: The 18th and 19th centuries. In F. Falkner & J. M. Tanner (Eds.), *Human growth: A comprehensive treatise* (2nd ed., Vol. 3). New York: Plenum Press.
- Fogel, R. W. (1994). Economic growth, population theory and physiology: The bearing of long-term processes on the making of economic policy. *American Economic Review*, 84(3), 369-395.
- Fogel, R. W. (2004). *The escape from hunger and premature death, 1700-2100*. Cambridge, UK: Cambridge University Press.
- Foster, A., & Rosenzweig, M. R. (2007). Does economic growth reduce fertility: Rural India, 1971-1999. In S. Bery & A. Panagariya (Eds.), *India policy forum 2006-07*. Delhi, India: Brookings and NCAER.
- Frankenberg, E. (1995). The effects of access to health care on infant mortality in Indonesia. *Health Transition Review*, 5(2), 143-163.

- Frankenberg, E., Sikoki, B., Suriastini, W., & Thomas, D. (2003). Contraceptive use in a changing service environment: Evidence from Indonesia during the economic crisis. *Studies in Family Planning*, 34(2), 102-116.
- Frankenberg, E., Suriastini, W., & Thomas, D. (2005). Can expanding access to basic health care improve children's outcomes? *Population Studies*, 59(1), 5-19.
- Freedman, R., (1975) *The Sociology of Human Fertility*. New York: John Wiley.
- Gallup, J. L., & Sachs, J. D. (2001). The economic burden of malaria. *The American Journal of Tropical Medicine and Hygiene*, 64(1), 85-96.
- Galor, O. (2005). From Stagnation to Growth: Unified Growth Theory, in *Handbook of Economic Growth Vol 1*, editors P. Aghion & S. Durlauf, 171-293, Amsterdam: Elsevier.
- Galor, O., & Weil, D. N. (1999). Malthusian stagnation to modern growth. *American Economic Review*, 89(2), 150-154.
- Galor, O., & Weil, D. N. (2000). Population, technology, and growth: From Malthusian stagnation to the demographic transition and beyond. *American Economic Review*, 90(4), 806-828.
- Gertler, P. (2004). Do conditional cash transfers improve child health? Evidence from Progresa's control randomized experiment. *American Economic Review*, 94(2), 336-341.
- Ginsburg, E., Livshits, G., Yakovenko, K., & Kobylansky, E. (1998). Major gene control of human body height, weight, and BMI in five ethnically different populations. *Annals of Human Genetics*, 62, 307-322.
- Glewwe, P. (2005). The impact of child health and nutrition on education in developing countries. *Food and Nutrition Bulletin*, 26(2), S235-S250.
- Glewwe, P., & Jacoby, H. (1995). An economic analysis of delayed primary school enrollment in a low income countries: Role of early childhood nutrition. *Review of Economics and Statistics*, 77(1), 156-169.
- Glewwe, P., Jacoby, H., & King, E. (2000). Early childhood nutrition and academic achievement: A longitudinal analysis. *Journal of Public Economics*, 81(3), 345-368.
- Glewwe, P., & Miguel, E. A. (2008). The impact of child health and nutrition on education in less developed countries. In T. P. Schultz & J. Strauss (Eds.), *Handbook of development economics Vol. 4*. Amsterdam: Elsevier.

- Gluckman, P., & Hanson, M. (2005). *The fetal matrix: Evolution, development and disease*. Cambridge, UK: Cambridge University Press.
- Gluckman, P., & Hanson, M. (Eds.). (2006). *Developmental origins of health and disease*. Cambridge, UK: Cambridge University Press.
- Goldin, C., & Katz, L. F. (1998). The origins of technology-skill complementarity. *Quarterly Journal of Economics*, 113(3), 693-723.
- Goldin, C., & Katz, L. F. (2000). Education and income in the early twentieth century: Evidence from the Prairies. *Journal of Economic History*, 60(September), 782-818.
- Goldin, C., & Katz, L. (2008). *The race between education and technology*. Cambridge, MA: Harvard University Press.
- Gorgens, T., Meng, X., & Vaithianathan, R. (2007). Stunting and selection effects of famine: A case study of the great Chinese Famine. IZA Discussion Paper No. 2543. Bonn, Germany: Institute for the study of labor (IZA).
- Griliches, Z. (1979). Sibling models and data in economics: Beginnings of a survey. *Journal of Political Economy*, 87(5, Part 2), S37-S64.
- Gruber, J., & Wise, D. (1995). *Social security programs and retirement around the world*. Working Paper No. 6134. Cambridge, MA: National Bureau of Economic Research.
- Gruber, J., & Wise, D. (2004). *Social security programs and retirement around the world: Micro-estimation*. Chicago: University of Chicago Press.
- Hanewald, K. (2009). Mortality modeling: Lee-Carter and the macroeconomy. SFB Discussion Paper No. 2009-008. Berlin: Humboldt-Universität zu Berlin.
- Hazen, M. (2007). Longevity and lifetime labor input: Data and implications. Working Paper. Jerusalem: Hebrew University. SSRN.com/abstract=9419361
- Heckman, J. J. (2008). Schools, skills and synapses. ISA Discussion Paper No. 3515. Bonn, Germany: Institute for the study of labor (IZA). iza@iza.org.
- Higgins, M., & Williamson, J. G. (1997). Age structure dynamics in Asia and dependence on foreign capital. *Population and Development Review*, 23(2), 261-294.
- Holick, M. F. (2004). Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. *American Journal of Clinical Nutrition*, 80(Suppl.), 1678S-1688S.

- Hubler, Olaf, (2006) The nonlinear link between height and wages: an empirical investigation, IZA discussion paper 2394, October. <iza.org/publications/dps/>.
- Imbens, G. W., & Angrist, J. D. (1994). Identification and estimation of local average treatment effects. *Econometrica*, 62(2), 467-476.
- Johnson, D. G. (1999). Population and development. *China Economic Review*, 10(1), 1-16.
- Joshi, S., & Schultz, T. P. (2007). Family planning as an investment in development: Evaluation of a program's consequences in Matlab, Bangladesh. Economic Growth Center Discussion Paper No. 951. New Haven, CT: Yale University Press.
- Kelley, A. C., & Schmidt, R. M. (1995). Aggregate population and economic growth correlations: The role of the components of demographic change. *Demography*, 32(4), 543-555.
- Koenig, M. A., Fauveau, V., & Woityniak, B. (1991). Mortality reduction from health interventions: The case of immunization in Bangladesh. *Population and Development Review*, 17(1), 87-104.
- Komlos, J. (Ed.). (1994). *Stature, living standards, and economic development*. Chicago: University of Chicago Press.
- Kravdal, O. (2001). Has population growth restricted improvements in food availability per head, 1970-1995. *Population Studies*, 55, 105-117.
- Kuznets, S. (1966). *Modern economic growth: Rate, structure and spread*. New Haven, CT: Yale University Press.
- Kuznets, S. (1967). Population and economic growth. *Proceedings of the American Philosophical Society*, 3(3), 170-193.
- Lapham, R. J., & Mauldin, W. P. (1984). Family planning effort and birthrate decline in developing countries. *International Family Planning Perspectives*, 10(4), 109-118.
- Liu, Y. Z., Xu, F.-H., Shen, H., Liu, Y. J., Zhao, L. J., Long, J. R., et al. (2004). Genetic dissection of human stature in a large sample of multiplex pedigrees. *Annals of Human Genetics*, 68(5), 472-488.
- Lleras-Muney, A. (2005). The relationship between education and adult health in the United States. *Review of Economic Studies*, 72, 189-221.
- Lokshin, M., & Radyakin, S. (2009). Month of birth and children's health in India. Policy Research Working Paper No. 4813. Washington, DC: World Bank.

- Lorentzen, P., McMillan, J., & Wacziarg, R. (2008). Death and development. *Journal of Economic Growth*, 13, 81-124.
- Lucas, R. E., Jr. (2002). *Lectures on economic growth*. Cambridge, MA: Harvard University Press.
- Lutz, W., & Goujon, A. (2001). The world's changing human capital stock: Multistage population projections by educational attainment. *Population and Development Review*, 27, 323-339 (see projection database on ASSA website).
- Maccini, S., & Yang, D. (2006). *Under the weather: Health, schooling, and socioeconomic consequences of early-life rainfall*. Ann Arbor, MI: University of Michigan.
- Maluccio, J. A., Hoddinott, J., Behrman, J. R., Martorell, R., Quisumbing, A., & Stein, A. D. (2006). The impact of an experimental nutritional intervention in childhood on education among Guatemalan adults. Working Paper No. 0614. Middlebury, VT: Middlebury College.
- Martorell, R., & Habicht, J. P. (1986). Growth in early childhood in developing countries. In F. Falkner & J. M. Tanner (Eds.), *Human growth: A comprehensive treatise* (2nd ed., Vol. 3). New York: Plenum Press.
- McKeown, T. (1976). *The modern rise of population*. New York: Academic Press.
- McKeown, T. (1979). *The role of medicine: Dream, mirage, or nemesis*. Princeton, NJ: Princeton University Press.
- Mensch, B., Lentzner, H., & Preston, S. (1985). *Socioeconomic differentials in child mortality in developing countries*. New York: United Nations.
- Miguel, E., & Kremer, M. (2004). Worms: Identifying impacts on education and health in the presence of treatment externalities. *Econometrica*, 72(1), 159-217.
- Mincer, J. (1963). Market prices, opportunity costs and income effects. In C. Christ (Ed.), *Measurement in economics*. Stanford, CA: Stanford University Press.
- Modigliani, F., & Brumberg, R. (1954). Utility analysis and the consumption function. In K. K. Kurihara (Ed.), *Post-Keynesian economics* (Chapter 15). New Brunswick, NJ: Rutgers University Press.
- Moffitt, R. (2005). Remarks on the analysis of causal relationships in population research. *Demography*, 42(1), 91-108.
- Murphy, K. M., & Topel, R. H. (2006). The value of health and longevity. *Journal of Political Economy*, 114(5), 871-904.

- Mwabu, G., (2008), Health economics in low income countries, in Handbook of Development Economics vol 4, editors, T.P. Schultz & J. Strauss, Amsterdam: Elsevier.
- Mwabu, G., (2009) The production of child health in Kenya: a structural model of birth weight, *Journal of African Economies*, 18:212-260.
- Nabli, M. K., Silva Fauregui, C., & De Silva, S. J. (2007). Job creation in a high growth environment: The MENA region. Middle East and North Africa Working Paper No. 49. Washington, DC: World Bank.
- National Family Health Survey 3, Indian Government (2007, October 15). Summary Final Report. alerts@measuredhs.com
- National Research Council (1986). Population growth and economic development: Policy questions. Washington, DC: National Academy Press.
- Oeppen, J., & Vaupel, J. W. (2002). Broken limits to life expectancy. *Science*, 296, 1029-1031.
- Ozler, S. (2000). Export orientation and female share of employment: Evidence from Turkey. *World Development*, 28(7), 1239-1248.
- Panizza, U. (2003). The public sector premium and the gender gap in Latin America: Evidence for the 1980s and 1990s. Washington, DC: Inter-American Development Bank (SSRN, id = 238700).
- Parker, S. W., Rubalcava, L., & Teruel, G. (2008). Evaluating conditional schooling and health programs. In T. P. Schultz & J. Strauss (Eds.), *Handbook of development economics*. (Vol. 4). Amsterdam: North Holland Press/Elsevier.
- Preston, S. H. (1975). The changing relation between mortality and level of economic development. *Population Studies*, 29(2), 231-248.
- Preston, S. H. (1980). Causes and consequences of mortality declines in less developed countries during the twentieth century. In R. A. Easterlin (Ed.), *Population and economic change in developing countries*. Chicago: University of Chicago Press.
- Preston, S. H., Hill, M. E., & Drevenstedt, G. L. (1998). Childhood conditions that predict survival to advanced ages among African Americans. *Social Science & Medicine*, 47(9), 1231-1246.
- Pritchett, L. H. (1994). Desired fertility and the impact of population policies. *Population and Development Review*, 20(1), 1-55.

