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Health Human Capital and Development*

I. Introduction

The average length of life in the world has about doubled from the start of 19th to the start of the 21st centuries, from 30-35 years to 60-70 years, with the exception of several states in Southern Africa where the HIV/AIDS epidemic in the last decade has sharply reversed previous secular gains. By some calculations this advance in length of life is more equitably shared in the world, at least until recently, than is the increase in personal consumption of market goods and services, which we commonly aggregate into national income (Weil, 2005; Becker, et al. 2005). But income and health are positively related in most cases across countries and persons, suggesting that a more comprehensive measure of well being that combined both health and economic resources would be distributed less equally than income alone . To go beyond the broad historical record of the concurrent increase of life span and economic prosperity, and understand how and why these parallel developments have occurred, involves new research, and could depend on how health is measured for comparative purposes.

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Although interpretations of the causal connections between health and economic development are controversial, evidence is accumulating in a variety of disciplines and subfields of economics, and a number of hypotheses merit more conceptual refinement and empirical testing, where specific pathways may in the future be identified and quantified. The potential linkages between the conditions under which people live and their life span and health status, on the one hand, and the relationship between their health status and economic productivity and well being, on the other hand, is the subject of this paper. To draw causal inferences on this topic from our observations of the world and the people in it, working assumptions are required that are restrictive and unrealistic; the empirical patterns we thus derive from data cannot be interpreted as reliable or unbiased evidence of underlying causal connections on which to base policy evaluations or to predict future developments.

Improvements in data allow us to deal with some of these limitations of our knowledge. This represents one of the reasons the field has made progress, specifically, household surveys have become a fundamental building block for knowledge in the social sciences since the Second World War. Panel surveys that interview repeatedly the same individuals and families over their life allow researchers to control for persistent differences across individuals and families, even when they cannot measure all of these underlying differences – unobserved biological or genetic endowments, economic resources, or preferences. Recently, survey methods have been extended to include biological indicators of physical growth, self reported health status, functional disabilities, biomarkers and assessments of biological processes. Moreover, these household sample surveys have been implemented comparably throughout the world to describe

a wide array of economic, demographic and health-related features and behaviors of populations. The empirical reach of these more standardized household surveys is a valuable component of the information technology revolution which is enabling the field of economic development and health studies to progress.

Advancement has also occurred in the statistical modeling of processes which are both biological and behavioral, and which are likely to interact dynamically over the lifetime of individuals. These models tend to be simple representations of what is a complex interplay of behavioral and biological systems, and depend on research skills to assure that the model design clarifies our thinking about the processes we want to understand, and does not only assume sufficient structure to the process to produce a plausible simulation of reality. Even in the hands of the most able and innovative scientists, these models allow only a few of the many restrictive modeling assumptions to be relaxed at one time, in order to assess the conditional validity of our causal hypotheses. It is a sign of scientific vision (or some would say blindness) to concentrate on the right (or wrong) simplifications to our problem, and work out the often technically complex corrections required to cope with even one selected specification issue. Naturally, different individuals will take different approaches and have different visions, and thus the field includes a variety of formulations to address what appears to be a common question. This is where judgements of scientists will differ, or perhaps" vision" is a more appropriate description of this inherent heterogeneity in the application of the scientific method to frontier topics of research.

Another obvious aspect of the social sciences is that it typically deals with real people working out their own lives in a specialized institutional setting, and some might argue it is therefore context dependent. In many branches of the hard sciences the design of the laboratory experiment represents a powerful dissection tool to disentangle competing hypotheses offered to account for an empirical regularity or puzzle, while holding constant all but one confounding factor, and thereby providing insight into the contribution of the remaining factor manipulated randomly in the experimental design. Most social science and health questions are addressed in a social context where there are ethical, administrative, and behavioral limitations on how such experiments can be performed. However, the field of development economics has been shaken up in the last decade by the argument that the complex behavioral and institutional mechanisms at the core of economic development, including most government health and welfare policies, can only be evaluated if policies are implemented following a randomized social experimental design, which are then evaluated by comparing over time outcomes between the treated and control populations (Heckman, 1997; Duflo et al, 2007).

These experimental methods have been recently employed to evaluate cash transfers to poor mothers if their children engage in health and school activities offered to randomly selected poor rural villages in Mexico (Parker , et al. 2007), or to improve the health and schooling of children in Kenya by treating them in randomly selected schools for endemic intestinal worms (Miguel and Kremer, 2005), or to supplement the diet of mothers and children with proteins and micronutrients in randomly selected communities in Guatemala or Tanzania (Maluccio et al., 2005; Fields, 2007), or to provide adults with iron supplements in randomly selected households in Indonesia (Thomas et al, 2007). Each of these social welfare programs is premised on good

social and medical science, but to assess their administrative, biological, and behavioral consequences careful evaluations are nonetheless justified. This is hardly a novel insight; social experiments to test social programs have been periodically implemented by governments and endorsed by researchers. But in a growing number of low-income countries social development programs have not accomplished their objectives and critics have interpreted this evidence to question the efficacy of international development assistance programs in general. There is growing demand for improved program evaluations and more systematic effort to identify and promulgate "best practices" in social programs designed to promote health and development. Without randomized trials there are reasons to distrust what are reported to be evaluations, implemented only to justify " scaled up" national programs or replications. In the area of public health, the time frame for evaluation can be long and costly. For example, the "fetal origins hypothesis" suggests improvements in utero development of the fetus and reductions in early childhood malnutrition and exposure to infections have significant impacts on adult mortality and the cognitive and productive capacities of individuals in middle and late ages where some of these effects appear to involve distinct organ system developments and resulting diseases, such as heart, lungs, and diabetes (Barker, 1992, 2005; Gluckman and Hanson, 2005, 2006).

An outline of this talk is as follows. The next section briefly outlines the debate over how health and development may be related. Section III surveys the macroeconomic literature, which relies primarily on variation across countries and over time within countries to estimate the effect of health on economic growth, and the effect of income on the demand for and production of improved health. Section IV reviews the micro economic and demographic literatures where individuals, households, and communities are the observations from which

health production functions are estimated and the productive returns to health human capital are estimated. The "fetal origin hypothesis" for modifying adult health is the theme for Section V. Section VI concludes.

II. Evidence on Health, Demographic Change, 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, which led to the discovery that the current era of "modern economic growth" had sustained growth in per capita output of 1 to 3 percent per year over a century or longer and is therefore a unique historical period (Kuznets, 1966). Demographic evidence in more developed countries accumulated showing that the expectation of life at birth also was increasing over time after about the start of the 19th century in the early industrializing countries (Oppen and Vaupel, 2005). 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 sample surveys at the household level provided parallel micro estimates of economic output to compare to national income estimates, there is no analogous or widely accepted 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 conceptually clear that life expectancy at birth is a good summary measure of the health objective of society or the productive return to 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 inter-census population growth rates and age compositions (United Nations, 1976). 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 classifications, possibly influenced by distinctive diseases and resulting regimes of mortality prevalent in these areas. Once a country is assigned to a regional class of life tables, the level of mortality within that class is approximated 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 (Coale and Demeny, 1966; Brass, 1977). 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 later 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). Comparisons of health status, morbidity, and mortality are not yet subject to comparisons based on macro and micro indicators. 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 foreign exchange rates conversions, but this excluded cross country variation in the prices for untraded goods and services. 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 exchange rates were extrapolated to the majority of countries where such price surveys did not exist (Summers and Heston, 1991).

Figure 1 illustrates the challenges posed by health and development. The association between health status (H) and income per capita (Y) is positive. This may happen because increased income leads people to (1) demand better health as a consumer good adding utility per year and increasing longevity, or (2) their increased standard of living contributes to consumption patterns which on balance contribute to health improvements, e.g. increased nutrition and reduced crowding of housing more than offsets the health losses due to increased smoking. Alternatively, improved health status can raise the productivity of people which will add to their market income summed into national income, unless their market labor supply

decreases by a larger percentage than their wage opportunities over their lifetime increase.[1] These national estimates of income and mortality, summarized generally by variations across and within countries in life expectancy and the market income guide most macro economic studies discussed in the next section. Household surveys provide empirical evidence for micro economic studies on how on the one hand, exogenous variation in income (due to Z) increases inputs for the production of health (H), and how on the other hand, the exogenous improvements in health status (due to change in Xs) enhances the economic output of workers (W) and boosts income. These empirical relationships may tell us how increased income from an exogenous improvement in a country's terms of trade (Z) contributed to the purchase of more food or better living conditions, to the adoption of healthier behaviors over the lifecycle, 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 , (Acemoglu and Johnson, 2006) or public health expenditures on HIV drugs provided by a philanthropic foundation reduce the prevalence of disease and its consequences on disability and

[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 assumes that a fixed lifetime is allocated between human capital investment and market earnings, ignoring 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 which induces the observed reduction in lifetime labor supply (Gruber and Wise, 1995, 2002).

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 is likely to 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 are likely to be controversial ? Without the assessment of these causal effects it is unlikely that policy priorities can be defended or specific pathways, health-related inputs, or subsidies can be targeted to efficiently to foster development and health.

III. Macro Economic Literature: Cross Country Evidence

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 1. If income is expressed in logarithmic (proportional) terms, the semi-log-linear fit or R^2 is relatively high, .885 in 1930s and .880 in 1960s. 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 mis-specification.[3] This exponential fitted model is

[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] He transforms his income variable to a standardized scale from 0 to 100, and fits an exponential function, thereby selecting an additional parameter in the numerator (80) to

plotted in Figure 1, and reveals that in each of the three years, countries with higher income tended to have lower mortality and a longer life span. 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 to 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. [4]

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maximize the fit of the function to the data (Preston, 1975, footnote 20). The exponential function also has the feature that it implies an upper asymptote to life expectancy which some observers associate with a biological limit on life span, potentially extended by improvements in private and public health technology and environmental conditions.

[4] Preston (1980) 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 ten percentage point increase in literacy is related to a two year gain in life expectancy, whereas a ten percent increase in per capita income is related to about a half year increase in life expectancy. The coefficient on income is reduced by 40 percent when literacy is included, whereas the calories variable is not significantly related to life expectancy. Others have found child mortality in the first five 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, whereas 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

Strauss and Thomas (2007) update these patterns by adding World Bank estimates from 2004. The fitted lines are nonparametric flexible approximations.[5] The increases in life expectancy controlling for income from 1930 to 1960 among countries with $200 to $500 dollars of income (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 by using Preston's exponential function, it is clear that most countries with incomes greater than $1,000 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 identified in Figure 2, and are notably in Subs Saharan Africa and are those countries where HIV/AIDS has raised adult mortality to exceptional levels, disrupting the past empirical pattern between health and development.[6]

What types of information would help to disentangle from the gross relationship between health and development the underlying causal links and specific pathways that might have

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with infant or child mortality, although it is evident in studies of crude death rates and life expectancy as well.

[5] The fitted curves are locally weighted smoothed scatterplots, using a tricube weighting function with a bandwidth of .35 years for 1960 and 2004, and .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 purchasing power parity, the slope of the health-income curve would be presumably increased across countries.

[6] HIV/AIDS may reduce life expectancy by 10 to 20 years in a decade as in Bostwana, 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.

implications for the choice of policies contributing to health or economic development? Researcher looks for a variable affecting either health (H) or income (Y), but not directly affecting the other variable. In Figure 1 one could imagine independent changes in terms of trade (Z) affecting a nation's income but not health, or a shift in locally effective health technology (X_2) which was produced for other purposes in high income countries, or climate variability (X_2) which affects the local prevalence of disease There are few obvious candidates for such a forcing variable, and this realization contributes to the appeal of social experiments in which the access to health treatments, or subsidies for health-related inputs, or income transfers are offered to randomly selected populations to assess health outcomes. Household surveys may still be analyzed to learn how health is produced, but they require the researcher to model the policy related determinants of health input demands and risk related behaviors. To make private and social choices we need to know how much health is produced by investments (I) of private time, goods, services, and healthier behaviors, as well as of public goods, services, institutional change, and finally the research and development costs of creating new medical knowledge and applying it, namely H= H(I(Y, Z, X)) in Figure 1. Household surveys may also be useful for assessing the market productive value of changes in individual health status, assuming health human capital raises worker productive potential and allows them to command a higher wage, namely W= W(H(X, Y, Z)) in Figure 1. Of course, willingness to pay for health may exceed the productive return because health is directly valued as a consumption good, and hence parents may be willing to pay more for child health even when the present discounted productive value over the child's lifetime of the health enhancing inputs are less than the cost of the inputs. The productive returns to health may also be unknown and uncertain, leading individuals to under-

invest in the production of health.