- Pritchett, L., & Summers, L. H. (1996). Wealthier is healthier. *Journal of Human Resources*, 31(4), 841-868.
- Raiten, D. J., & Picciano, M. F. (2004). Vitamin D and health in the 21st century: Bone and beyond. Executive summary. *American Journal of Clinical Nutrition*, 80(Suppl.), S1673-S1677.
- Rose, E. (2000). Gender bias, credit constraints and time allocation in rural India. *Economic Journal*, 110(465), 738-758.
- Rosenzweig, M. R. (1990). Population growth and human capital investments: Theory and evidence. *Journal of Political Economy*, 98(5), S38-S70.
- Rosenzweig, M. R., & Schultz, T. P. (1983). Estimating a household production function: Heterogeneity, the demand for health inputs, and the effect on birthweight. *Journal of Political Economy*, 91(5), 723-746.
- Rosenzweig, M. R., & Schultz, T. P. (1985). The supply and demand of births and their life cycle consequences. *American Economic Review*, 75(5), 922-1015.
- Rosenzweig, M. R., & Schultz, T. P. (1987). Fertility and investment in human capital in Malaysia. *Journal of Econometrics*, 36, 163-184.
- Rosenzweig, M. R., & Wolpin, K. I. (1980a). Testing the quantity-quality fertility model. *Econometrica*, 48(1), 227-240.
- Rosenzweig, M. R., & Wolpin, K. I. (1980b). Life cycle labor supply and fertility: Causal inferences from household models. *Journal of Political Economy*, 88(2), 328-348.
- Rosenzweig, M. R., & Wolpin, K. I. (1985). Specific experience, household structure and intergenerational transfers: Farm family land and labor arrangements in developing countries. *Quarterly Journal of Economics*, 100(Suppl.), 461-487.
- Rosenzweig, M. R., & Wolpin, K. I. (1986). Evaluating the effects of optimally distributed public programs: Child health and family planning interventions. *American Economic Review*, 76(3), 470-482.
- Rosenzweig, M. R., & Zhang, J. (2009). Do population control policies induce more human capital investment? Twins, birthweight, and China's one child policy. *Review of Economic Studies*, 76, 1149-1174.
- Royer, H. (2009). Separated at Girth: U.S. twin estimates of the effect of birth weight. *American Economic Journal: Applied Economics*, 1(1), 49-85.

- Ruhm, C. J. (2000). Are recessions good for your health? *Quarterly Journal of Economics*, 115(2), 617-650.
- Savedoff, W., & Schultz, T. P. (Eds.). (2001). *Wealth from health*. Washington, DC: Inter-American Development Bank.
- Schultz, T. P. (1980). Interpretation of relations among mortality, economic of the household, and the health environment. In *Socioeconomic determinants and consequences of mortality differentials*. Geneva: World Health Organization.
- Schultz, T. P. (1981). *Economics of population*. Reading, MA: Addison-Wesley Publisher.
- Schultz, T. P. (1982). Notes on the estimation of migration decision functions. In R. Sabot (Ed.), *Migration and the labor market in developing countries*. Boulder, CO: Westview Press.
- Schultz, T. P. (1985). Changing world prices, women's wages and the fertility transition: Sweden 1860-1910. *Journal of Political Economy*, 93(6), 1126-1154.
- Schultz, T. P. (1988a). Education investments and returns. In H. Chenery & T. N. Scrinivasan (Eds.), *Handbook of development economics* (Vol. 1, Chapter 13, pp. 543-630). Amsterdam: North Holland Publishing Company.
- Schultz, T. P. (1988b). Heterogeneous preferences and migration: Self-selection, regional prices and programs and the behavior of migrants in Colombia. In *Research in population economics* (Vol. 6). Greenwich, CT: JAI Press.
- Schultz, T. P. (1990). Women's changing participation in the labor force. *Economic Development and Cultural Change*, 38(3), 451-488.
- Schultz, T. P. (1994). Human capital, family planning and their effects on population growth. *American Economic Review*, 83(2), 255-260.
- Schultz, T. P. (1995). *Investment in Women's Human Capital*, editor, Chicago: University of Chicago Press.
- Schultz, T. P. (1997). The demand for children in low income countries. In M. R. Rosenzweig & O. Stark (Eds.), *Handbook of population and family economics* (Vol. 1A). Amsterdam: North Holland Publishing Company.
- Schultz, T. P. (2001). Women's roles in the agricultural household: Bargaining and human capital investments. In B. Gardner & G. Rausser (Eds.), *Agricultural and resource economics handbook*. Amsterdam: Elsevier.

- Schultz, T. P. (2002). Wage gains associated with height as a form of health human capital. *American Economic Review*, 92(2), 349-353 (Economic Growth Center Discussion Paper No. 841, Yale University Press for appendix tables).
- Schultz, T. P. (2003). Wage rentals for reproducible human capital: Evidence from two West African countries. *Economics & Human Biology*, 1(3), 331-366.
- Schultz, T. P. (2004a). Human resources in China: The birth quota, returns to education and migration. *Pacific Economic Review*, 93(3), 245-267.
- Schultz, T. P. (2004b). Demographic determinants of savings: Estimating and interpreting aggregate associations in Asia. Discussion Paper No. 1479. Beijing: China Center for Economic Research. <http://www.iza.org>
- Schultz, T. P. (2005). Productive benefits of health: Evidence from low income countries. In G. Lopez-Casasnovas, B. Rivera, & L. Currais (Eds.), *Health and economic growth: Findings and policy implications*. Cambridge, MA: MIT Press.
- Schultz, T. P. (2008a). Population policies, fertility, women's human capital, and child quality. In T. P. Schultz & J. Strauss (Eds.), *Handbook of development economics* (Vol. 4, Chapter 52). Amsterdam: North Holland Press/Elsevier (Economic Growth Center Discussion Paper No. 954, Yale University Press).
- Schultz, T. P. (2008b). Can family planning programs promote development beyond fertility reduction? Evidence from a long-term social experiment in Bangladesh—1977-1996, presented at a Gates foundation/center for international development conference, on Health Improvements for Economic Growth.
- Schultz, T. P., & Tansel, A. (1997). Wage and labor supply effects of illness in Cote d'Ivoire and Ghana: Instrumental variable estimates for days disabled. *Journal of Development Economics*, 53(2), 251-286.
- Schwartz, A. (1976). Migration, age and education. *Journal of Political Economy*, 84(4, Part 1), 701-720.
- Scrimshaw, N. S., Taylor, C. E., & Gordon, J. E. (1968). *Interactions of nutrition and infection*. World Health Monograph Series No. 57. Geneva: World Health Organization.
- Silventoinen, K. (2003). Determinants of variation in adult body height. *Journal of Biosocial Science*, 35, 263-285.

Silventoinen, K., Kaprio, J., Lahelma, E., & Koskenvuo, M. (2000). Relative effect of genetic and environmental factors on body height: Differences across birth cohorts among Finnish men and women. *American Journal of Public Health*, 90, 627-630.

Steckel, R.H., (1995) Stature and the standard of living, *Journal of Economic Literature*, 33,1903-1940.

Steckel, R. H. (2008). Height and human welfare: Recent developments and new directions. Working Paper No. 14536. Cambridge, MA: National Bureau of Economic Research.

Strauss, J. (1986). Does better nutrition raise farm productivity? *Journal of Political Economy*, 94(2), 297-320.

Strauss, J., & Thomas, D. (1998). Health, nutrition, and economic development. *Journal of Economic Literature*, 36(2), 766-817.

Strauss, J., & Thomas, D. (2008). Health over the life course. In T. P. Schultz & J. Strauss (Eds.), *Handbook of development economics* (Vol. 4). Amsterdam: Elsevier.

Stunkard, A. J., Foch, T. T., & Hrubec, Z. (1986). A twin study of human obesity. *Journal of American Medical Association*, 256, 51-54.

Su, D. (2005). Body mass index and old age survival. *American Journal of Human Biology*, 17(3), 341-354 and presented at the Annual meetings of the population association of America, Los Angeles, 31 March-1 April, 2006. Chicago: Center for Population Economics, University of Chicago.

Summers, R., & Heston, A. (1991). The Penn World Table (Mark 5). *Quarterly Journal of Economics*, 106(2), 327-368.

Sunder, M. (2005). Toward generation XL: Anthropometrics of longevity in late 20th century United States. *Economics of Human Biology*, 3(3), 271-295.

Sunder, M., & Woitek, U. (2005). Boom, bust, and the human body: Further evidence on the relationship between height and business cycle. *Economics & Human Biology*, 3(3), 450-466.

Tanner, J. M. (1981). *A history of the study of human growth*. Cambridge: Cambridge University Press.

Tanner, J. M. (1982). The potential of auxological data for monitoring economic and social well-being. *Social Science History*, 6(4), 571-581.

Tapia Granados, J. A. (2008). Macroeconomic fluctuations and mortality in postwar Japan. *Demography*, 45, 323-343.

- Tarozzi, A. (2009). Growth reference charts and the nutritional status of Indian children. *Economics & Human Biology*, 6(3), 455-468.
- Tarozzi, A., & Mahajan, A. (2007). Child nutrition in India in the nineties. *Economic Development and Cultural Change*, 54(3), 441-486.
- Thirumurthy, H., Zivin, J. G., & Goldstein, M. (2008). The economic impact of AIDS treatment: Labor supply in Western Kenya. *Journal of Human Resources*, 43(3), 511-552.
- Thomas, D., (1990) Intrahousehold resource allocation: an inferential approach, *Journal of Human Resources*, 25,4, 635-664.
- Thomas, D. (2008). Health and socioeconomic status: Isolating causal pathways. In ABCDE World Bank conference, Cape Town, South Africa, 9-11 June 2008.
- Thomas, D., Frankenberg, E., Friedman, J., Habicht, J.-P., Jones, N., McKelvey, C., et al. (2006). Causal effect of health on labor market outcomes: Experimental evidence. Los Angeles, CA: UCLA.
- Tolley, G., Kenkel, D., & Fabian, R. (Eds.). (1994). *Valuing health for policy: An economic approach*. Chicago: University of Chicago Press.
- Topel, R. (1999). Labor markets and economic growth. In O. Ashenfelter & D. Card (Eds.), *Handbook of labor economics* (Vol. 3c). Amsterdam: Elsevier.
- United Nations (1967). *Methods of estimating basic demographic measures from incomplete data, Manual IV. Population Studies No. 42*. New York: Department of Economic and Social Affairs, United Nations.
- United Nations (2003). *World population prospects: The 2002 revision* (Vol. 1). New York: Department of Economic and Social Affairs, Population Division, United Nations.
- United Nations (2004). *World population policies*. New York: United Nations.
- United Nations Fund for Population Activities (UNFPA/NIDA) (2009). *Resource Flows Project Report* (Tables 1-6; 3 March 2009). <http://www.resourceflows.org/index.php/articles/288>.
- Usher, D. (1973). An imputation to the measure of economic growth for changes in life-expectancy. In M. Moss (Ed.), *The measurement of economic and social performance*. New York: Columbia University Press.
- Van den Berg, G., Lindenbroom, M., & Portrait, F. (2006). Economic conditions early in life on individual mortality. *American Economic Review*, 96(1), 290-302.

- Van Wieringen, J. C. (1986). Secular growth changes. In F. Falkner & J. M. Tanner (Ed.), *Human growth: A comprehensive treatise* (2nd ed., Vol. 3). New York: Plenum Press.
- Waller, H. T. (1984). Height, weight, and mortality: The Norwegian experience. *Acta Medica Scandinavica*, 77(Suppl. 679), 1-56.
- Weil, D. N. (2007). Accounting for the effects of health on economic growth. *Quarterly Journal of Economics*, 112(3), 1265-1306.
- Wooldridge, J.M., (2002) *Econometric Analysis of Cross Sectional and Panel Data*, Cambridge MA:MIT Press
- White, K. M. (2002). Longevity advances in high-income countries, 1955-96. *Population and Development Review*, 28(1), 59-76.
- Willis, R. J. (1974). Economic theory of fertility behavior. In T. W. Schultz (Ed.), *Economics of the family*. Chicago: University of Chicago Press.
- Wilson, E. O. (1975). *Sociobiology*. Cambridge, MA: Harvard University Press/Belknap Press.
- Wood, A. (1995). North-South trade and female labour in manufacturing: An asymmetry. *Journal of Development Studies*, 27(2), 168-189.
- World Bank (1993). *World development report 1993*. New York: Oxford University Press.
- World Bank (2008). *Growth report*. Washington, DC: World Bank.
- World Health Organization (WHO) (1995). *Physical status: The use and interpretation of anthropometry*. Technical Report Series No. 854. Geneva: WHO.
- World Health Organization (2001). *Macroeconomics and health: Investing in health for economic development*. Report of the commission on macroeconomics and health. Geneva: WHO. <http://ww3.who.int/shosis/cmh>
- World Health Organization (2003). *Guide to producing national health accounts with special applications for low-income and middle-income countries*. Geneva: World Health Organization
- World Health Organization (2006). *Child growth standards: Length/height for age, weight for age, weight for length, weight for height, and body mass index for age: Methods and development*. Geneva: WHO.
- Xiong, D. H., Xu, F. H., Liu, P. Y., Shen, H., Long, J. R., Elze, L., et al. (2005). Vitamin D receptor gene polymorphisms are linked to and associated with adult height. *Journal of Medical Genetics*, 42, 228-234.

Table 1 Expenditures on health as percent of GDP and percent spent in public sector: 1990 and 2000

Region/country	Percent of GDP spent on health		Percent of health in public sector	
	1990	2000	1990	2000
<i>1. South Asia</i>				
Bangladesh	3.2	4.1	44	37
India	6.0	4.9	22	18
Sri Lanka	3.7	3.7	49	49
<i>2. Latin America and Caribbean</i>				
All	4.0	NA	60	NA
Brazil	4.2	8.3	67	41
Chile	4.7	3.2	72	78
Cuba	NA	7.1	NA	86
Mexico	3.2	7.3	50	42
<i>3. East Asia</i>				
China	3.5	5.4	60	37
Indonesia	2.0	2.7	35	22
Thailand	5.0	3.7	22	57
<i>4. Sub-Saharan Africa</i>				
All	4.5	NA	56	NA
Cote d'Ivoire	6.2	2.8	52	36
Ghana	3.5	4.1	49	54

Kenya	4.3	8.8	63	27
Nigeria	2.7	1.7	44	29
<i>5. Developed countries</i>				
All	9.2	NA	61	NA
Canada	9.2	9.0	74	72
Norway	7.4	7.6	95	86
Germany	8.0	10.6	73	75
Japan	6.5	7.7	74	77
United States	12.7	13.1	44	44

NA, not available from source. *Sources:* 1990 data: *World development report 1993*, World Bank, Oxford University Press, New York (Table A-9) and 2000 data: *Human development report 2003*, United Nations Development Programme, Oxford University Press, New York (Table 6).

Table 2 Percentage of countries by development level and year according to their views on population policies

1. Fertility policies

Objective	Raise	Maintain	Lower	No intervention
<i>Least developed countries (N = 49, 1996)</i>				
1976	2	5	14	79
1996	0	6	65	29
2004	0	8	69	22
<i>Less developed countries (N = 146, 1996)</i>				
1976	5	10	34	50
1996	8	10	56	26
2004	8	11	57	24
<i>More developed countries (N = 48, 1996)</i>				

1976	21	21	0	59
1996	33	8	2	56
2004	44	10	0	46
2. Access to contraceptive methods				
Objective	Limits	No support	Indirect support	Direct support
<i>Least developed countries</i>				
1976	10	33	14	43
1996	0	6	6	88
2004	0	2	10	88
<i>Less developed countries</i>				
1976	6	21	9	64
1996	1	10	8	82
2004	0	5	10	85
<i>More developed countries</i>				
1976	9	12	18	62
1996	2	25	15	58
2004	2	12	38	48
3. Concerned about adolescent fertility				
Objective	Major	Minor	Not a concern	
<i>Least developed countries</i>				
1996	48	27	24	
2004	63	24	12	
<i>Less developed countries</i>				

1996	53	28	19
2004	61	29	10
<i>More developed countries</i>			
1996	27	36	36
2004	33	48	19
4. Accept mortality levels			
Objective	Acceptable		Unacceptable
<i>Least developed countries</i>			
1976	5		95
2004	4		96
<i>Less developed countries</i>			
1976	24		76
2004	35		65
<i>More developed countries</i>			
1976	79		21
2004	69		31

Source: United Nations, *World population policies, 2004*, New York, 2004 (Tables 6-8, 10, 17, 19).

Table 3 Estimates of the partial association between height and log hourly wage in Ghana, Brazil, and the United States

Country, year, survey, age, and sex	Ordinary least squares	Instrumental variable			Sample size	Mean (standard deviation)
		Regional and household characteristics	Regional characteristics and parent education	Ethnic or race groups		Height (cm)

<i>Ghana, 1987-1989</i>						
Living standard measurement survey age 25-54						
Males	0.0154 (4.78)	0.0943 (3.71)	0.0934 (4.27)	-0.1017 (3.66)	2876	169 (6.49)
Females	0.0167 (4.04)	0.1091 (4.14)	0.1522 (6.56)	-0.0765 (2.37)	2754	158 (6.16)
<i>Brazil, 1989</i>						
Health and nutrition survey age 25-54						
Males	0.0140 (10.3)	0.0775 (9.34)	NA	0.0701 (12.4)	7808	168 (7.20)
Females	0.0166 (8.13)	0.1086 (8.12)	NA	0.0862 (8.11)	4047	156 (6.63)
<i>USA, 1989-1993</i>						
National labor force survey of youth age 24-36						
Males	0.0056 (5.12)	0.0210 (1.75)	0.0353 (4.92)	0.0183 (3.08)	18,491	178 (7.65)
Females	0.0043	0.0453	0.0473	-0.0023	16,695	163

	(3.62)	(1.93)	(4.06)	(.48)		(6.98)
--	--------	--------	--------	-------	--	--------

Notes: beneath the estimated coefficient on height in the log wage equation are reported in parentheses the absolute value of the *t* ratio. In addition to height in the wage function, the standard specification includes a spline in years of schooling completed by levels, postschooling years of experience (age-schooling-6), experience squared, and rural residence.

Source: Schultz (2002) Appendix Table.

Table 4 Coefficients on four indicators of human capital inputs in log wage equation in Ghana, 1987-1989

Gender and variable	(1) OLS	(2) OLS	(3) OLS	(4) OLS	(5) IV	(6) IV
<i>Males: sample size 3414</i>						
Education	0.0521	0.0475	0.0449	0.0437	0.0445	0.0445
	(11.7)	(10.7)	(10.1)	(9.86)	(2.46)	(9.95)
Migration		0.388	0.360	0.348	0.218	0.295
		(7.48)	(6.97)	(6.75)	(2.26)	(5.34)
BMI			0.0542	0.0530	0.0793	0.0658
			(6.93)	(6.80)	(1.95)	(1.76)
Height				1.48	5.69	5.56
				(5.02)	(3.45)	(3.58)
<i>Females: sample size 3400</i>						
Education	0.0481	0.0425	0.0395	0.0375	0.0356	0.0346
	(9.23)	(8.22)	(7.69)	(7.26)	(2.69)	(6.56)
Migration		0.617	0.537	0.531	0.361	0.447
		(9.85)	(8.55)	(8.46)	(2.98)	(6.51)
BMI			0.0425	0.0420	0.0981	0.0881
			(7.72)	(7.63)	(4.11)	(4.32)

Height	1.29	7.48	7.62
	(3.63)	(3.44)	(3.80)

^aVariable is assumed endogenous and estimated by instrumental variables, which include parent education and occupation, local health infrastructure, and food prices.

^bOther control variables include region of birth, ethnic group, age and season of interview. Beneath regression coefficient is the absolute value of the *t* ratio in parentheses in columns (1)-(4) and asymptotic *t* ratio in columns (5) and (6).

Source: Schultz (2003) Table 4.

Table 5 Means and standard deviations in parentheses of height and schooling by country, for selected age groups, by sex, and change over 30 years

Country and age	Height in cm (standard deviation)		Schooling in years (standard deviation)	
	Female	Male	Female	Male
<i>Ghana, 1987-1989</i>				
Age 25-29	158.53 (6.25)	169.46 (6.63)	5.29 (4.97)	8.29 (5.09)
Age 55-59	156.93 (5.96)	169.00 (6.51)	2.12 (4.29)	5.68 (5.97)
Change	+1.60 (+0.29)	+0.46 (+0.12)	+3.17 (+0.68)	+2.61 (−0.88)
<i>Cote d'Ivoire, 1985-1987</i>				
Age 25-29	159.11 (5.67)	170.11 (6.70)	2.78 (3.99)	6.12 (5.07)
Age 55-59	157.57 (6.11)	168.48 (6.88)	0.23 (1.32)	2.30 (3.98)
Change	+1.54 (−0.44)	+1.63 (−0.18)	+2.55 (+2.67)	+3.82 (+1.09)
<i>Brazil, 1989</i>				
Age 25-29	156.27 (6.62)	168.90 (7.27)	6.36 (4.31)	5.66 (4.22)
Age 55-59	153.16 (6.59)	165.79 (7.47)	2.21 (2.86)	2.52 (3.16)
Change	+3.10 (+0.03)	+3.11 (−0.20)	+4.15 (+1.45)	+3.14 (+1.04)
<i>Vietnam, 1992-1993</i>				

Age 25-29	152.16 (5.39)	162.10 (5.39)	7.90 (3.21)	8.35 (3.38)
Age 55-59	148.73 (5.64)	159.19 (5.93)	3.74 (2.59)	6.48 (3.82)
Change	+3.43 (−0.25)	+2.91 (−0.54)	+4.16 (+0.62)	+1.87 (−0.44)

Sources: Schultz (2005) Table 3.