Health status reflects the accumulation over a lifetime of three types of factors. A genetic endowment which is assumed fixed after conception and is generally not affected by policy, though taking genetic variation into account may increase the effectiveness of policy interventions and the personal distribution of benefits from any particular intervention. Developmental variation in health caused by events after conception may have two components, one an investment choice and the other due to uncontrolled shocks. The health care system as well as the family and individual choices affect exposure to environmental risks, modify health related behavior, and provide inputs relevant for the production of health. These factors are called here investments in health human capital. The final health component is might include stochastic factors associated with disease prevalence, climate variability, and economic conditions that are uncontrolled by the person or family or community institutions. Whatever indicators of health are empirically studied, it is likely that many inputs to health are omitted from the analysis, and omitted variable bias when estimating health production functions is likely to be a serious problem, if the omitted variables are correlated with those health inputs that are empirically analyzed (Rosenzweig and 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 subsequently affected by uncontrolled 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 look the same in a survey because surveys tend to rely on more objective proxies for latent health conditions, but these proxies for health may have heterogeneous effects on worker productivity, which is the focus of this paper. Therefore, to evaluate the effects of health status for policy purposes, the productive effects of changing the *reproducible* component of health is most relevant to social policy choices. For evaluations of health policies, we are primarily interested in the production of health and more specifically the productive returns to the reproducible health human capital component of measured health which can be scaled up by individual and social investments.

At the end of the 20th century there appears to have occurred a transition from increasing longevity by controlling communicable infectious diseases, for which the costs of applying and diffusing the newly developed knowledge and technologies for controlling disease were relatively cheap even in low income settings where health care systems are simple. After these options were exhausted which had the primary effect of reducing sharply infant and child mortality, the most costly interventions for dealing with degenerative diseases non communicable diseases have become most active frontiers of medical and public health research, and it remains less clear that these new approaches to chronic diseases of heart, lungs, cancer, arthritis, diabetes, etc. will become replicable at sufficiently low cost to be demanded widely in the low income world. There is also the worry that diseases which are not prevalent in high income countries will not receive the attention of researchers, because controlling these diseases would not benefit their citizens or taxpayers, and would not promise to be privately

profitable in the marketplace if they are demanded only by people in low income counties. These developments of medical technology as well as public health programs providing access to medical care for the poor may become a larger factor in the personal distribution of health among countries and within countries.

White (2002) examines mortality patterns across and within 21 high income countries from 1955 to 1996 and finds convergence in life expectancy between countries, but an unchanging average increase in life expectancy across all countries. As noted earlier, Oppen and Vaupel (2002) note that there has been little variation in the annual average rate of increase of life expectancy of 0.25 years per calendar year in the country with the highest life expectancy among the early industrializing countries for which there are reliable mortality data going back almost 200 years. But this relatively steady improvement in life expectancy at birth across high income countries is dominated from about 1875 until the 1950s by the rapid decline in infant and child death rates, due to the control of infectious diseases. After about 1955 the control of degenerative diseases among the older population begins to save more lives among the elderly than among the child population. Presumably many developing countries are reaching the stage today when further mortality reductions will also be achieved in these countries by preventing adults from dying of degenerative diseases, namely those associated with the cardiovascular system, cancer, diabetes, and respiratory illnesses, but there are few reliable cause specific mortality data to document these developments in the lower income countries.

Edwards and Tuljapurkar (2005) propose to study, in addition to the mean life expectancy in a country, the variance in the age at death within a life table cohort, and view this

variance in life span as an indicator of personal inequality in health. This measure of variance in life span at birth has declined as the mean life expectancy has increased, attributable mostly to the diminished variance in infant and child mortality across countries. The variance in life span for the cohort of survivors to age 10 , however, has not shown a similar tendency to decline within high income countries since 1960, and the level of this adult variation in mortality differs substantially across countries and by race or ethnic group within a country, such as the United States where medical care for the non elderly depends on employer subsidized private insurance. Where new health technologies are apparently reducing adult mortality, they appear to not have narrowed health inequality recently, as reflected by the variance in the length of adult life, i.e. after age 10. Analyses of adult mortality patterns using life tables seem to be uncommon for low-income countries, probably because the registration of deaths is incomplete, and they are often imputed on the basis of survey estimates of child mortality and thus do not yield reliable residual estimates of adult mortality. To account for the effects of health on economic growth Weil (2005) analyzes life expectancy from age 15 to 65, which is a plausible indicator of economically productive health in the working ages, but this does not improve the empirical foundations for his adult health indicator, which is essentially a residual imputation of deaths to adults which are not attributed to children dying, and may be particularly prone to errors in low-income countries.

The primary cause of death before the mid 20th century in more developed countries, and in most less developed countries until the end of the 20th century, were infectious and parasitic diseases. Today deaths due to these diseases are geographically concentrated in a few tropical

regions of the world with very poor health infrastructure, extreme poverty, and other unfavorable conditions. These geographic patterns of health and the prevalence of particular diseases, such as malaria, TB, and HIV/AIDS, have been used to account for development in a variety of ways. Poor health, short life span, and specific diseases can be treated 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 reported to be greater in countries for which life expectancy is longer, but these partial correlations 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 and Sala I Martin, 1995; Barro, 1997; Bloom and Sachs, 1998; Gallup and Sachs, 2001; WHO, 2001; Bloom, et al, 2004). If these inter-country comparisons are extended to interpret changes over time within countries (i.e. allowing for fixed country effects), invariant geographic and climatic indicators are, of course, not identified. It is only reasonable to expect unobserved factors which are correlated with levels and changes in measured health conditions to be also related to income levels and growth, for reasons not directly related to health, biasing upwards cross country regression estimates of health effects on income and economic growth (Weil, 2005; Acemoglu and Johnson, 2006). 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, which the social scientist must understand and explain if they are to clarify how these general health outcomes causally affect the economic development of countries, as illustrated in the stylized Figure 1.

Acemoglu et al. (2001, 2003) provide a historical dynamic framework involving a two-stage explanation for the diversity in development. In the first stage the health disease environment impacts the establishment of settlements which facilitate social institutions that favor or discourage long term economic growth. They observe that European colonialists settled permanently in temperate climates where their native agricultural crops and production practices could be transferred with the least investment in local adaptation, and they emphasize they also selected locations where diseases were least lethal to the European immigrants, e.g. North America, Argentina, Chile, and South Africa. Agricultural settlers accepted the concept of property rights for farmers and they replicated institutions of governance that corresponded roughly to those they had known in Europe. Conversely, where colonial regimes confronted tropical agricultural climates and the endemic diseases that were especially lethal for the European immigrants, the regimes resorted to extractive industries, which exported precious metals, natural resources, or slaves, and often depended on restrictive governing arrangements to assure a reliable supply of labor for export industries. The resulting institutional system in these "extractive colonies" did not facilitate long term development and these regions tended to become the laggards in terms of economic growth in subsequent centuries. Acemoglu and his colleagues hypothesize that the institutions evolved by Colonial regimes offer one explanation for the subsequent poor economic performance of colonial sub-Saharan African and some tropical areas of Latin America and Asia, if not the later collapse of Argentina. If their model is valid, the contemporary efforts to control tropical diseases and improve health in areas such as Africa will not necessarily achieve by themselves the economic benefits attributed by some

observers to these constraining health conditions (WHO, 2001), because the institutions which are beneficial for sustained economic development – protection of private property and good governance – might still be lacking.

To test their ideas further and assess how health directly affects growth in low income countries since World War II, Acemoglu and Johnson (2006) identify the time when new effective public health technologies were introduced in the world against seven major infectious diseases or causes of death for which the league of nations provides country specific estimates, and these discontinuities over time by disease are interacted with the initial prevalence of each disease in each low income country. These disease specific technological changes and initial severity of the disease become instrumental variables that account for a significant share of the increase in life expectancy (and in population growth) in the developing world from 1940 to 1980. But these predicted increases in life expectancy do *not* account for the level or growth in income in this period. They cast "doubt on claims that unfavorable health conditions are the root cause of the poverty of some nations." As already discussed, life expectancy is not a good indicator of the productive benefits of health or one which is well measured in many low income countries, and introducing discrete lags of 5, 10, or 20 years to allow the gains in life expectation to affect income growth does not necessarily capture the dynamic life course biological process by which healthier children improve the productivity of mature adults and thereby enhance the possibilities for economic growth. African countries are also under-represented in their sample, because of the scarcity of mortality data.

The opposite causal connection is explored by Pritchett and Summers (1996), who examine five year intervals from 1965 to 1985 for low income countries , examining a sample of 143 to 184 country-year observations. Their direct estimates, controlling for time period and adult schooling level, suggests an increase in income of 10 percent is partially associated with a decline in infant mortality rate of 1.9 percent. Because other factors could affect both income and infant mortality, they then rely on alternative instrumental forcing variables (IV), among which I find the most convincing is the "terms of trade" (Z in figure 1) as a determinant of income, but a variable which does not directly affect health. Presumably an increase in the price of a country's exports relative to the price of their imports is a "shock" promoting domestic income growth, but because export prices are determined outside of the country in the global economy, this shock should not be related to other factors affecting health of the domestic population. 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 ratio yields a second-stage estimate which is statistically significantly different from zero, implying a 10 percent increase in income is associated with a 3.5 percent decline in the infant mortality rate over the five year period. The validity of the investment ratio, however, as an instrument is not clear a priori, since it could be affected by many factors in the domestic economy such as weather, civil conflict, or development policies, all of which could reasonably affect health. When Pritchett and Summers consider five year differences in life expectancy as their dependent health variable, the direct association implies a 10 percent increase in income is associated with a smaller 0.15 percent increase in life expectancy, and even this direct (OLS) association is not significant. More relevant perhaps, none of the IV estimates for income's

direct effect on life expectancy differ notably from zero, a finding corresponding to Acemoglu and Johnson (2006). Although Pritchett and Summers conclude that "gains from rapid economic growth flow into health gains", the actual evidence they present suggests the direct linkage captured in the cross section of countries is not significant when the analysis is performed for five year changes in life expectancy *within* low income countries. Relying on the more sensitive measure of health in the form of infant mortality, there is a direct association though presumably biased, and when a credible instrument (terms of trade) is used to predict exogenous variation in national income there is no significant relationship. It should be noted that the authors do not attempt to find a suitable instrument which they could argue affects mortality and thereby influence productive health and output of workers, but which is arguably unrelated to national income, in order to assess empirically the strength of causation flowing in the opposite direction, from population health to national income. I conclude from these macro studies that there is as yet no convincing basis for identifying the relationships of interest from the existing fragile and misspecified measures of mortality and inadequately lagged relationships to income. The historical relationships Acemoglu et al (2001, 2003) describes between institutional developments and disease conditions during the period of colonial settlement may be intriguing, but it is not particularly relevant for explaining contemporary effects of health on income, or income on health.

Alternative Measure of Health Status and their Change : National and Individual Level

Contemporary and historical studies have documented from many perspectives early life health conditions experienced by the pregnant woman, her fetus, and young child are

significantly associated with the child's survival probabilities after age 50. If this early health effect on later life longevity also reduces morbidity and disability among adults, then about five decades after an improvement in maternal and child health occurs, adult health and productive potential should increase. Direct evidence of this long gestating effect of child health on productivity has not been estimated to my knowledge.[7]

However, the productive gains of early childhood health may emerge at a younger age, especially if health and other forms of human capital are complementary, and yield returns before middle age when mortality and disability would otherwise increase sharply. Several recent studies have found reduced morbidity of children is associated with their increased school attainment, while others have found impacts of early child health on cognitive test scores, and adult wage rates (Miguel and Kremer, 2004; Bleakly, 2007; Almond, 2006; Maccini and Yang, 2006; Glewwe, et al. 2001). A literature has also sought to link the birth weight of a child, which is highly correlated at low birth weights with child survival, to the child's cognitive performance, school attainment, adult earnings, and late life survival and health status (Alderman and Behrman, 2006). The most compelling evidence is based on differences in birth weight of twins and their later life outcomes, which purge estimates between twins of the omitted variable bias due to unobserved period and family fixed effects (Behrman and

[7] 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 then account for national income growth, but they rather lagged life expectancy in a period when its change was dominated by the declining trend in child mortality, by only five to twenty years. The lack of sufficiently long time series for mortality and subsequent income in either low or high income countries appears to limit for the time being this line of inquiry.

Rosenzweig, 2004; Black, et al., 2007). This literature is reviewed later in the paper 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 20th century, might be expected to impact economic growth after a lag of several decades. Moreover, declines in child mortality are frequently associated with declines in fertility within a decade or two, and are likely to facilitate economic growth if they increase women's participation in the market labor force as they reallocate time from child care and home production, and eventually increase life cycle savings and investments for retirement and the formation of human capital in their children. Change in life expectancy at birth is a poorly designed indicator of changes in the stock of health of the working age population that could be expected to affect economic growth, but how might change in the productive stock of health human capital be more appropriately measured?

The present discounted valuation of future production of a life saved might be a preferred measure of the value of health human capital accumulated by saving a live, applying a reasonable (i.e. 3-6 percent) discount rate to changes in future productivity. Such a present value calculation would undoubtedly assign a larger productive value to savings a life of a 25 year old adult compared with saving the life of an infant, whereas 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 discount future life-years and therefore attaches the greatest value to the earliest improvements in the survival of a cohort which has the longest remaining expected life. On the other hand, national income statistics do

not currently treat health or even schooling as an investment which can add to future growth. If national income growth statistics treated human capital as they do physical capital, then the present discounted value of lifetime total earnings (in market or nonmarket activities) would provide a basis for imputing the value of health improvements on future total national income. Valuing lives among the elderly will require an elaborated conceptual framework that may recognize a social utility function, a consumer's willingness to pay for life, and a public good aspect of public health (e.g. Usher, 1973; Tolley et al, 1994; Murphy and Topel, 2006). Observed wages are also an increasingly biased indicator of the value of time for the average member of an elderly cohort, when those who decide to continue to work are self selected and institutionally influenced by widespread taxes on earnings among those who are eligible for pensions (Gruber and Wise, 1995, 2004).

As noted earlier, the pattern of mortality change by age has evolved through time, possibly due to changes in health technologies, the economic benefits and health costs of urbanization, the diffusion of diseases in the world, and reduced severity of childhood illnesses , public and private health practices which reduced the spread of infectious disease, immunization against diseases in the 20th century, and finally the control of some diseases with antibiotics after the 1930s, and the eradication of some, such as smallpox, etc.(McNeill, 1976). Consequently, a year increase in life expectancy at birth is associated at different times and places with changes in different causes of mortality affecting persons of different ages; the age-specific mortality changes occurring today are likely to be quite different from those occurring fifty years ago, or two hundred and fifty years ago, with different implications for per capita productivity and

fertility.

Three phases of the mortality decline might be distinguished. First, the slow decline in death rates from 1750 to 1875 in countries that today have high income, which appear to have benefitted most age groups, though urbanization was an offsetting factor increasing mortality until the 20th century. Second, with the gradual acceptance of the germ theory of disease after about 1875 in Europe, and later in the United States, effective changes in medical and sanitation practices were introduced to combat infectious diseases, especially those causing frequent death in childhood. Broadly based public education, open to women as well as men, may have facilitated the introduction of practices of personal hygiene and child medical care that hastened the decline in childhood mortality and infectious morbidities, or at least women's education by the mid 20th century has become significantly related to lower infant and childhood mortality in surveys and censuses from most parts of the world. Finally, in the 1950s evolving medical knowledge led to the systematic research and development of drugs and medical interventions which along with preventive health practices began to rollback mortality due to degenerative as well as infectious diseases within older age groups, due first to cardiovascular disease, but also to digestive and respiratory illnesses, and eventually to reduce age-specific death rates for some cancers (Cutler and Meara, 2004).

But as already noted in this third phase of the mortality transition, the economic convergence in health status across or within high income countries has decreased, and the variance (inequality) in a life table cohort's duration of life at age ten stabilizes (Edwards and

Tuljapurkar, 2005). It remains to be seen whether low-income countries will be able to continue to catch up to the life expectancy achieved by the high-income countries as first noted by Preston as of the 1960s (1975). The rising costs of treatments for degenerative diseases is reflected by the large and growing share of national income expended on health-related goods and services in rich countries, and the luxury value of health to consumers. Unless these health-related expenditures in high-income countries have the consequence of reducing substantially the cost of health therapies available to low-income countries, the convergence in health status across countries is likely to diminish or peter out. However, the cost of anti-retroviral drug therapies for those with HIV/AIDS has decreased abruptly in the last five years, suggesting spillover benefits of modern medical technology may continue to benefit the world's poor who could not afford to pay the introductory prices. Nonetheless, to sustain the convergence in health across countries and within countries, growing foreign assistance and social redistribution of health and education costs will probably be needed, and intellectual property rights surrounding new drugs and medical inputs will have to be relaxed further for poor countries and poor populations. Otherwise the secular convergence in health, approximated tentatively here by the variance in life expectancy among adults at age 10, is likely to end.

The Transition and Health : Centrally Planned and Market Oriented Economies

Another major change in health conditions may be associated with the transition of economic and political systems from a communist centrally-planned economy to a decentralized market-oriented economy which may be more responsive to public interests operating through democratic institutions and independent media, but also distribute health services to the benefit

of the rich (Sen, 1999). As a new country the Soviet Union was credited with increasing the fraction of national resources allocated to higher education of women and men, for increasing the training of doctors and nurses, and providing free medical care for the entire population (Lorimer, 1946). Estimates of life expectancy in the Soviet Union, as well as Communist China, Vietnam, and Cuba are initially higher than would be predicted on the basis of their respective per capita national income (Caldwell, 1986), although these favorable comparisons typically exclude periods of revolution, war, and famine (e.g. in the Ukraine in 1931-34 associated with rural collectivization, and China in 1959-61 in the wake of the "great leap forward". The exceptional health gains achieved in these centrally-planned states may be attributed to public policies for effectively dealing with infectious diseases, reducing the private costs of basic medical care, and investing in complementary education, sanitation and water supplies.

However, after the Second World War the health systems of Eastern Europe and Western Europe had different degrees of success in reducing mortality. Guo (1993) decomposed mortality in these two regions by age, sex, and causes of death, and shows that from the 1960s to the 1980s the Eastern European mortality rates remained constant for women over age 40, and increased substantially for men over age 35, while at the same time these mortality rates decreased steadily in Western Europe for both men and women over age 35. Guo concludes that the West had greater success in coping with heart disease, and some success in lowering mortality due to cancer, digestive and respiratory system diseases among the middle aged and elderly. Guo concludes that the political weakness of the consumers of health care in the centralized Communist states prevented them from lobbying effectively to allocate public

resources to the health sector, while political leaders assigned higher priority to other sectors and objectives. As a result, public expenditures in Eastern Europe to treat and cure adult degenerative diseases were neglected compared with Western Europe and other high and middle income countries.

These differential health trends in Eastern and Western Europe continued in the transition period when the Soviet Union began to reorganize its economy. The exception occurred when Gorbachev raised briefly the price of alcohol in Russia (1985-87) which contributed to a dip in Russian mortality (World Bank, 2005). As the Soviet Union itself reorganized after 1989, life expectancy of Russian males declined by an unprecedented 4 years from 1991 to 1994, due to increased cardiovascular disease and a disproportionate increase in deaths attributed to trauma, i.e. accidents, homicides, suicides, and alcohol poisoning (Nolte, et al., 2004). Mortality of males age 35 to 64 decreased slightly from 1995 to 1998, but then increased again during the devaluation crisis in 1999 as the Russian economy contracted further and personal economic inequality increased. Russian women age 35-64 experienced the same time pattern of mortality changes, but the amplitude was less than for males, and life expectancy of women had by 2000 regained peak levels achieved in the 1980s, although this was not the case for males. Similar, though less pronounced increases occurred in adult mortality at the start of the transition in neighboring states of Belarus and Ukraine, but not in Eastern Europe or the Caucasus (Nolte, et al., 2004). The anomalous Russian experience of declining male adult life expectancy is unique outside of HIV/AIDS ravaged sub-Saharan Africa. What are the explanations for this development and do they offer lessons for other countries in their transition from centrally-

planned to decentralized market-oriented economies and health care sectors. The transition from a centralized authoritarian system to a more decentralized economy left the public health sector in Russia underfunded from central government budgets. Some of these same problems must explain the deterioration of health services in rural China by the 1990s leading to widely heralded Health reforms promised later this year, a topic of Professor Gregory Chow will undoubtedly discuss tomorrow. The bureaucratic incentives to distribute basic health services to all citizens in a stable centralized system were probably sacrificed in Russia as public health resources became scarce, and consumers demanding health services were allowed to make side payments to assure timely treatment. Undoubtedly the distribution of medical services became skewed toward the rich at a time when economic inequality was increasing in many areas. But the unpredictable transition toward privatization of health care may not have been the dominant cause for the increase in adult mortality and disability. Rather it was probably the uncertainty of the economy and arrears in the payment of wages and pensions which contributed to shortfalls in income and unfamiliar economic anxiety. New forms of corruption and the lack of government penalties for anti-social behavior may have also contributed to risky and self-destructive health-related behavior, including the excessive consumption of alcohol.

There is little evidence that widespread malnutrition or deterioration in the diet was responsible for the rise in mortality in Russia, and the continued gradual decline in mortality among infants and children under age 15, and the stability in mortality among those over age 65 suggest that a severe deterioration in the Russian medical and public health system was not the dominant cause for the rise in adult mortality, concentrated as it was among males.

Environmental pollution may also have been a contributory factor, but not one which could reasonably cause the observed upsurge in mortality among men age 25 to 64. One is left with non-quantifiable socio-psychological risk factors which result in stress and may have been expressed in excessive alcohol consumption and risky behavior among males (Brainerd and Cutler, 2004; Suhrcke, et al. 2005). Mental illness could have also contributed to the rise in mortality in Russia, which might also be reflected in increased morbidity, disability, and decreased labor productivity. But household sample surveys generally exclude persons in institutions, and in Russia it is believed that political considerations as well as conventional clinical assessments of mental illness are used as criteria for committing people to psychiatric institutions. In addition to premature death among adults in this period in Russia, there are indications of reduced participation in the labor force and diminished productivity of those in paid employment.

One possible indicator of health disability is not participating in the labor force during a person's prime working ages of 25 to 64. This could be due to poor health status which prevents them from working, or not working for a wage or salary. It is also an empirical regularity that adults with sufficiently low productivity, and hence low wage offers in the formal labor force, will tend to withdraw entirely from the labor force and work only in nonmarket activities or home production, especially in a period when customary social pressures to hold a job diminish. Individuals are also less likely to work in the labor force, if they have alternative means of support other than their earnings, from either a social safety net of welfare programs, or transfers from their extended family, or income from personal wealth. Labor force surveys for Russia are

able to document a widespread decline in male adult labor force productivity and participation during the 1990s, that occurred at about the same time that adult mortality increased.

Disability in Russia as of 2004 will be summarized here by the non-participation of the individual in the labor force, or not working for a formal wage[8], or working for fewer hours per month, or receiving a lower wage rates per hour worked, holding other factors constant which should affect their productivity, job opportunities, and nonearned income . The Russian Longitudinal Monitoring Survey confirms an unusual decline in participation and productivity of males over the age of 35 during the transition period after 1990. Figure 4 shows that eighty percent of Russian men age 25-35 in 2004 report themselves as in the labor force, whereas the proportion falls to 76 percent at age 35-44, to 71 percent at age 45-54, and to 49 percent at age 55-64. These male participation rates are lower than commonly observed in other high income countries, i.e. members of OECD. For women age 25 - 34, 72 percent participate in the labor force, increasing to 76 percent at age 35-44, and declining to 71 percent at age 45-54. Most women are eligible for a central state pension at age 55, and their participation in the labor force declines to 33 percent at age 55-64. The likelihood of participation (OLS or Probit) in the labor force in 2004 among males age 25 to 64 is negatively associated with self reports of poorer current health status, reports of the number of health problems in the last month, and being categorically classified by the government as disabled (Schultz, 2006).