* The comments of various audiences at several institutions are appreciated, especially those of Germano Mwabu, Mark Rosenzweig, David Sahn, and John Strauss. The research has been supported by the MacArthur Foundation especially that summarized in [Section 8.4](#).

¹ [Ben-Porath \(1967\)](#) is the first to link formally the increase in expected life span to increases in human capital investments and growth in income, but he assumes that a fixed lifetime is allocated between human capital investment and market earnings, ignoring changes in leisure or nonmarket production or labor supply, and the endogeneity of life span or health. Implicit in Ben-Porath's framework is the idea that with a longer lifetime to collect the returns on human capital, individuals are encouraged to allocate longer periods to (schooling) human capital investment at the start of the life cycle. [Hazen \(2007\)](#) shows that lifetime hours in the market labor force for male workers in the United States has decreased since the cohort born in 1840, measured in synthetic period rates or more appropriately in cohort rates, and he concludes that this reduced lifetime labor supply when survival and human capital are increasing is inconsistent with his revised Ben-Porath model. But he neglects to account for nonmarket production or full income, and the offsetting increase in female market labor supply over time, and institutional changes in pensions, which impose additional implicit taxes on the earnings of workers after they become eligible for social security pensions, and may be responsible for the observed reduction in lifetime labor supply ([Gruber & Wise, 1995, 2004](#)).

² Preston subsequently excludes the seven Soviet-bloc countries in the 1960s from his cross-country analysis, perhaps because Soviet national income estimates are based on different accounting practices, and international exchange rates into dollars for the Soviet-bloc currencies are not determined in a "free market."

³ Preston, however, prefers a more flexible exponential functional form, because the simpler semi-log fitted function systematically overestimated life expectancy at low-income levels, a

sign of misspecification. This exponential fitted model is plotted in [Figure 2](#), and reveals that in each of the 3 years, countries with higher *per capita* income tended to have lower mortality and a longer life span. [Preston \(1980\)](#) subsequently extended his analysis of the association of life expectancy and income using data for 1940 and 1970 by adding adult literacy and *per capita* calorie supply as determinants of life expectancy. Literacy is significant positively associated with life expectancy suggesting a 10% point increase in literacy is related to a 2-year gain in life expectancy, whereas a 10% increase in *per capita* income is related to about a half year increase in life expectancy. The coefficient on income is reduced by 40% when literacy is included, whereas the calories variable is not significantly related to life expectancy. Others have found child mortality in the first 5 years of life is significantly related to FAO calorie supplies across countries and over time within countries, but in a nonlinear functional form such as the quadratic form, suggesting that the effect of calories on child mortality is subject to diminishing returns ([Schultz, 1997](#)). The strongest covariate with child mortality in Schultz's study is the years of *female* education. Preston does not distinguish between literacy of men and women separately, although the empirical tendency in numerous studies at various levels of development and aggregation is that female education is associated with a large reduction in mortality, whereas male education is generally not statistically significant once female education is controlled. This pattern is especially pronounced when the analysis focuses on infant or child mortality, although it is evident in studies of crude death rates and life expectancy as well.

⁴ The fitted curves are locally weighted smoothed scatter plots, using a tricube weighting function with a bandwidth of 0.35 years for 1960 and 2004, and 0.70 for 1930. Following Preston, Strauss and Thomas rely on foreign exchange rates to convert incomes into 1995 dollars. If incomes are expressed in units of PPP, the PPP incomes of the poorest countries would increase or be shifted to the right in [Figure 3](#), and the slope of the health-income curve would be increased across countries.

⁵ HIV/AIDS may reduce life expectancy by 10-20 years in a decade as in Botswana, but may affect the productivity of infected individuals only in the last year of life with the emergence of full blown AIDS. In the past, those dying between ages 15 and 50 were more likely to have chronic illnesses for many years and consequently experience diminished productivity for much

of their lifetime. HIV/AIDS has altered the relationship between life expectancy and productivity.

⁶ The increase in the wage due to improved health of the worker may also affect the market labor supply of the worker, increasing hours worked if the substitution effect (positive) of the wage on labor supply outweighs the respective income effect (negative), or decreasing hours worked if the income effect exceeds the substitution effect (Schultz & Tansel, 1997).

⁷ Age of mother affects slightly the probability of having a twin, and could influence the timing costs of a twin. Rosenzweig and Zhang (2009) therefore control for age of mother at birth, which is also a choice variable of the family that could be associated with other child investments and family behavior. Conditioning on maternal age at birth purges the first-stage prediction of the probability of having a twin from its possible dependence on age. Age of mother is, nonetheless, endogenous to child investments, such as birth weight and length, and cannot be treated as independent of the error in the later child, mother, or family outcome variables.

⁸ Because the children in the Kunming China sample are still of school age, it is not possible to follow them into the labor force and estimate wage functions in order to evaluate the exogenous family size effects on adult productivity.

⁹ With such imprecise estimates of adult mortality and cause-specific deaths, there is much uncertainty, as recently reflected in the downward adjustments in global estimates of mortality due to HIV/AIDS and more recently malaria.

¹⁰ To account for the effects of health on economic growth, Weil (2007) analyzes the survival rate for adults from age 15 to 60, which is a reasonable refinement compared to crude death rates or life expectancy at birth, because the goal is to describe the productive health status of the population in the prime working ages, not early childhood or the elderly. Lorentzen, McMillan, and Wacziarg (2008) also treat both infant mortality and adult mortality (age 15-60) as determinants of growth through their effect on investment, schooling, and fertility. But this step toward a more reasonable measure of adult mortality does not improve the empirical foundations for his adult health indicator. It remains essentially a residual imputation of deaths to adults which are not attributed to children dying, and may be prone to large and possibly systematic errors in low-income countries, an error which is difficult to assess without better death registration systems.

¹¹ IIASA ([Lutz & Goujon, 2001](#)) has developed and projected estimates of the educational attainment distribution of the populations of countries by age and sex, using cohort methods that rely on additional data sources than the UNESCO published tabulations adapted by Barro-Lee ([2000](#)).

¹² To assess this lagged effect of childhood health on economic productivity and growth, one might have asked researchers such as [Acemoglu and Johnson \(2006\)](#) to lag their increases in child survival 50 years and thereby to account for national income growth. The lack of sufficiently long time series for mortality and subsequent income in either low or high-income countries appears to limit this attractiveness of this line of inquiry.

¹³ BMI is typically measured by dividing the individual's weight in kilograms by her height in meters squared, and it is thus weight approximately normalized for the individual's height. Variation in both height and BMI is typically expressed as Z scores to be in comparable units of standard deviations. The high intercorrelation between height and weight would otherwise make it difficult to estimate jointly outcomes as a function of both dimensions of stature.

¹⁴ Activities of daily living are used to assess functional disability among the elderly in high-income countries, although their value as measures of health status among young or middle aged individuals is not clear, nor is their validity in the context of low-income countries or across countries ([Schultz, 2005](#)). [Banerjee and Duflo \(2007\)](#) show for a poor tribal area of India and Indonesia that ADLs of poor and relatively rich individuals only diverge after about age 55. Since they conclude that mortality and bad health are more common among the poor than the rich, their results suggest ADLs are not a discriminating measure of reproducible health differences until individuals are over the age of 60 in low-income countries. Other measures of health stocks are needed for persons under age 60, such as possibly height and BMI.

¹⁵ [Case, Fertig, and Paxson \(2005\)](#) report persisting associations between economic circumstances at birth and childhood illness, self assessed health, and socioeconomic status as an adult. They conclude from multiple regressions fit to the UK 1958 National Child Development Study that birth weight, chronic health conditions in childhood from age 7 to 16, and height at age 16 and maternal smoking are related to adult outcomes, even after controlling for socioeconomic characteristics of the origin family. They conclude that the "gradient" or gap between the health of children by parent socioeconomic status (SES) widens (or accumulates) as

the child ages. In other words, socioeconomic inequality in health increases over the child's life cycle. [Currie and Stabile \(2003\)](#) estimate similar tendencies for the SES gradient between the health of children to widen with age in Canada, while [Currie and Hyson \(1999\)](#) examine the impact of low birth weight on schooling and labor market outcomes, as the children experience repeated health shocks. For these estimates to be interpreted as causal effects, the initial features of the child's health must be assumed to be exogenous as a right-hand side determinant of subsequent child outcomes. Even birth weight embodies the endogenous effects of parent behavioral choices and differences in environmental conditions ([Rosenzweig & Schultz, 1983](#)) introducing heterogeneity bias into causal interpretation of regressions of lifetime ordered health indicators. These issues are discussed later in this paper.

¹⁶ Cross-country study of child mortality found a significant nonlinear relationship between the availability of calories *per capita* and child mortality, with diminishing effects with increased calories ([Schultz, 1997](#)).

¹⁷ Little is known about the gene control of human height, weight, and BMI, but standard variance component analysis or correlations between relatives show that up to 60% of these anthropometric indicators of stature can be attributed to genetic factors ([Ginsburg et al., 1998](#)). A growing number of genetic dissections have been performed that suggest major genes are involved in genetic control of the traits and are reflected in widely dispersed population around the world ([Liu et al., 2004](#)). Assortative mating is operating on the traits as well and the joint distribution of the genes of mates will ultimately be part of the dynamic process determining height in the population.

¹⁸ The adolescent spurt in physical growth may be another period in the life cycle when the individual is especially vulnerable to nutritional deprivation and health care, and though the timing of this growth spurt can be delayed by malnutrition, it is unclear whether adolescent insults affect the height attained by the mature adult ([Floud et al., 1990](#); [Tanner, 1982](#)).

¹⁹ Notable regional variation in average adult height is evident in the world, although many studies since the mid-nineteenth century suggest shorter peoples have increased their height in periods when their nutrition and health conditions improved ([Tanner, 1982](#)). Nonetheless, outliers, such as the greater height of many contemporary African populations and those who migrated from Africa, suggest genetic traits of these populations express themselves in greater

height (Deaton, 2008). However, there is little evidence that these variations in height associated with ethnic identification, controlling for socioeconomic characteristics such as parent education and urbanization, are necessarily associated with differences in individual economic productivity (Schultz, 2002). In the Ghana LSMS, the tribal/language ethnic groups explain significant differences in height, and one might imagine they are genetically affected, but as instruments they do not predict variation in height that is positively related to wages (Schultz, 2002; see Table 3).