[8] An hourly wage can only be derived for individuals who report monthly earnings and hours worked last month in the Russian Longitudinal Monitoring Survey. This segment of the "wage" labor force excludes the self employed and many working in agriculture.

There are two problems with interpreting these correlations between self-reported health status and labor force participation and productivity as evidence of a causal effect : the association between self-reports of health problems and labor force participation might be biased by the respondent's inclination to rationalize his lack of a job by reporting his health status as relatively poor, and this would lead to an overestimate (a downward bias) of the direct (OLS) negative estimate of the impact of poor health on labor force participation. Alternatively, measurement error embodied in reports of health status could add to the imprecision of these health indicators, and such classical measurement error in an explanatory variable would bias the direct estimate of the effect of poor health on participation toward zero (upward biased) (Bound, 1991**)**. Both of these difficulties in estimating an unbiased labor supply response to self reported health status could be corrected by using a valid instrumental variables to predict current health status, health problems, and disability classification for the individual, and then in a second stage analysis relate the predicted health status to labor supply or productivity. The instruments specified here to predict current health in the Russian Longitudinal Monitoring Survey of 2004 include seven chronic preexisting illnesses, a nervous breakdown or mental depression in the last year, and high blood pressure, all of which are jointly significant explanatory variables in the estimation of the first stage equations for the three current health outcomes.[9] Using these nine instruments to predict the current health status, health problems, and disability classification, all three of these current health indicators have a significantly larger effect on worker productivity

[9] An F statistic in excess of ten is a conventional standard proposed for the explanatory power of the instrumental variables identifying the first-stage prediction equation (Stock, et al. 2002). This criteria for sufficient power of the instruments is satisfied for the instruments in all three health indicator first-stage regressions, for either men or women in the 2004 RLMS (Schultz, 2006).

according to the IV estimates than implied by the direct (OLS) estimates, although both sets of estimates imply health problems decrease participation of men in the labor force, reduce their likelihood of working in a wage job, and reduce hours worked last month. The empirical regularities relating health to labor productivity for Russian women are qualitatively similar to those for men with regard to participation and working for a wage. The instrumental variable estimates for women are also larger in absolute value (negative) terms than the direct Probit-OLS estimated labor productivity derivatives. According to the interpretation of Bound (1991), this suggests that in Russia estimation bias due to measurement error of health dominates the bias due to endogeneity of health self-reports, and the direct associations of health and productivity understate the magnitude of the lost output in the Russian economy due to the health crisis.

To be more specific, a Russian male who is predicted to have one more health problem last month is estimated to participate 21 percent less often in the wage labor force, holding other determinants of labor supply constant, when the sample mean participation rate for males age 25 to 64 is 60 percent. For Russian females in this age group the wage labor force participation is 11 percent lower when she has one more predicted health problem last month, where the female sample mean participation rate is 57 percent (Schultz 2006: Tables 1 and 5). This study suggests that the rise in Russian adult mortality, especially among men age 25 to 64, is occurring at the same time that these adults have increasing health problems and these chronic health problems appear to be causally associated with their reduced participation in the labor force.

This reversal in Russia of the global trend of health improvements occurred at the same

time that Russian national income fell sharply during the economic transition. To my knowledge, no study has linked the collapse in Russian national income to the declines in adult health, or even to the more readily measured contraction of the labor force. Little study has focused on the adult health crisis and its implications for the Russian economy, other than to search for a medical disease-based explanation for the upsurge in male mortality. The disparity between the behavioral consequences of the transition on the health of men and women suggests a social-psychological aspect to the adjustments in living conditions that occurred during the transition in Russia. The Russian Communist state had been established for 72 years (1989-1917) erasing most memories of how individuals might cope for themselves with the uncertainties of a market economy or a labor market with wage arrears, whereas in the Communist regimes in other countries in transition in Eastern Europe and China many individuals had experience with the market economy or could recall how small farms and firms could function. It is perhaps understandable, therefore, that Russians had greater difficulties in adapting to the market transition compared with those in younger regimes. But to my knowledge, no research has identified what public policies or economic conditions were responsible for the health crisis, or has shown that alternative structural reform policies could have mitigated the health consequences in Russia.

IV. Micro Literature: Cross Sectional and Panel Data of Households and Individuals

Direct and indirect evidence is accumulating regarding 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 lifespan, but also affect in a

complementary direction cognitive performance, schooling, and thus affects the productivity of adults as measured by wages and earnings as well as life span. Research on this topic is extensively reported by epidemiologists, medical researchers, 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 reasonable to expect more social attention to be directed to pregnant women and the health environment of children in the first few years of life (Gluckman and Hanson, 2005). I can only summarize a few segments of this broad field, and suggest how the measurement of health has evolved, linking inputs and outcomes to economic behavior, key working hypotheses, and their statistical testing.

McKeown (1976), in one of the most influential early studies, 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, since the decline in death rates could not be explained by effective medical interventions or urban 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 morality up to the end of the 19th century was related to the improvement in nutrition and perhaps the reduced crowding of poor populations in urban housing.

Fogel (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 consumed to maintain basic

metabolism and physical maintenance activities, as well as used to fight off infections and disease. Only then are surplus calories available to support productive "work" activities or to allocate to 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 costs of additional calories begins to exceed the value of their marginal contribution to productive work. Increases in the consumption of calories may also be translated into rising weight as measured by a Body Mass Index (BMI)[10] which is initially associated with improved health and increased physical capacity for work. Eventually unproductive levels of obesity can develop, or energy-consuming leisure activities may increase and as a consequence maintain BMI despite the increased consumption of calories. Fogel (2004) compares these WHO derived calorie requirements to his historical estimates of the personal distribution of calories available to the populations of England and France from 1700 to 2000, and thereby offers an 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 other words, the current flow of calories into nutrition minus its contribution to work capacity are then related to hypothetical capacity to survive illness, where BMI can then be viewed as a buffer stock of health accumulated by the population. These health stocks are then approximated in terms of the physical growth (i.e. height and weight) of better nourished individuals. Fogel hypothesizes, as others have, that a worker's productive capacities respond to

[10] Body Mass Index (BMI) is typically measured by dividing the individual's weight in kilograms by their height in meters squared

their consumption of calories, but are eventually subject to diminishing returns, holding other factors constant. His subsequent research has documented how adult chronic health conditions improve for those who have experienced less nutritional deprivation and less exposure to infectious disease, especially as a child (Fogel, 2004). The challenge is to quantify those flows of nutrition, various claims on these calories, that together affect stature, and the pathways from these measurable indicators of health status that appear to be important for physical and mental development as well as later life expectancy.

Height, Weight, Body Mass Index, and Birthweight as Indicators of Stocks of Health

Anthropometric indicators of height and weight have gradually become key indicators for comparative studies of nutritional and health status of populations for different historical periods and populations. Height (HT) as an adult reflects long run childhood "stunting", and weight-for-height (BMI) represents a shorter run outcome associated with "wasting" determined by the recent balance of nutritional inputs versus the energy expended on potentially productive activities as well as calories used to combat infection and disease (Fogel, 2004). Adult height is thought to be largely determined by individual's very early development, and be more or less fixed by the age of four (Martorell, and Habicht, 1986). Because adult height is more or less stable over the life course from age 25 to 55, the age profile of heights in a cross section of the population suggest smoothly evolving trends in nutrition and exposure to infections (scarring), but these historic trends of development may be reversed by swings in mortality that cull from the survivors of a cohort less healthy individuals who tend to be of shorter height. Scarring and selection may thus offset each other under some conditions of extreme mortality crises, such as

the Chinese famine of 1959-61 (Schultz, 2004). Waaler (1984) documented that the risk of mortality relative to others in a age and sex group declines for taller Norwegians over the age of 50, with the pattern of declining mortality risk for those with greater height being approximately linear, and especially marked due to cardiovascular diseases, tuberculosis and chronic obstructive lung disease, as shown in Figure 5 for Waaler male sample (1970s) and for Fogel's Union Army Veteran sample (1870s) (Costa and Steckel, 1997). Elo and Preston (1992) conclude their review of the mortality literature : "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".

Life tables in low income countries for adults are often determined as a residual after controlling for the level of childhood mortality and may thus be unreliable and difficult to disaggregate to smaller groups in the population, whereas height can be measured both for aggregate populations and even for individuals from a single survey, recognizing the information signal contained on health in adult height is small. Height is also more objective than many self reported health indicators and therefore less likely to be biased by subjective considerations or cultural conditioning to acceptable health burdens, and may be better able to discriminate the health status of young and middle aged adults than their ability to perform physical activities of daily living (ADLs), which are widely used to assess functional disability among the elderly in high income countries (Schultz, 2005). Retrospective survey questions regarding childhood health conditions have not yet been used extensively in studies, but could be used as exogeous

instruments to explain later health status as it affects behavior and welfare of the adult (Haas, 2007). Panel data on the evolution of health outcomes and potentially exogenous shocks over time provide the researcher with information on changes in stocks of health, holding individual initial health endowments constant. But even correlations between changes over time in health and productivity may still not capture without bias causal effects, if the earlier outcomes are themselves endogenous or responding to prior choices and preferences of the parents and children, and to their family's available health care.[11]

The mean height across birth cohorts over age 25 as observed in a representative survey signals how health conditions have changed in the last fifty years in a closed population, namely one not affected by turnover such as immigration or emigration or mortality selecting on height. In a low income country where death registration is incomplete and mortality data are unreliable, height data on adults can be readily collected in a representative survey, and provide a historical window on the evolution of health human capital in the population, and time trends by ethnic and

[11] Case et al. (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 age 7-16, 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 widens (or accumulates) as the child ages, or inequality in health increases over the child's life cycle. Currie and Stabile (2003) estimate similar tendencies for the gradient between the health of children to widen with age in Canada, while Currie and Hyson (1999) examine the impact of low birthweight on schooling and labor market outcomes, as the children experience repeated health shocks. It is often difficult to interpret as causal this literature, which conditions the child's health and adult outcomes on many endogenous forms of household behavior over the life cycle. Even birth weight has been shown to respond as an endogenous variable that is determined by a variety of socioeconomic behavioral choices.

socioeconomic groups that do not change over a lifetime, that could be interpreted as inequalities in health status (Schultz, 2003). Figures 6 and 7 report the pattern of average height for rural/urban/total female populations by five year moving averages of year of birth in Ghana (1987-89), a country experiencing little economic growth after its independence in 1957, and Brazil (1989) a country that has grown relatively rapidly since 1940. The economic gains in per capita national income would appear to be an obvious explanation for these different rates of increase in height among younger women in the two countries (Schultz, 2005).

Heterogeneity of Height as a Measure 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 another component which is not readily explained from observed socioeconomic variables, or affected by measures of environment risk. Only about a tenth of the variation in 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, while the rest of the variation in height can be attributed to characteristics which may be genetic in origin or conditions affecting unpredictably biological development, as well as errors in survey measurement of actual height.

By comparison, schooling has been interpreted as another form of reproducible human

capital created by investments of student time and social resources in school attendance. A larger fraction of the variance in schooling tends to be explained by family and student exogenous characteristics and family access to education, and a smaller fraction of the variation in schooling is attributed to intrinsic ability of the student and measurement error as they affect the individual productivity in the labor market or wage rate, compared with analogous studies of height as a latent indicator of health human capital which may also affect adult productivity and wages (Schultz, 2003). 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, because it is assumed that ability and schooling are complements in raising the productivity of labor and wage rates of an individual, and individuals with more ability are likely to complete more years of schooling. But studies designed to purge the wage function of this omitted ability variable bias by using instruments to predict schooling that are not likely to be associated with ability have yielded estimates of private wage returns to schooling that do not differ substantially from those estimated directly by ordinary least squares and implicitly neglect any correction for ability bias (Card, 1999; Duflo, 2003; Schultz, 2002, 2003).

Several studies have introduced height as a measure of human capital or a productive characteristic of the worker as an argument in the wage function, and they find an added centimeter in height is associated with about a 1.5 percent increase in wages in low income countries and as little as 0.5 percent increase in a country such as the United States, presumably because of diminishing returns to height at higher levels of health and nutrition (Strauss and Thomas, 1998 ; Schultz 2002). Because BMI could possible affect wage productivity while at the

same time a more productive worker could spend more on food which would raise his BMI, an argument has be made to treat BMI as endogenous in the wage function and identify its effect with instrumental variable methods, such as the local prices of food. Thus, studies have used instrumental variables to predict individual variation in BMI that is then used to explain variation in family income or individual wages. Generally the IV estimates of the effect of BMI on wages are significant and positive, though subject to diminishing returns (Strauss and Thomas, 1998). Also a number of studies have used instruments from family background or local health services, local disease risks, or weather shocks at birth to predict adult height as an explanatory human capital variable in estimating the determinants of the wage rate of individuals. These instrumental variable (IV) estimates of the log wage returns to height tend to also be several times larger than the direct ordinary least squares (OLS) estimates of height on log wages, holding constant for post-schooling potential experience and sex (Savedoff and Schultz, 2001; Schultz , 2002, 2003, 2005; Maccini and Yang, 2006). Several possible explanations may be considered: (1) the measurement error in height in household sample surveys may be relatively large, leading to a downward bias when height is treated as measured without error in OLS estimates; (2) the residual or genetic variation contributing to height may be treated as an omitted variable in the wage function, and it is naturally positively associated with observed height, but might be negatively associated with labor productivity or be a substitute for reproducible health human capital; or (3) measured height is fundamentally heterogeneous and the health human capital component identified by variation in parent socioeconomic characteristics and local health conditions of the family contributes to larger gains in health and wage productivity than does the residual unexplained variation in height. Although measurement error in anthropometric

indicators may not be negligible, and indeed repeated measures of adult height from a panel survey in Cote d'Ivoire did yield larger estimates of measurement error in height than for years of schooling, but not of a magnitude to explain the larger IV than OLS estimates for height's effect on wages (Schultz, 2003). The second explanation posits an implausible negative effect of residual height on productivity. I regard the third hypothesis as the most reasonable that height as a latent variable for health human capital captures the effect of early biological development processes that also contributes to the individual's later health, productive capacities, and longevity, but the directly estimated (OLS) effect is attenuated by the dominance of the genetic variation that functions as measurement error embodied in height.

Investments in health human capital are thought to occur throughout life, but as determinants of height, they appear to make a critical difference in biological development during specific periods in fetal development in utero and during very early childhood. Height is likely to be affected by household resources and the environment of the mother during pregnancy and during the child's first few years, and possibly interacted with her education which may facilitate her adoption of best health practices for her child (Barker, 1992, 2005; Strauss and Thomas, 2007; Gluckman and Hanson, 2006; Rosenzweig and Schultz, 1983). Childhood height also forecasts adult height and is generally correlated with lifetime health and life span, mental or cognitive capacity, learning of skills, and productive outcomes for the individual over the lifecycle (Martorell and Habicht, 1986; Elo and Preston, 1992; Floud, et al. 1990; Komlos, 1994; Fogel, 2004 ; Schultz, 2003, 2005, Maluccio, et al. 2005; Almond, 2006; Cuno and Heckman, 2007).

Weight is generally converted into a body mass index (BMI), defined as the weight in Kg. divided by height in meters squared. BMI normalizes weight and thereby reduces its multicollinearity with height, facilitating joint estimation of the partial association of both BMI and height with health, wage rates, and other life course outcomes. Waaler (1984) in his study of the Norwegian population (1963-1979) also showed how the relative risk of mortality by age, sex , and cause of death generally followed a U shaped pattern with respect to BMI, with above average risks of death associated with BMIs less than 21 and over 29 (Figure 8). Reporting these patterns for specific causes of mortality among persons over the age of 50, he could link the effects of BMI on health to the functioning of particular organs and resistence to specific diseases. As obesity has become more prevalent in high income countries and a growing number of low income countries (e.g. Mexico see Strauss and Thomas, 2007), it may be recalled that individuals with very low BMI are still the most likely to die in broad regions of the developing world, including South Asia and sub-Saharan Africa. There are few references to other large studies that replicate Waaler's association between height and BMI and older adult mortality in other countries, or for that matter over time within a country, but they are necessary to generalize Waaler's findings to low income countries today.

Dejun Su (2006) allows a comparison over time in the Waaler curve for U.S. white males age 50-59. He relates the overall mortality risk to BMI, within much smaller samples than Waaler analyzed, based first on the Union Army Veteran Survival sample followed from about 1870 to 1890, and then for a representative US survival sample from the 1971-74 NHANES-I to the epidemiological vital status follow up survey in 1992. Su reports, as did Waaler, a U shaped

variation in the relative risk of death with increasing BMI, measured over a 18 year period (Figure 9). Using a variety of methods to fit his quintiles in BMI to relative mortality rates in both 1870 and 1970, Su shows that the healthier BMI values appear to have shifted to the right or to higher values of BMI, between these two observations separated in the United States by a century. The optimal BMI , namely that with the lowest relative mortality risk, increases from about 21 in 1870 to nearly 26 in 1970, although the lowest mortality segments of the Waaler curve are quite flat. Linearlly interpolating values between the quintiles Su reports, individuals have at least 20 percent higher mortality than the average in 1870 if their BMI is 19 or less, whereas this elevated risk of mortality (i.e. 1.2) is associated in 1970 with a BMI of about 21, an increase of about two units of BMI. The relative mortality risks in 1870 suggest a 20 percent higher than average mortality occurs for union veterans whose BMI is 27 or more, whereas in 1970 individuals with BMI of 31 or more are observed to experience mortality 20 percent higher than average. This mortality threshold shifted to the right by about four BMI units in this century. If these small samples of 800-600 individuals are sufficient to draw conclusions, in this century the mortality minimizing level of BMI has increased, and of course the proportion of the U.S. population at higher values of BMI has also increased, shifting the population distribution to the right. Many factors could have contributed to this shift in the Waaler curve for BMI in the US, and to the difference between Norway and the US in the 1970s, but I do not know of research on this development.[12]

[12] The distribution of diseases and health conditions causing death may differ, public and private health institutions and health related behaviors 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 addition to height and BMI, birth weight or birth weight adjusted for gestational age, is a measure of fetal growth rate or rate of uterine development which is often related to later life health status and economic performance as explored in the next section. The relationship between birth weight and developmental consequences for child survival, health status, schooling attainment, and economic performance of the mature child can be interpreted as consistent estimates of the causal effects of birth weight only if parents have no differential effect on a child's birth weight. More realistically, parents do influence birth weight by their prenatal care and most distinctly by their smoking behavior (Rosenzweig and Schultz, 1983). It is, therefore, likely that birth weight will then be correlated positively with other unobserved child health and developmental inputs, because all of these parent health behaviors may be affected by unobserved parent preferences regarding investments in child health and development. Other unobserved constraints affecting the family and its children may also impact both birthweight and subsequent health inputs for the children. To avoid this dilemma Rosenzweig and Schultz (1983) propose using exogenous local prices of health inputs and the availability of maternal health services as instruments to explain variation in parent provision of prenatal health inputs, which are then used to explain birth weight.

The most compelling basis for identifying variations in birth weight that are not affected by parent preferences or unobserved constraints on the family involve comparisons between identical twins. Birth weight differences between homozygotic or identical twins that share the same genes, period of gestation, mother's health status, household resource constraints, and parent preferences for health, can only capture the exogenous effect of in utero nutrition and

uncontrolled health conditions and thus cannot reflect endogenous parent choices. But even when identical female twin differences in adult height, adult BMI, schooling attainment, and log wage rates at about age 40 of the child are regressed on differences in fetal growth rate (birth weight in oz./weeks gestation) in a large U.S. twin sample, these estimated within twin effects of fetal growth are significant, except the effect on adult BMI (Behrman and Rosenzweig, 2004: Table 2). A one pound increase in full term birth weight is equal to a .4 ounce per week of gestation increase in fetal growth rate (a standard deviation in the twin sample). This increase in fetal growth rate is estimated to cause a .26 year increase schooling or a gain of 1.9 percent, and to increase adult height by 0.6 inches, and raise the wage rate by 5 to 9 percent at age 42. The effect of fetal growth on the log wage is larger for the lower half of the birthweight distribution, suggesting that if increases in birth weight could be achieved among lighter twins, the life cycle productive benefits would be proportionately larger for these children than for the heavier children.[13] Their analysis indicates that fetal growth and presumably nutrition is a significant pathway for lifetime effects on health and economic outcomes of the adult, but this literature does not shed much light on how health inputs or behavior of parents might affect birthweight, or be targeted to improve the birth weight of the lighter twins.

[13] Finally, they consider the intergenerational effect of mother's fetal growth on the twin's child's birthweight, by estimating the intergenerational relationship for all of the twins who became mothers. The intergenerational correlation is significantly positive, indicating a persistence in birthweight 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 birthweight 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 for form of birthweight.

Black et al. (2007) examine Norwegian births from 1967 to 1997 to estimate the effect of within twin birth weight differences on infant health indicators (e.g. APGAR), completing high school, and labor market success. They find impacts of birth weight, presumably related to fetal growth rate, and they observe similar estimates for monozygotic twins and non-identical twins, suggesting the genetic component of the variance in birth weight for non-identical twins is not a differentially important determinant of birthweight as it affects these later life outcomes of the child. They also can assess IQ for males who were required to take a military exam at age 18-20, and find within twin ten percent differences in birthweight are associated with 1/20 stanine difference in IQ .[14] With such substantial causal effects of birth weight on human development and productivity, Alderman and Behrman (2006) make the economic case for policies that reduce the incidence of low birthweight children in low income countries. However, as with most of this literature the precise policies that would achieve this objective of reducing the proportion of low birth weight children is not clarified.

As suggested earlier, studies across all births of the relationship between birthweight, height, or BMI, and later life health outcomes and socioeconomic performance are difficult to interpret as a causal relationship, because these early child health endowments are affected to

[14] Males with 10 percent greater birth weight are 1.2 percent more likely to be full time workers (Table III). Estimates of the birthweight 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 one-year mortality, APGAR score, and full time work. A ten percent greater birth weight within twins is associated with only a one percent 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 .15 .

some degree by maternal health and behavior, such as smoking (Rosenzweig and Schultz, 1983). Moreover, these anthropometric indicators of physical growth and endowments may also embody effects of parent preferences between children, and embody compensatory or reinforcing investments in a child's health in response to the children's initial endowments and early development, including their gender. Consequently, these anthropometric indicators of physical growth are not exogenous except in the restricted sample of twins with fixed twin effects, but are likely to reflect parental choices in typical cases informed by their understanding of the child health production technology and their preferences for different health and welfare outcomes for their children, which can affect postnatal health investments in their different children.

The task of estimating how height, BMI and birthweight affects the child's later adult's life requires that the researcher first understand what exogenous factors motivate variation in these early health indicators, and more specifically the variables that affect these health indicators which can be influenced by policy to encourage investments in health human capital. How is health human capital produced? For example, good rainfall in a location in Indonesia in the year before an individual is born may be associated with an increase in height for this adult. In Indonesia local rainfall deviations appears to predict the height of women born locally in that year, and also which women marry a taller husband, who has higher expected earnings, and will contribute to her residing in a household with higher per capita consumption (Macinni and 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 variable, would mix together genetic factors permutating height which could be less closely related to lifetime productivity than the local

rainfall induced component of height, which could be thought of as simulating a quasi experimental effect of variation in health human capital. It has been observed in several other country studies that by using socioeconomic background characteristics of the parents, and of the local health infrastructure, and disease environment at birthplace, as instruments for an adult's height and BMI, the estimated effect of height and BMI on log wage rates is a much larger positive value than if the wage effects of height and BMI are estimated directly (i.e. OLS) (Savedoff and Schultz, 2001; Schultz, 2003, 2005). This tendency for instrumental variable estimates of the productive payoff to anthropometric health human capital indicators to be larger in absolute values than the simple partial correlation is consistent with the previously advanced hypothesis that the reproducible health human capital component of health identified by socioeconomic instruments or weather operating on income has a larger beneficial effect on the child than does the unexplained residual associated with genetic variation in height. 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 must be filtered through the choice of an appropriate instrument to approximate the effect of economic policies which might effect the develoment capacity of an adult through its effect on fetal growth.