²⁰ Linearly interpolating values between the quintiles that Su (2005) reports, I find individuals who have a BMI less than 19 exhibit a mortality rate that is 20% above the average in 1870, whereas this elevated risk of mortality (i.e., 1.2) is associated in 1972 with a BMI of less than about 21, an increase of about two units of BMI. The relative mortality risks in 1870 suggest a 20% higher than average mortality occurs for Union Veterans whose BMI is about 27 or more, whereas in 1972 individuals with BMI of 31 or more are observed to experience mortality at least 20% higher than average. This mortality threshold shifted to the right by about four units of BMI in this century. If these small samples of 800-600 individuals are sufficient to draw tentative conclusions, in this century the mortality minimizing level of BMI has increased, and of course the proportion of the US population at higher values of BMI has also increased, shifting the population distribution substantially to the right (see also Sunder, 2005; Sunder and Woitek, 2005).

²¹ If health policy priorities target reducing “obesity” or the frequency of BMI in the population above some critical threshold value, these values should be assessed with confidence for a variety of populations, and if possible over time. The distribution of diseases and health conditions causing death, public and private health institutions, and health-related behaviors may differ, occupations and life styles may have changed. For example, the decline in cardiovascular deaths may have been due to the use of drugs reducing blood pressure, which could have disproportionately extended the lives of those who have relatively high BMI in the 1980s in the United States.

²² They consider the intergenerational effect of mother’s fetal growth on the twin’s own child’s birth weight, by estimating the intergenerational relationship for all of the twins who became mothers prior to the resurvey. The intergenerational correlation is significantly positive,

indicating a persistence in birth weight health across generations as typically reported. But estimated within twin mothers, which thereby removes the effect of shared nutritional habits and preferences between mothers who were twins, the effect of mother's fetal growth becomes statistically insignificant as a determinant of the differences between the twin's children's birth weight. They conclude the intergenerational correlation of birth weight is due to the transmission of preferences, habits, and matching a mate in the marriage market with favorable endowments, and not due to the biological transmission of health endowment from one generation to the next in the form of birth weight.

²³ Males with 10% greater birth weight are 1.2% more likely to be full time workers (Table 3). Estimates of the birth weight effect on indicators of child development tend to decrease in absolute value when estimated within twins compared to for all singleton or twin births, especially for 1-year mortality, APGAR score, and full time work. A 10% greater birth weight within twins is associated with only a 1% difference in log full time earnings, a much smaller effect than estimated for Minnesota female identical twins by Behrman and Rosenzweig (2004). Among female twins who both have children in the sample, the within twin estimated effect of log birth weight on the log birth weight of their first child is 0.15, substantially larger than estimated by Behrman and Rosenzweig (2004).

²⁴ Data et al. (2007) argue that Indian rural villages with higher mortality risks evidence larger differences in sibling health inputs (e.g., breastfeeding and immunization) between small-at-birth and large-at-birth siblings. They interpret their findings to suggest that in a higher mortality risk environment, Indian parents increase their concentration of child health inputs on their better endowed at birth of their children.

²⁵ This pattern of larger estimated effects of child health on adult productivity using instrumental variables for economic origins and health conditions at birthplace is also consistent with there being substantial measurement errors in anthropometric health and fetal growth variables, and that the observed variation in individual height may prudently be filtered through the choice of an appropriate instrument to approximate the effect of economic policies which might increase the productive capacity of an adult through its effect on increasing the rate of fetal growth.

²⁶ There is no particular reason to prefer this semi-log functional form except Mincer used it to analyze schooling and earnings. An analysis of data for men and women from the German Socio-

Economic Panel suggests that the fit of the log wage function by OLS is improved when the logarithmic transformation of height is the conditioning variable (Hubler, 2006; Table 5). That paper finds evidence that males receive more wages with greater height until they exceed two standard deviations above the sample mean height, whereas the wage gains from height for women reaches its maximum at the mean height for German women. There are many possible interpretations to these nonlinear patterns that have not been extensively explored.

²⁷ The effect of education on health in the United States is appraised by Lleras-Muney (2005) but using OLS and IV estimates identified by changes in state of birth compulsory school attendance legislation. The lack of significant change between the OLS and IV estimates suggests that schooling is exogenous with respect to the determination of health in the US in recent times.

²⁸ If the selection effect of mortality by height were important in low-income countries, as conjectured by Deaton (2007), then the older cohorts would actually have been born shorter on average than the surviving cohorts, and my imputation of a gain in wages by younger cohorts due to their greater height is understated. In other words, the selection effect, to the extent that it occurs in low-income countries, would add to the wage returns to height as reported here due to the increased life span associated with height. The analogous argument applies to cross cohort estimates of returns to schooling, where most evidence suggests survival tends to be greater among the better educated.

²⁹ Famines have also been studied as an instrument affecting health of those born in a specific “window” and compared to birth cohorts before and after. Development economists have frequently used droughts, rainfall, or floods to randomly affect the income constraint in poor populations, providing the instrument to identify the effects of income on health and behavior of family members. An early example is Rosenzweig and Wolpin (1985).

³⁰ Infant mortality is also higher among spring births, and following colder winters, after bad harvests in Sweden (e.g., Eckstein et al., 1985). This raises the possibility that birth cohorts from less healthy months would be culled of the less healthy members, leaving the more fit to survive and live relatively longer lives, other things equal. This countervailing selection effect would conceal the pattern implied by the fetal origins hypothesis analyzed by Doblhammer (2004). Few studies have sought to assess how the selectivity of reproductive behavior modifies the composition of cohorts born in crises periods.

³¹ To the extent that the stressing conditions are somewhat predictable, the socioeconomic composition of those having children may vary according to their preferences and resources to have healthy or high-quality children. [Buckles and Hungerman \(2008\)](#) show that women who bear children in the winter months in the United States are disproportionately unmarried, teen age, not a high school graduate, and nonwhite. Thus, season of birth may not only reflect the weather during a child's early development, but capture the consequences of being born to a socioeconomic class who for other reasons experiences less adequate health care and educational preparation. Compare with the interpretation of the estimates of compulsory schooling derived from quarter of birth in [Angrist and Krueger \(1991\)](#) or estimates of late age mortality from season of birth in [Doblhammer and Vaupel \(2001\)](#).

³² In another study, [Almond, Edlund, and Palme \(2007\)](#) analyze data on the radioactive fallout from the Chernobyl accident of 1986, as geographically distributed by rain and wind across Sweden, and find its deposition is associated with lower levels of school completion and IQ of the cohort exposed from the 5th to 25th week of gestation, compared with birth cohorts born before and after this accident, allowing for region fixed effects and time trends.

³³ The vitamin D receptor (VDR) affects human stature and mediates calcium and phosphate homeostasis and has been linked to specific gene polymorphisms with wide ramifications ([Holick, 2004](#); [Xiong et al., 2005](#)). Since skin pigmentation (i.e., melanin) blocks UVB radiation, lighter skinned races as well as those who are exposed to more intense sunlight (i.e., lower latitudes) appear to have greater levels of vitamin D and tend to be taller, although variations in height is more generally attributed to nutritious diet and reduced exposure to inflammatory diseases of early childhood, within biological limits (Finch, 2007; [Fogel, 2004](#); [Ginsburg et al., 1998](#)). Vitamin D deficiencies in the US population in the last decade, especially among African Americans, despite the vitamin D fortification of milk started in the 1930s, has led public health experts (e.g., NICHD and CDC) to search for better biomarkers of adequate vitamin D levels, and a better understanding of their consequences on not only bone health, but also associated with other chronic diseases. The determinants of vitamin D deficiency and their consequences on height and health are becoming a public health priority, and economists could analyze this pathway to assess the economic consequences of different sources of variation in height, based on different instruments for height, including geography and diet.

³⁴ Contrary to expectation, some studies find contemporaneous procyclical movement in mortality with the business cycle in today's high-income countries. In the twentieth century with rising levels of income in Netherlands, *current* adult mortality is positively related to the business cycle (Van den Berg et al., 2006). Ruhm (2000) has also shown that adult mortality varies procyclically in the United States given existing social safety net programs and household assets. He explores pathways such as a possibly healthy reallocation of time from work to families, and from work to leisure activities in periods of recession, despite the concurrent decline in income and increase in unemployment. The changes in mortality rates from age 30 to 85 and real GDP growth rates appears to be positively correlated in six OECD countries until about 1990, as Ruhm (2000) noted in the US and Tapia Granados (2008) in Japan. But after 1990, the correlation becomes more negative (Hanewald, 2009). Unemployment rates also confirm the macroeconomic timing of changes in elderly mortality evolves from procyclical to countercyclical, possibly due to changes in the causes of mortality with the mortality transition (Tapia Granados, 2008). Studies at the macroeconomic level cast doubt on the robustness of the relationship between income growth and gains in life expectation, or even in improvements in infant mortality (Deaton, 2003). This does not dispel the idea that income growth allows for improvements in consumption which are likely to reduce in the long-run mortality, though clearly institutional arrangements and the diffusion of knowledge about the process producing health are important as well.

³⁵ These cross-country regression studies do not control for the inputs to agriculture or the shift of population out of agriculture, such as investments in rural infrastructure, research and development of modern agricultural varieties and inputs, extension services, mechanization, irrigation, or schooling of the labor force. It is difficult to attribute the stagnation in *per capita* availability of calories in Africa to the region's population growth compared to any number of alternative factors. Analogous cross-country regressions of growth in *per capita* income and savings rates on population growth rates also finds that until the 1980s, the simple correlations are positive, in contrast to Malthusian predictions of diminishing returns to population. Only after 1980 does a negative association begin to emerge (Kelley & Schmidt, 1995). After 1980 population growth rates are subsiding in much of Asia and Latin America, and rapid population growth continues mainly in sub-Saharan Africa which has been subject to economic stagnation

for many other reasons, including civil conflict, corruption, and government policies. Removing the many small African countries from these regressions that are not weighted by population size weakens the negative associations between population growth or age composition changes and growth in income, savings, and food availability.

³⁶ Schultz (1985) proposes the sharp decline in the world price of cereal grains compared with butter and meat products after 1850, which triggered in Sweden a rise in women's wages in agriculture compared with men's wages across provinces and over time from 1860 to 1910. Because of the specialization of women and men in the production of animal products and grains, respectively, the global change in their relative prices after the opening of the US Middle West to European markets, may have contributed to the coincidence of fertility declines in many agricultural regions of Northern Europe.

³⁷ In Colombia in 1974 women of a specific age and education who had migrated from the rural to urban areas revealed preferences for urban opportunities, which in this setting include better employment prospects for women in the wage labor market, better health services which are particularly effective in preventing child mortality, and better schooling services for youth. Consequently, the rural-urban migrant woman matched by education and age has on average fewer children, and their children are less likely to have died, and more likely to have completed schooling than those women who did not migrate and stayed in the rural areas. More surprising, the rural-urban migrant woman will also have no more children than the urban native women at her destination of the same age and education, and again the migrant's children appear to have no less schooling and about the same child mortality. The migrant woman will again be more likely to be in the labor force than the match of her urban native counterpart. If the heterogeneity in preferences between migrants and natives helps to explain these behavioral differences, it is also clear that the urban-rural differences in health and schooling services will in the cross section be correlated with larger differences in the beneficial outcomes associated with these services than can be reasonably attributed to the program treatments alone (Schultz, 1988b).