Table 5 reports for three countries, Ghana in 1987-89, Brazil, 1989, and the United States in 1989-93, the coefficient on height in centimeters in a standard log wage equation, separately for men and women, age 25 to 54 who report hours and earnings. The first column (a) reports the

direct ordinary least squares coefficient which implies a male one centimeter taller receives a wage in Ghana that is 1.5 percent higher, and females 1.7 percent higher. In Brazil the association is 1.4 and 1.7 percent higher wages associated with being one centimeter taller for males and females. In the United States where nutrition is better and people are taller on average (ten centimeters taller than Brazil) a gain of one centimeter is associated with 0.6 percent higher wage for males and 0.4 percent for females. Columns (b) reports the instrumental variable estimate based on height predicted on the basis of regional and household characteristics at birthplace, and they increase the estimated effect of height on log wage on the order of five fold. Clearly, the systematic variation in height associated with socioeconomic background and regional birthplace are much more steeply associated with wage variation than the entire distribution of height, and if parent years of schooling is added to the list of instruments in Ghana and the United States, the relationship of the estimated IV effect of height remains roughly the same but tends to becomes more statistically significant (Schultz, 2002). If the regional and birthplace characteristics identify the reproducible health human capital component of height, and the IV estimates in (b) or (c) therefore omit the residual genetic variance in height which is less closely related to latent health than the socioeconomically correlated component, then the relevant effect of nutrition and health interventions that increase height would be represented by these much larger IV estimates.

Table 6 reports the joint estimation in Ghana (1987-89) of the wage effects of four forms of human capital: years of education, migration from birthplace, adult current BMI, and adult height, first assuming the are homogeneous, exogenous, and measured without error by using

ordinary least squares as reported in columns (1) - (4) . The first concern is that because the four forms of human capital tend to be positively associated across people, the conventional approach to estimation of wage functions which omits several of these human capital factors would be inclined to attribute too large an effect on productivity to the included form of human capital. The conventional returns to schooling are only moderately diminished by the inclusion of the other three forms of mobility and health human capital, declining for males from .052 to .044. But if these forms of human capital are endogenous, heterogeneous, or measured with error column (5) provides the consistent instrumental variable estimates based on the individual's birthplace health and educational environment, and parent education and occupation. Although this 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 education or migration, it generally increases the coefficient on BMI significantly, and increases the coefficient on height several fold. 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 residual variation in these anthropometric indicators of adult health associated with genetic variability.

Table 7 illustrates again 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. Wage function estimates for Brazil would therefore suggest that the adult gains in height are able to explain male wage growth of 4.1 percent per decade in the last 30 years and female wage growth of 5.8 percent per decade. The

advance in schooling is associated with an increase in wages of males of 16 percent per decade, while for females the schooling gained would explain a 22 percent growth. Although both are large effects, the impact of increased schooling on income growth, according to these estimates, is able to account for two to four times more growth than that associated with the increase in height health human capital (Schultz, 2005).

To the extent that the instruments represent credible policy variation that can be changed to increase health human capital through improving fetal growth, the instrumental variable estimate is the more relevant private wage return on the investment in this form of health human capital. This interpretation implies that the anthropometric health human capital indicators such as height and BMI are measures of health capacity, though potentially measured with much error at the individual level. They reflect on average a distinct secular improvement in fetal and early child development which occurs when nutrition and health and income conditions improve as in Brazil. Replacing these general parental characteristics with more specific policy initiated conditions in the location of birth, such as the availability of nutritional supplements or vaccination programs, would be a next step in this line of research to link the survey instruments to actual policy measures at the local level of the household. The resulting estimates would then be more credibly the "local average treatment effects" that could be expected from these measured and therefore more manageable policy interventions (Imbens and Angrist, 1994).

V. Pathways between Early Development of the Child and the Productive Adult

Much new research is being undertaken linking the early health environment of

individuals to their longevity, health status, and economic performance, and relating the consequences specifically of health for welfare outcomes and productivity over an individual's lifetime. This is a brief and selective summary reflecting my understanding of this large multi-disciplinary literature. More detail is found in Strauss and Thomas (2007) from an economic perspective, and Gluckman and Hanson (2006) from an epidemiological and medical perspective, though approaches to statistical modeling differ between fields.

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, due disproportionately to the failure of specific systems of organs, such as the heart and lungs (Barker, 1992). However, many factors could be related to birthplace health conditions and to later adult chronic health problems, reducing the credibility of these early epidemiological studies as tight causal tests of the plausible hypothesis. More discriminating tests of the hypothesis have sought to specify a "narrower" pathway for a "treatment" of the fetus, and differential effects on the treated and on a suitably defined control population, followed often in a representative sample or census. 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,

problem of sample selection bias arise, simply through survival removing from the birth cohort the more frail individuals, or due to other forms of sample selective attrition, such as due to migration or nonresponse.[15]

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 in which those in the Northern hemisphere, such as Denmark, who are born in the spring have below average life expectancy at later ages. [16] This is consistent with the peak rate of growth of the fetus occurring in the third trimester, and seasonal fluctuations in the nutrition and health of the mother being more critical during the winter or early spring months for the healthy development of the organ systems associated with the fetal growth hypothesis (Barker, 2005) . The seasonal pattern among natives in the Southern Hemisphere, such as Australia, is roughly reversed. Moreover,

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[15] 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. The effect of a nutritional insult to the fetus may be masked, however, by a selection effect of survivors who might be healthier than average fetuses and hence more likely to survive. In other words, if the most frail or damaged die before birth or before reaching the adult age when schooling and wages can be measured in a census or survey, the effect of the nutritional insult might be offset to some degree by the selection culling effect, as could explain the relatively small sum of these effects on the height of cohorts corn during and immediately after the Chinese famine of 1959-61 (Schultz, 2004). 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. The first example I know of is by Rosenzweig and Wolpin, 1985, though earlier work by economic historians is likely.

[16] 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 effect would conceal the pattern noted by Doblhammer (2004) which is consistent with the fetal origins hypothesis.

migrants born in the North and residing the South exhibit month of birth effects on their elderly life span similar to non-migrant Natives in the North, suggesting the regional effect of birth month on elderly longevity is imprinted through fetal growth and early development, and cannot be attributed to the disease environment or health infrastructure affecting elderly mortality differently in the two hemispheres. What specific diseases, health conditions, and institutions are associated with the higher elderly mortality through fetal growth rates remains an active areas of research. How are these limits to growth translated into higher mortality among the elderly, what chronic and acute health problems are observed with advancing age, and how do they affect the economic productivity of adult workers and impact their current consumption and health investments, and health inequalities by socioeconomic status? If seasonal variations in these conditions are important, then other insults to fetal growth may be caused as well by drought, floods, weather, pollution, epidemics, or radiation accidents, encouraging more narrowly focused analyses of cohort data to identify the consequences of these "quasi natural experiments" on aspects of the fetal development processes that may have measurable impacts on the mortality of older populations, and on other life cycle effects such as schooling, cognitive skills, earnings capacities, and consumption opportunities.

A study of the 1918 flue 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 the birth cohort. The severity and timing of the flue infections varied by states in the United States providing possible instruments to identify the likelihood a child born before, during, and after the pandemic had been

affected by the infection of her mother in utero. The cohort born from January to September 1919, in the wake of the pandemic, exhibit many deficiencies which can be documented in the Population Censuses of 1960 and 1980 and attributed to the epidemic. For example, the children of mothers who were likely to be infected were about 15 percent less likely to complete high school, while males in this effected birth cohort have wages which are 5 to 9 percent lower.[17]

Historical studies have frequently found that periods of economic and health crises are associated with birth cohorts that experience greater middle age mortality that do neighboring birth cohorts somewhat older and younger, associated often with cardiovascular and lung diseases and type 2 diabetes (Elo and Preston, 1992). Alters and Oris (2006) report in several preindustrial Belgium communities 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 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 at birth and at the end of their working career.

[17] In another study of a natural experiment affecting fetal development, Almond et al. (2007) analyze data on the radioactive fallout from the Chernobyl accident of 1986, as geographically distributed by rain and wind across Sweden, and find it's 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.

Preston et al. (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 20th 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 without initially the 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. Crimmins and Finch (2006) suggest that the reduction in childhood infections with their burden of inflammation is responsible for growth in adult height and longevity of birth cohorts over the 20th century, as portrayed in their biological representation of health external (observable) and internal determinants of health as shown in their Figure 10.

Van den Berg et al. (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 matter for later health and reduced 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 diverse literature has made less progress in describing how specific policies can effectively protect individuals during this vulnerable stage of fetal development and mitigate the longer run consequences on lifetime health, productivity, and longevity.[18]

VI. Conclusions

Environmental conditions existing at the time and location of the conception and birth of a child are related to the child's gestation, height and weight at birth, and these characteristics of the infant help to explain early child health endowments which are linked with increasing confidence to the child's later health status as an adult, their adult height, their achievements in school, IQ, labor market productivity, reduced middle and late age mortality, and delayed onset of many chronic health limitations which can be expected to impact labor force participation and productivity per hour worked or wage rates, as well as welfare. These delayed consequences of the latent health capability arising from ample fetal growth suggest the severe challenges facing those who want to learn how health human capital accumulates over a generation and is

[18] Contrary to expectation some studies find contemporaneous pro-cyclical movement in mortality with the business cycle in high income countries. In the 20th 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 pro-cyclically in the United States. 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. There are many studies at the macroeconomic level that also cast doubt on the robustness of the contemporaneous relationship between income growth and gains in life expectation, or even improvements in infant mortality (Deaton, 2003). This does not entirely dispel the idea that in the long run income growth allows for improvements in consumption which are likely to reduce eventually some forms of mortality, though clearly institutional arrangements and the diffusion of knowledge about the process producing health are very important as well.

transmitted between generations and the appropriate role of the state to encourage this early form of investment in child health. How does this health human capital releases economic returns to the individual and possibly create broader returns to other family members and society and thereby contribute to what we call modern economic growth.

Cross country macro economic comparisons do not appear capable of refining our understanding at this time of the specific pathways through which technological change, medical inputs, consumption patterns, and health-related behaviors might be channeled by the design of health insurance or subsidized by the state to improve important health outcomes, and enhance the productive potential of the working population. 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 documented, and then only at the microeconomic level of the individual. One potential reason to pursue aggregate comparisons is that they may yet become sufficiently focused to shed light on the productive spillovers of health status that are not entirely reflected in individual output and wages, and might otherwise be omitted from current microeconomic studies, but possibly could be captured in aggregate data for communities, health administrative areas, or states.

The most promising avenues for research on the connections between health and development are currently being discovered through the analysis of survey and census data for individuals and households. They must be augmented by the collection of matched biological and

health information to better define the causal pathways from the fetal growth hypothesis to the life course outcomes of interest to economists and health scientists. Realistic and replicable policy and program interventions can then be documented in the areas where individuals are born, and new programs implemented experimentally and thus potentially evaluated. However, where the levels of health are lowest in the world, and where health interventions are expected to be most valuable, and the contribution to this area of research would be greatest, the necessary panel survey data focused on health and economics are most scarce or nonexistent.

I was delighted to learn a few weeks ago that the National Institutes of Health in the United States has assigned its highest research priority score to start funding next year of a Health and Retirement type Survey in China, which may in a few years time be expected to contribute importantly to research on these issues in China, while holding the Chinese researchers to the highest academic standards.

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Figure 1: Scatter-Diagram relations between life expectancy at birth and national income per head for nations in the 1900s, 1930s, and 1960s

Source: Preston (1975)

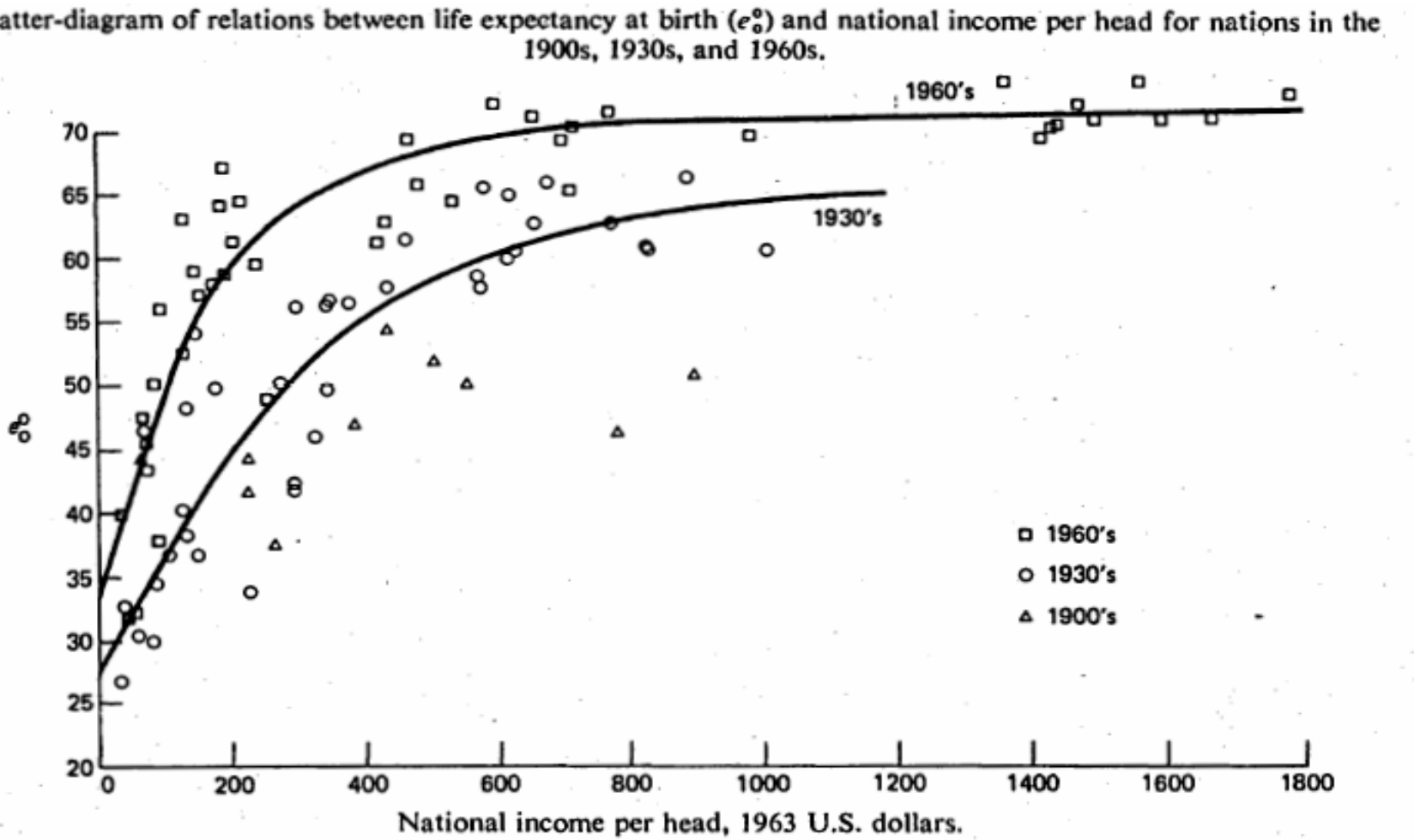


Figure 2: Life Expectancy and GDP per capita, Highlighting outliers in 2004

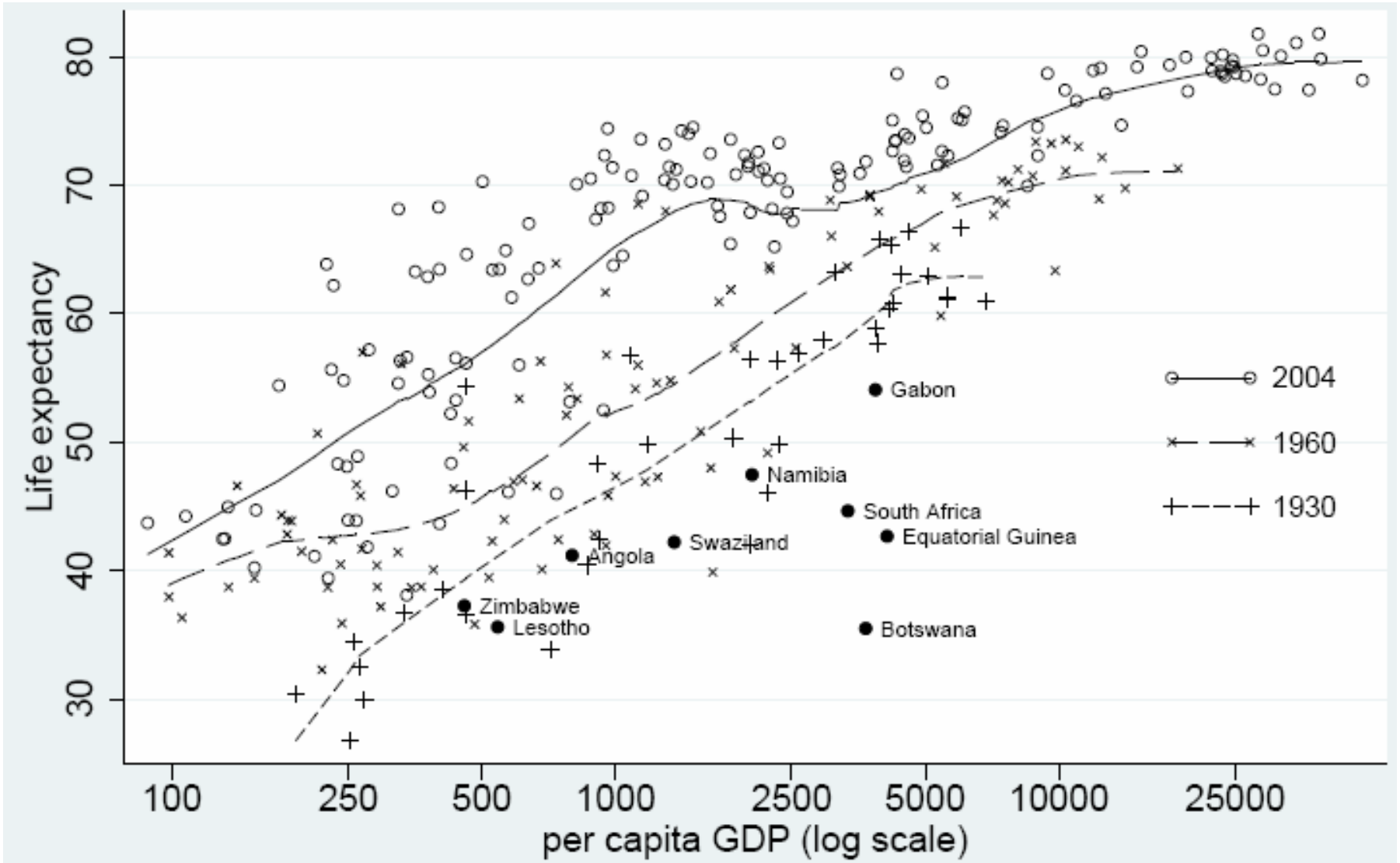


Figure 3

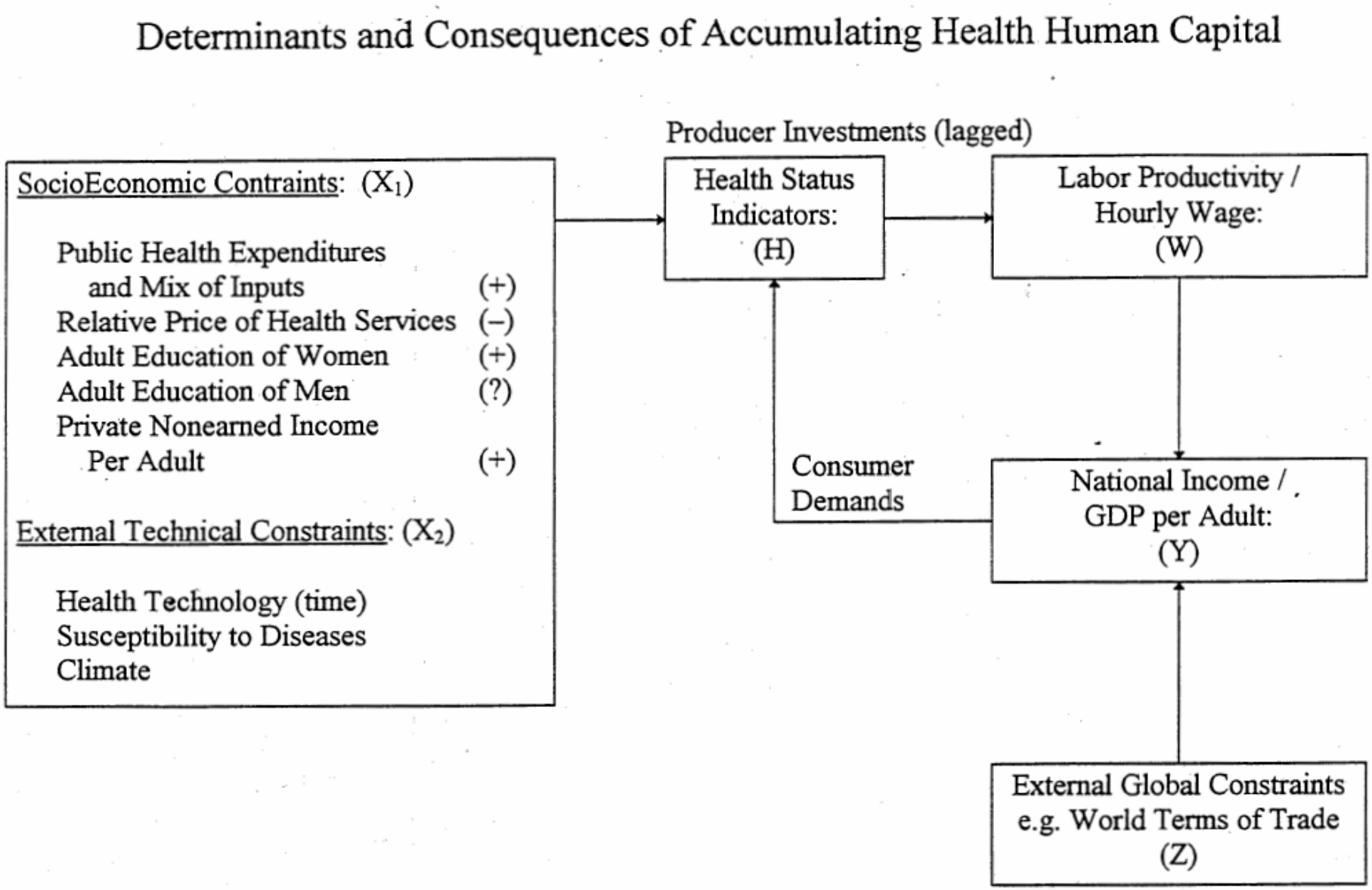

Determinants and Consequences of Accumulating Health Human Capital
Producer Investments (lagged)
SocioEconomic Contraints: (X1)
Public Health Expenditures and Mix of Inputs (+)
Relative Price of Health Services (–)
Adult Education of Women (+)
Adult Education of Men (?)
Private Nonearned Income Per Adult (+)
External Technical Constraints: (X2)
Health Technology (time)
Susceptibility to Diseases
Climate
Health Status Indicators: (H)
Labor Productivity / Hourly Wage: (W)
Consumer Demands
National Income / GDP per Adult: (Y)
External Global Constraints e.g. World Terms of Trade (Z)