³⁸ For children age 12-23 months old, the proportion having received all seven basic recommended vaccinations increased from 42% to 44% between the NFHS 2 collected in 1998/1999 and the NFHS 3 collected in 2004-2006. The prevalence of anemia for children age 6-59 months old increased to 70%, while it increased for all women to 55%, and rising also for

pregnant women ([National Family Health Survey 3, 2007](#)). [Tarozi and Mahajan \(2007\)](#) study the first two rounds of this NFHS from 1998/1999 and 1992/1993.

³⁹ Disability-adjusted life years (DALYs) are proposed as a summary measure of the objectives of the health system, refining life expectation at birth to include reductions in disability associated with the control of diseases. But even DALYs focus attention on the immediate decline in mortality and universal estimates of how specific diseases and chronic conditions among adults cause disability. They generally rely on “expert opinion” that is not subject to validation by estimation from primary data. Survey estimates of the prevalence of disabilities tend to be higher in high-income countries suggesting they are culturally conditioned. These current health outcomes do not allow for the cohort linkages emphasized here between fetal and childhood health conditions and their consequences on lifetime economic functioning of adults many years later.

Figure 1

Determinants and Consequences of Accumulating Health Human Capital

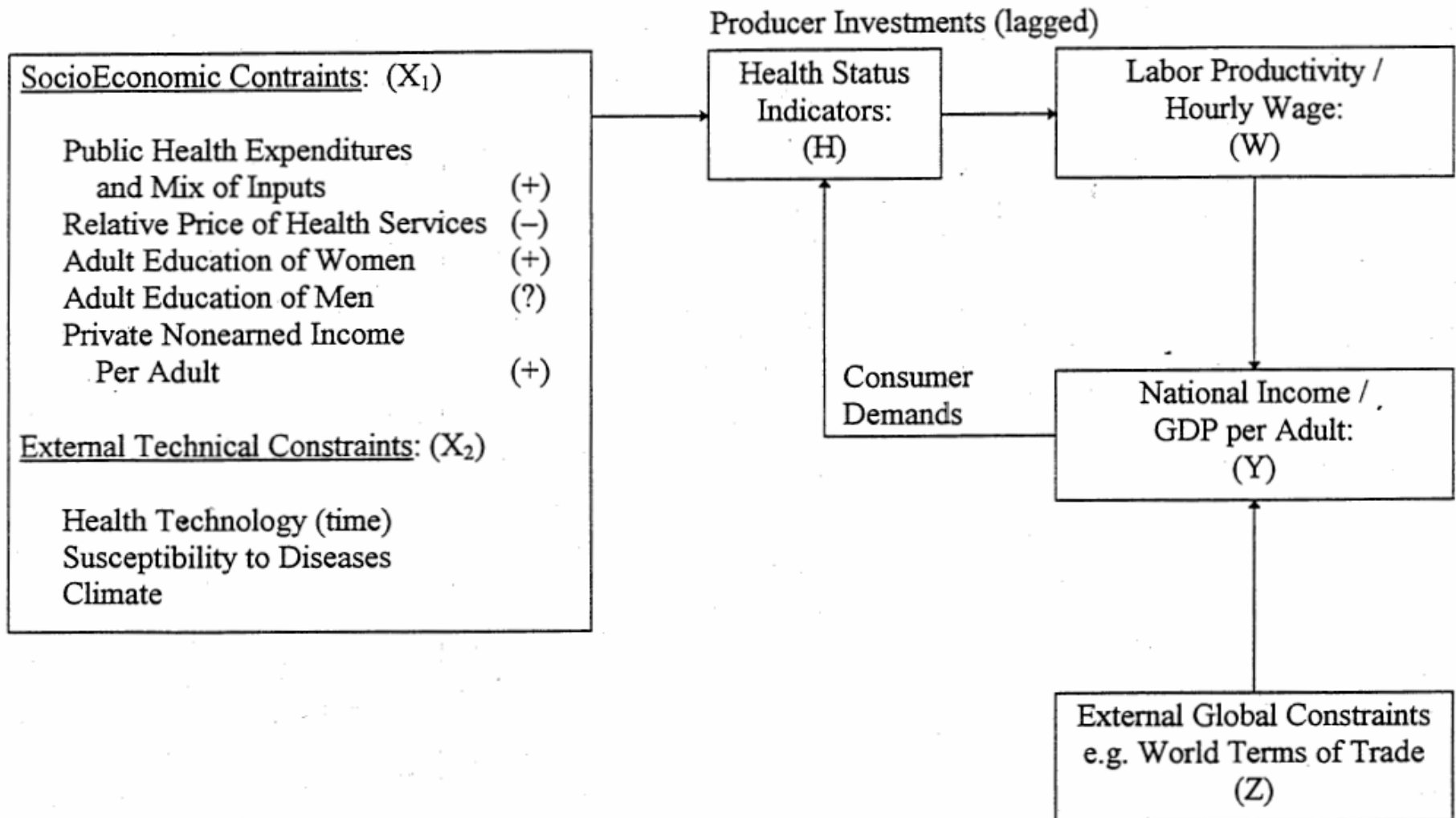
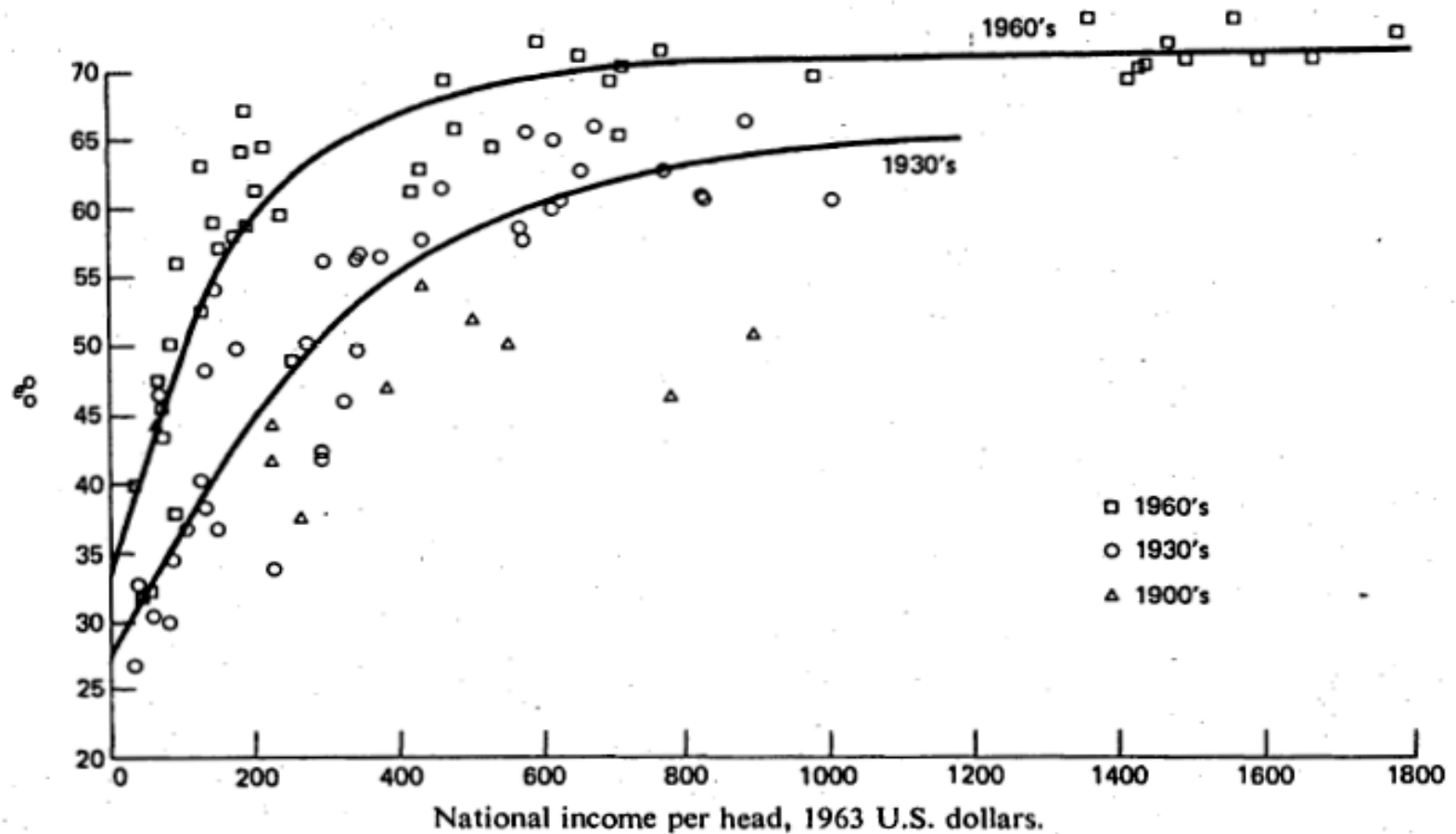


Figure 2: Scatter-Diagram relations between life expectancy at birth and national income per head for nations in the 1900s, 1930s, and 1960s

Source: Preston (1975)

Scatter-diagram of relations between life expectancy at birth (e_0^o) and national income per head for nations in the 1900s, 1930s, and 1960s.



Kenya

Figure 3: Life Expectancy and GDP per capita, Identifying outliers in 2004

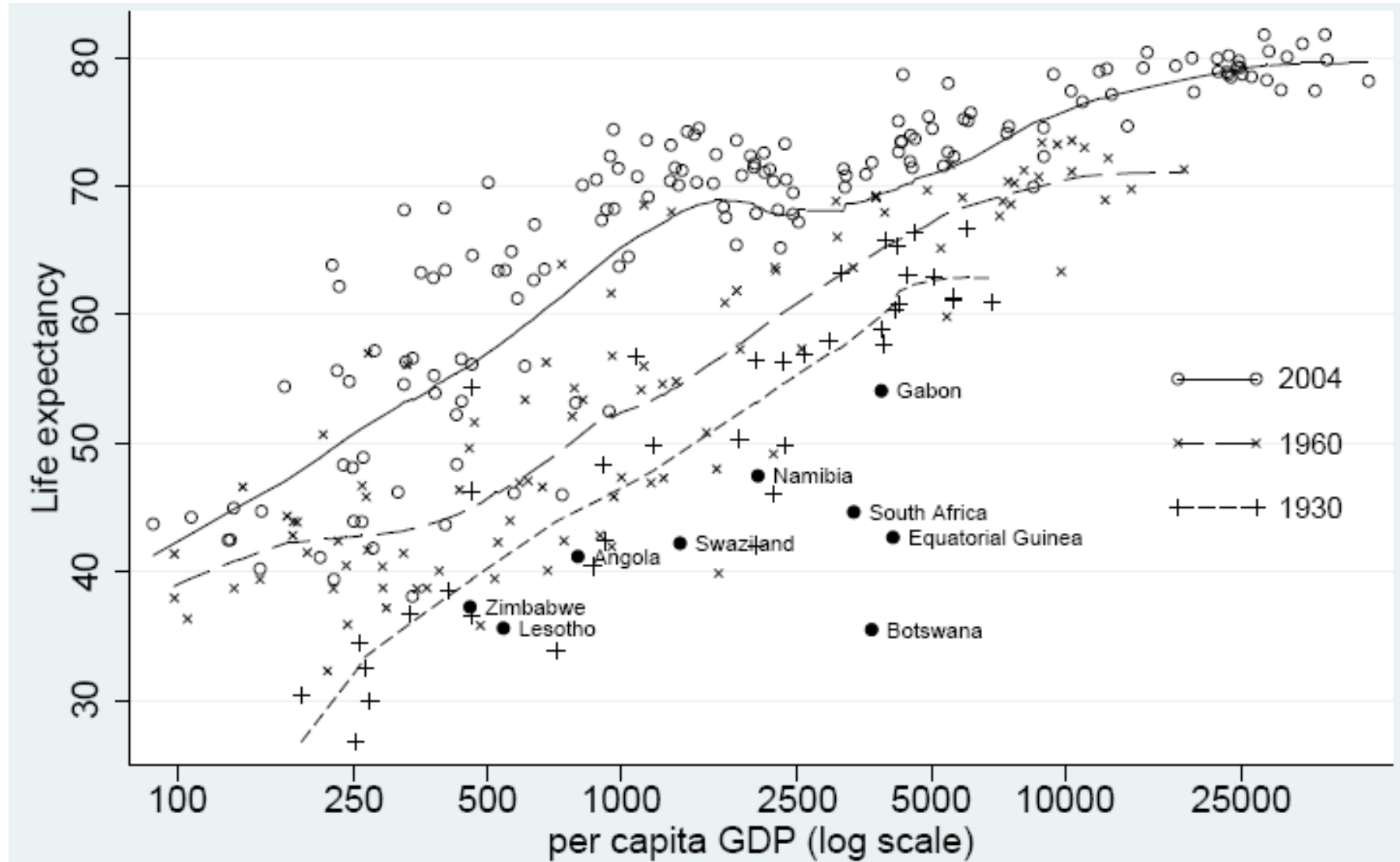


Figure 4: Relative Mortality Risk among Union Army Veterans and among Modern Norwegian Males.

Note: A relative risk of 1.0 means that the risk at that height was equal to the average risk of death in the entire population of males of the specified ages. Also note that the tallest data point, in both Norwegian and Union Army cases, is not statistically significant.

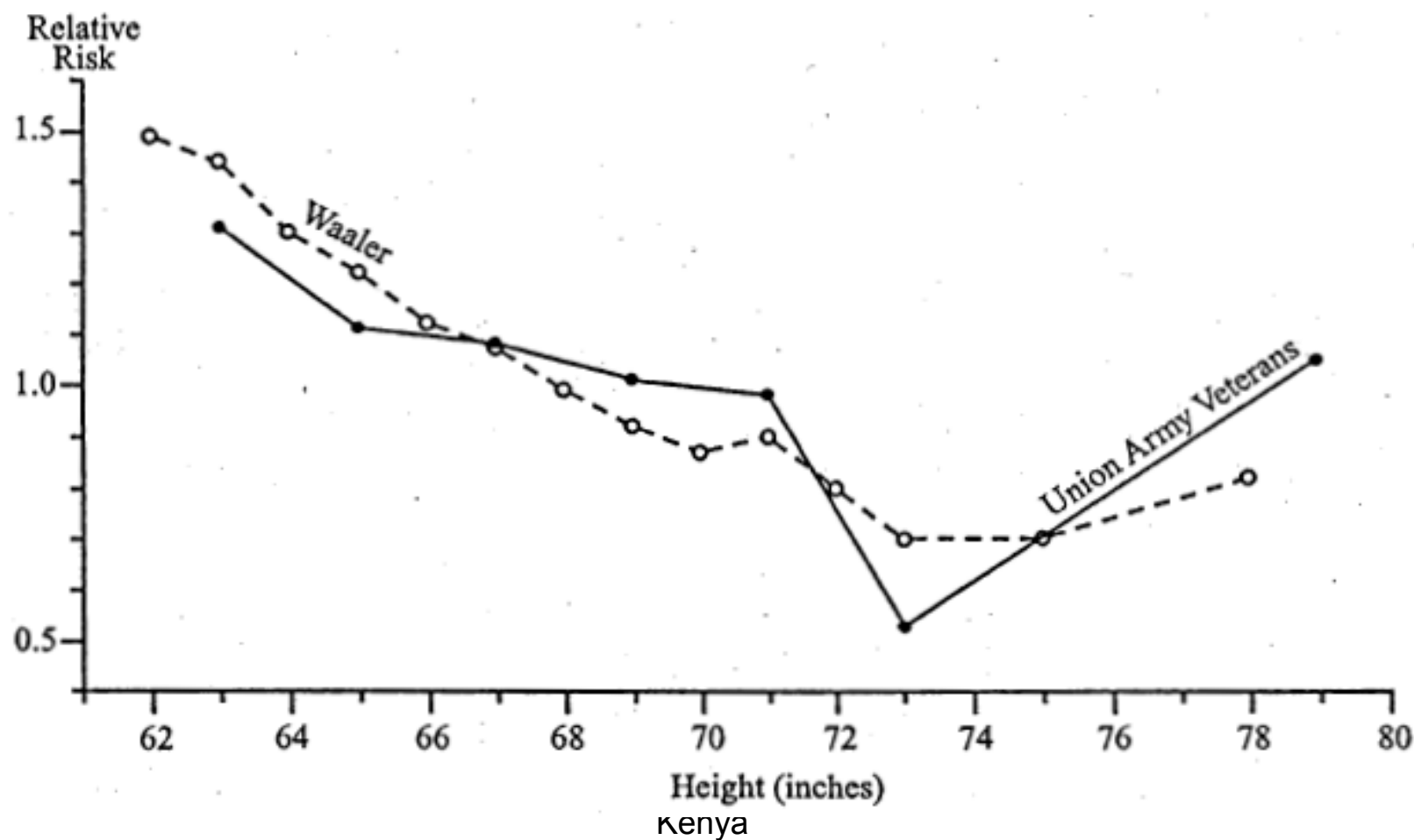


Figure 5: Height in Centimeters of Adult Females in Ghana
in 1987-1989

by year of birth, total (circle-o), rural (triangle- Δ), and urban (square- $\square$)
regions

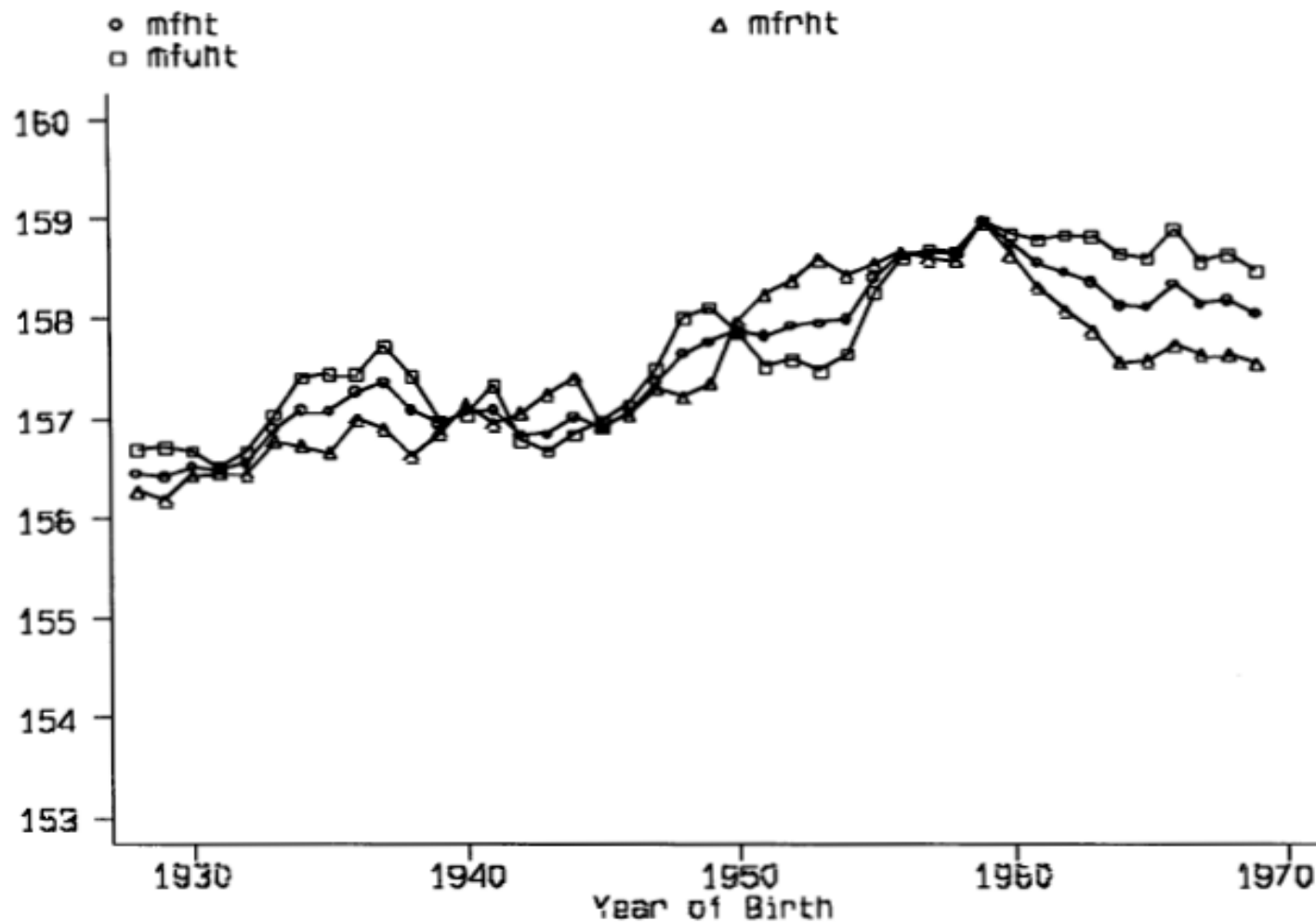


Figure 6: Height in Centimeters of Adult Females in Brazil
in 1989

by year of birth, total (circle-o), rural (triangle- Δ), and urban (square- $\square$)

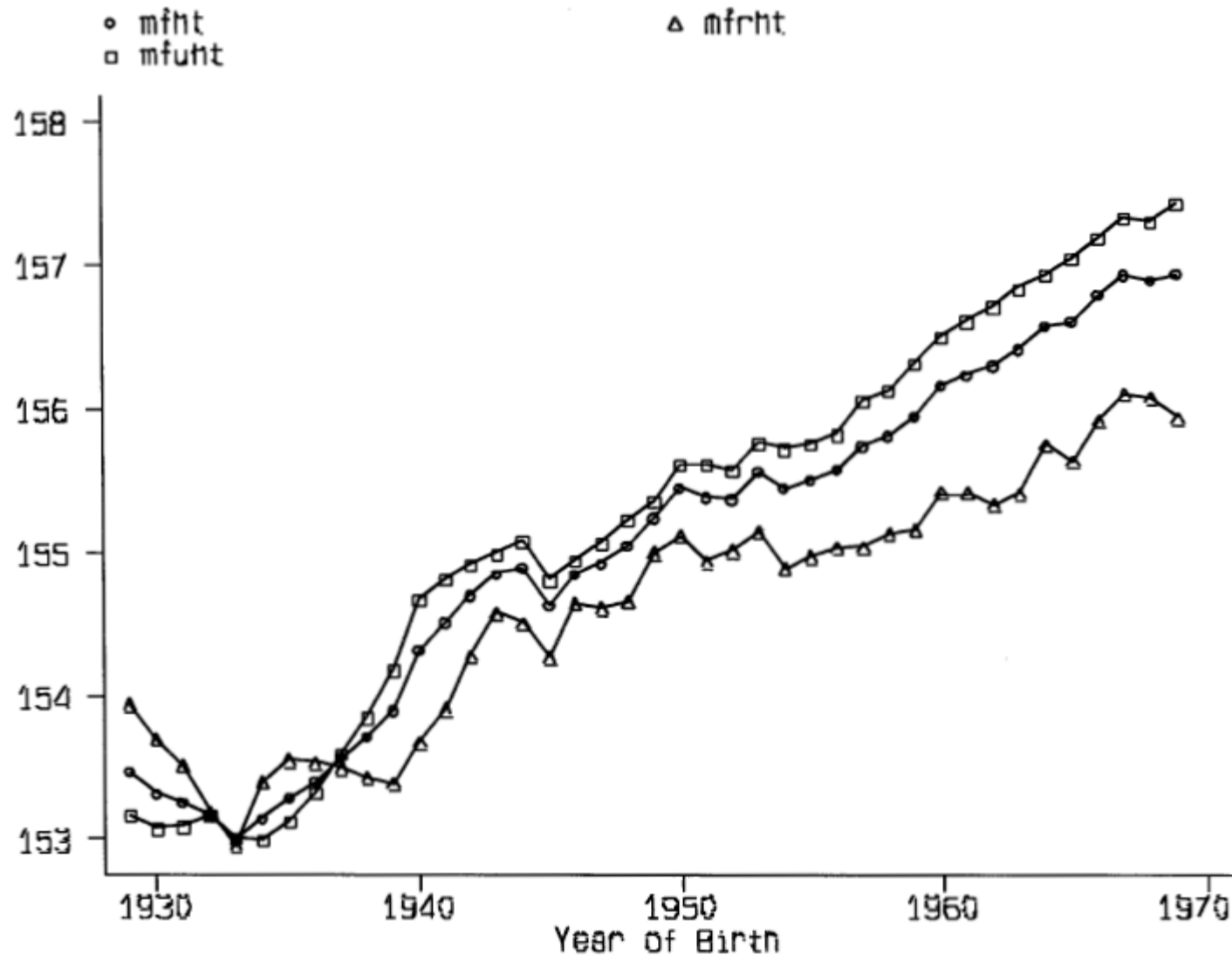


Figure 7: Comparison of Relative Mortality Risk by BMI among Men 50 Years of Age, Union Army Veterans around 1900 and Modern Norwegians.

Source: Costa and Steckel (1997)

Note: In the Norwegian data, BMI for 79,084 men was measured at ages 45-49, and the period of risk was 7 years. BMI of 550 Union Army veterans was measured at ages 45-64, and observation period was 25 years.

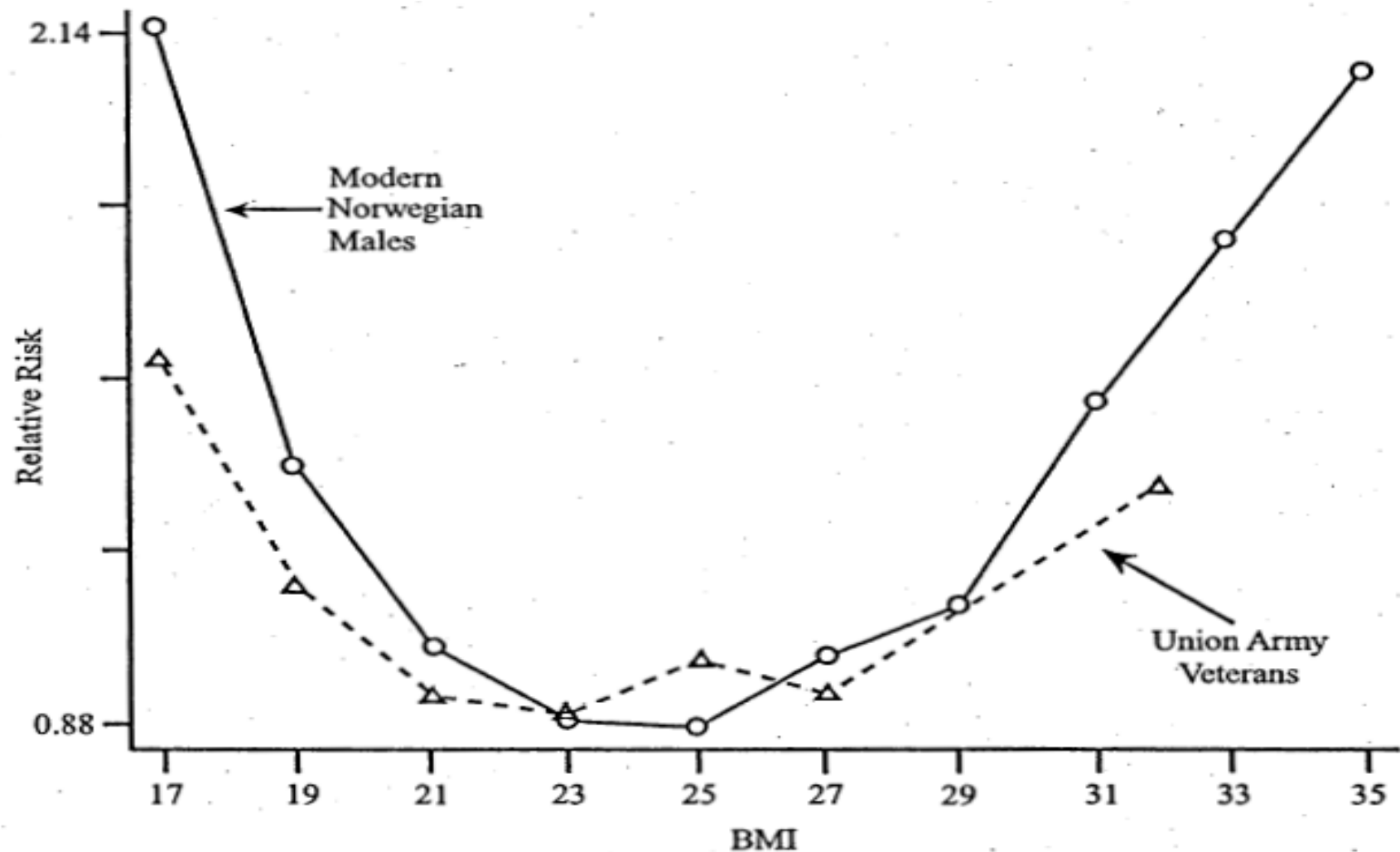
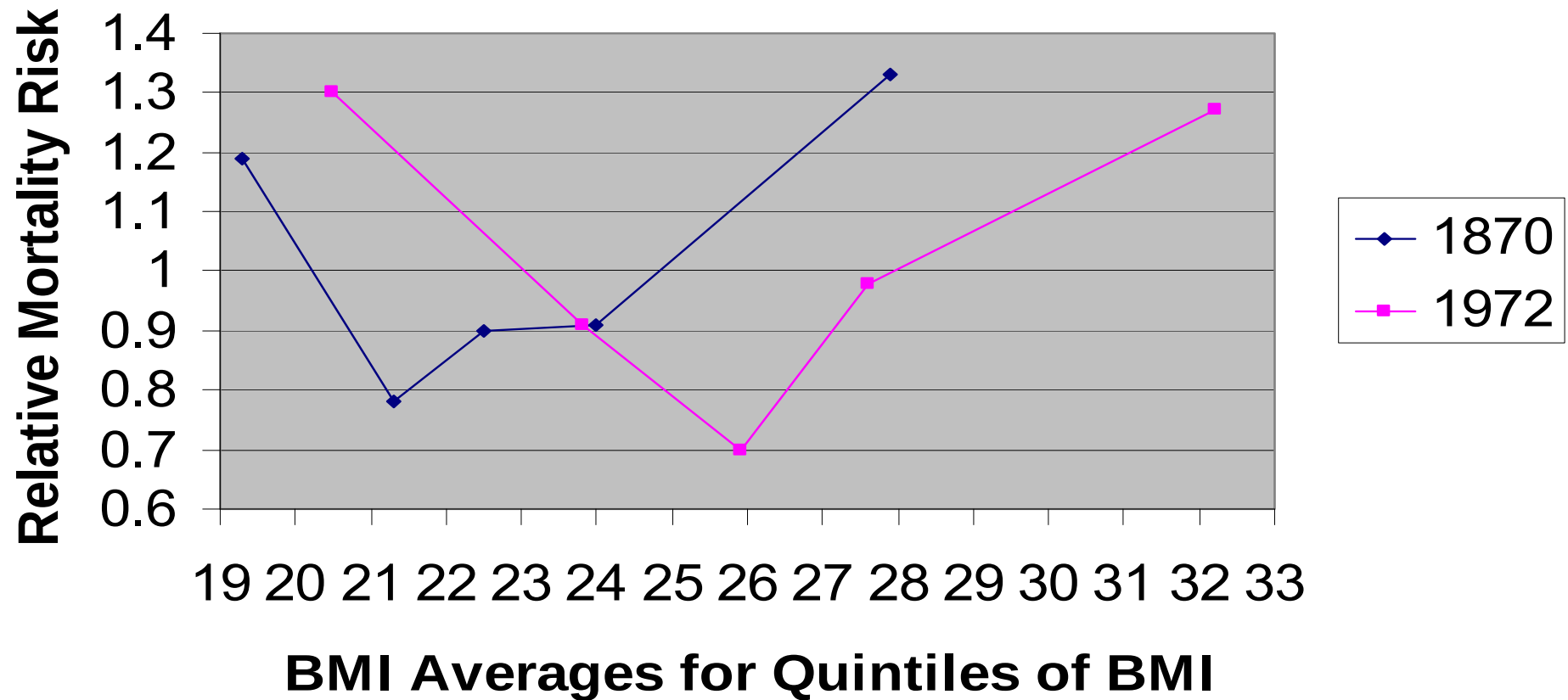


Figure 8

**Relative Risk of Mortality as Estimated from Union Army
Survival Sample US White Males age 50-59 about 1870,
and NHANES-I, US White Males age 50-59 about 1972**



**Source: Su (2006) Tables 4,5 and 6 and Figure 4. Model 1
reported based on all deaths.**