Figure 4: Economic Activity of Russian Population

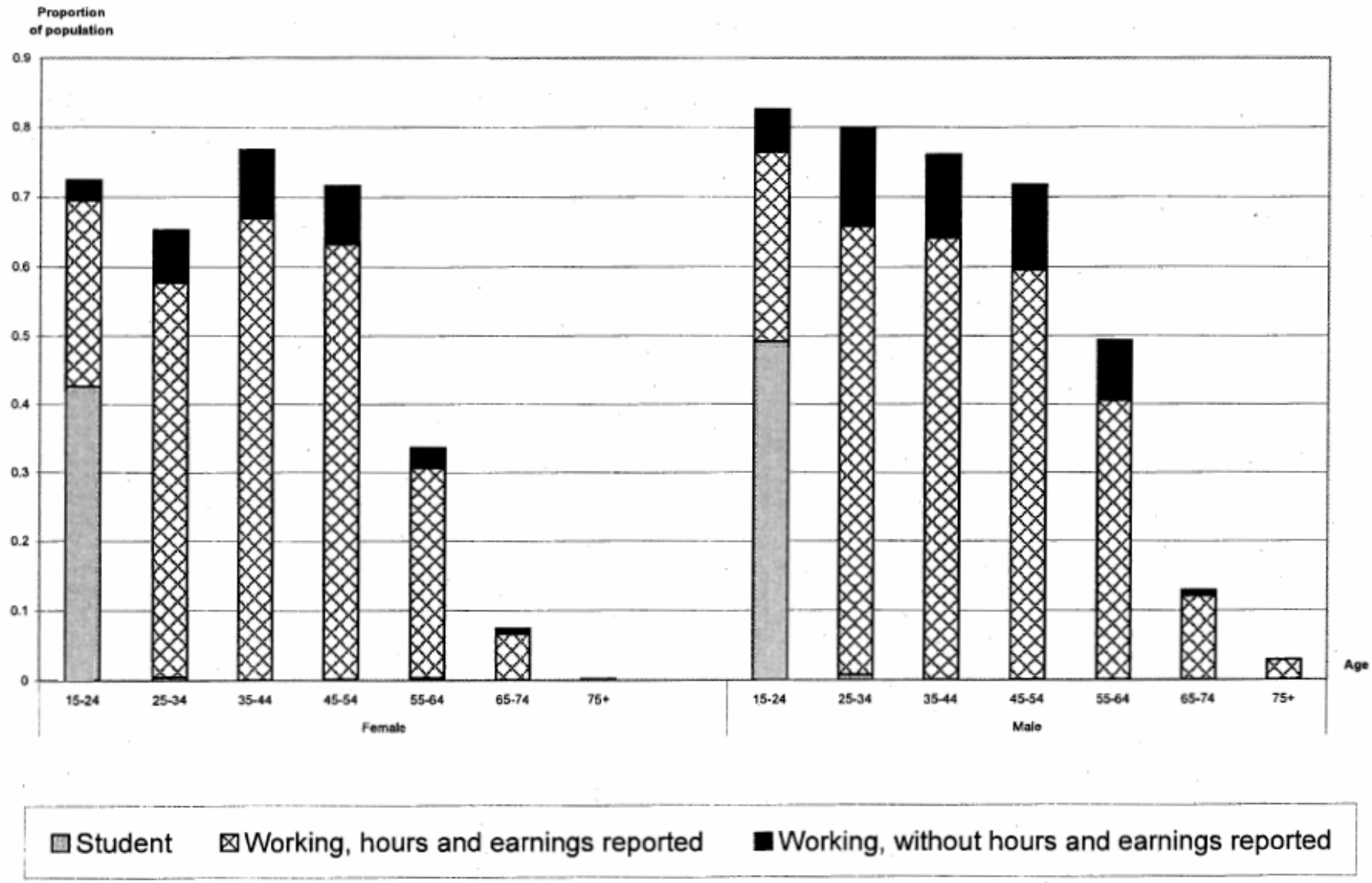


Table 1

Russian Longitudinal Monitoring Survey for 2004: Labor Market Partcipation, Hours and Wages by Age and Sex

Sample, Sex & Age	Total sample size	Proportion working in 30 days	Proportion report hours and earnings	Hours per month	Hourly wage rate	Montly earnings	Log hours	Log wage rate	Log earnings
Males, Ages:									
15-24	495	.66	.54	190	35.7	6261	5.20	3.27	8.47
25-34	969	.80	.66	187	45.3	7537	5.18	3.49	8.67
35-44	814	.76	.64	189	45.2	7511	5.18	3.43	8.61
45-54	736	.72	.60	187	39.4	6827	5.19	3.30	8.49
55-64	434	.49	.41	166	43.2	5890	5.04	3.37	8.41
65-74	391	.13	.12	164	24.0	4061	5.08	2.96	8.04
75 or more	140	.029	.029	154	20.0	2800	5.00	2.88	7.88
Total 25-64	2953	.72	.60	186	43.6	7190	5.17	3.41	8.58
Females, Ages:									
15-24	618	.52	.47	164	27.7	3994	5.02	2.99	8.01
25-34	1071	.65	.58	162	31.0	4567	5.03	3.09	8.12
35-44	903	.77	.67	166	30.3	4734	5.06	3.08	8.15
45-54	1021	.72	.63	164	36.6	4741	5.02	3.14	8.16
55-64	637	.33	.31	163	27.0	4202	5.03	3.04	8.07
65-74	783	.074	.066	141	24.6	3272	4.87	2.89	7.76
75 or more	470	.002	.002	168	4.17	700	5.12	1.43	6.55
Total 25-64	3632	.64	.57	164	32.3	4636	5.04	3.10	8.14

Table 2

Indicators of Poor Health, Classified Disabilities, Chronic Health Conditions, by Age and Sex

Sample, Sex & Age	Health status (1-5, 1= very good)	Health problem (0-1 in 30 days)	Days missed due to illness	Disability classi-fication	Chronic Disease Condition (M20.6)							Depression in last 12 months	High blood pressure	Diagnosed with diabetes
					Heart	Lung	Liver	Kidney	Stomach	Spinal	Other			
Males, Ages:														
15-24	1.36	.202	.465	.044	.040	.034	.040	.051	.083	.071	.131	.131	.100	.006
25-34	1.48	.224	.531	.048	.035	.037	.040	.030	.090	.070	.093	.116	.184	.006
35-44	1.64	.272	.554	.095	.053	.043	.047	.037	.122	.121	.108	.146	.269	.012
45-54	1.82	.294	.408	.222	.099	.053	.046	.046	.184	.176	.172	.147	.354	.016
55-64	2.09	.441	.404	.373	.205	.092	.071	.071	.154	.199	.242	.145	.424	.044
65-74	2.26	.550	.271	.413	.319	.141	.090	.118	.203	.214	.290	.160	.545	.064
75 or more	2.55	.750	.157	1.059	.421	.158	.129	.080	.259	.207	.496	.175	.584	.058
Total 25-64	1.70	.286	.488	.152	.081	.051	.048	.042	.132	.129	.139	.136	.285	.016
Females, Ages:														
15-24	1.50	.295	.427	.039	.044	.023	.029	.081	.141	.062	.109	.219	.103	.006
25-34	1.61	.313	.540	.043	.034	.029	.057	.094	.120	.065	.157	.200	.191	.008
35-44	1.83	.352	.566	.068	.082	.042	.081	.100	.158	.143	.219	.243	.330	.017
45-54	2.01	.504	.555	.153	.156	.066	.134	.119	.192	.233	.280	.254	.532	.038
55-64	2.17	.612	.400	.268	.311	.078	.190	.186	.287	.270	.342	.269	.691	.110
65-74	2.49	.753	.126	.515	.481	.093	.235	.184	.305	.299	.381	.289	.752	.139
75 or more	2.79	.816	.021	.564	.519	.068	.241	.137	.267	.274	.473	.240	.767	.145
Total 25-64	1.87	.429	.526	.120	.129	.051	.108	.119	.179	.169	.240	.238	.409	.037

Table 3
Health-related Characteristics and Behaviors, by age and sex

Sample, Sex & Age	Years of Schooling	Cigarettes per day	Alcohol grams per day	Medical checkup 3 months	Exercise Frequency	BMI greater than 27 (0-1)	Waist to hip ratio	Height in cm	Cohabiting and married
Males, Ages:									
15-24	11.8	10.4	86.5	.168	.584	.095	0.850	176.7	.337
25-34	12.4	11.9	108.0	.160	.496	.243	0.881	176.4	.764
35-44	12.3	12.5	115.0	.125	.361	.308	0.898	175.0	.854
45-54	12.2	11.9	104.0	.136	.333	.398	0.914	173.2	.874
55-64	11.8	11.0	84.9	.118	.252	.385	0.927	170.8	.885
65-74	9.46	6.61	63.2	.123	.487	.436	0.938	168.4	.831
75 or more	9.52	2.64	35.90	.171	.360	.399	0.938	167.6	.629
Total 25-64	12.2	11.90	105.0	.140	.382	.320	0.900	174.4	.834
Females, Ages									
15-24	12.8	3.01	37.90	.251	.434	.109	0.774	164.0	.523
25-34	13.1	3.15	41.90	.229	.371	.234	0.784	164.0	.763
35-44	.12.7	2.88	41.90	.207	.342	.403	0.803	162.7	.729
45-54	12.5	1.79	30.90	.211	.314	.615	0.827	160.7	.689
55-64	12.1	1.06	23.00	.152	.446	.704	0.844	159.7	.560
65-74	9.44	0.238	11.90	.105	.345	.675	0.867	157.1	.377
75 or more	7.34	0.106	5.12	.070	.284	.585	0.881	154.6	.143
Total 25-64	12.7	2.33	35.50	.205	.357	.465	0.811	162.0	.701

Table 4

Two Stage Least Squares and Ordinary Least Squares Estimates of Productivity Effects of Health States and Health Inputs

Explanatory Variable Coefficient by Estimation Method	Participation		Reports Hours and Earnings		Log Hourly Wage		Log Hours per Month	
	Male	Female	Male	Female	Male	Female	Male	Female
1. Health Status								
OLS	-0.0849	-0.0459	-0.0800	-0.0541	-0.135	-0.0520	0.0170	-0.0028
	(6.96)	(3.66)	(5.84)	(4.09)	(4.08)	(1.73)	(1.07)	(0.16)
2SLS	-0.159	-0.0697	-0.142	-0.0926	-0.0243	-0.0330	-0.0703	-0.0333
	(5.96)	(2.27)	(4.75)	(2.86)	(0.29)	(0.43)	(1.75)	(0.75)
2. Health Problem								
OLS	-0.0825	-0.0444	-0.0740	-0.0416	-0.0423	0.0214	-0.0357	-0.0201
	(4.90)	(2.98)	(3.92)	(2.65)	(1.00)	(0.66)	(1.77)	(1.08)
2SLS	-0.248	-0.0943	-0.209	-0.109	-0.0827	-0.0049	-0.100	-0.0270
	(5.66)	(2.44)	(4.28)	(2.69)	(0.73)	(0.05)	(1.84)	(0.53)
3. Disability Class								
OLS	-0.127	-0.122	-0.118	-0.111	-0.133	-0.0293	-0.0114	-0.0530
	(9.88)	(9.07)	(8.16)	(7.75)	(2.83)	(0.68)	(0.51)	(2.14)
2SLS	-0.303	-0.159	-0.247	-0.129	-0.121	-0.0587	-0.269	-0.0216
	(7.35)	(3.13)	(5.40)	(2.39)	(0.58)	(0.22)	(2.59)	(0.14)
4. Jointly Estimated Four Inputs								
Medical Visits								
OLS	0.089	0.167	0.081	0.163	-0.0258	-0.143	0.0031	0.0167
	(3.99)	(9.45)	(3.23)	(8.50)	(0.52)	(3.99)	(0.13)	(0.81)
2SLS	-1.10	-0.306	-0.821	-0.456	1.82	-0.648	0.462	-0.125
	(2.50)	(0.74)	(2.20)	(0.98)	(1.55)	(1.44)	(0.87)	(0.51)
Exercise								
OLS	0.0003	-0.0092	0.0025	-0.0115	0.0223	0.0189	-0.0014	0.015
	(0.04)	(1.23)	(0.28)	(1.47)	(1.19)	(1.08)	(0.16)	(1.47)
2SLS	0.352	-0.0928	0.241	-0.035	0.393	-0.0582	-0.257	-0.083
	(1.51)	(0.66)	(1.28)	(0.22)	(0.76)	(0.20)	(1.11)	(0.52)
Alcohol								
OLS	0.001	0.00831	0.0011	0.016	0.0019	0.0069	0.0021	-0.0007
	(0.89)	(3.44)	(1.80)	(4.56)	(0.49)	(1.11)	(1.17)	(0.19)
2SLS	0.0025	0.0134	0.02	0.0201	0.0901	0.0025	0.0153	0.0297
	(1.06)	(1.79)	(1.02)	(2.37)	(1.45)	(0.03)	(0.55)	(0.65)
Alcohol Squared								
OLS	-0.0000	-0.00025	-0.0002	-0.0004	-0.0000	0.0001	-0.0006	0.0000
	(2.02)	(2.92)	(3.74)	(4.02)	(0.06)	(0.52)	(1.31)	(0.22)
2SLS	-0.00098	-0.00005	-0.0006	-0.008	-0.003	0.0011	-0.0091	-0.0001
	(2.11)	(1.75)	(1.63)	(2.30)	(1.44)	(1.32)	(1.16)	(0.55)

Figure 5: 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.

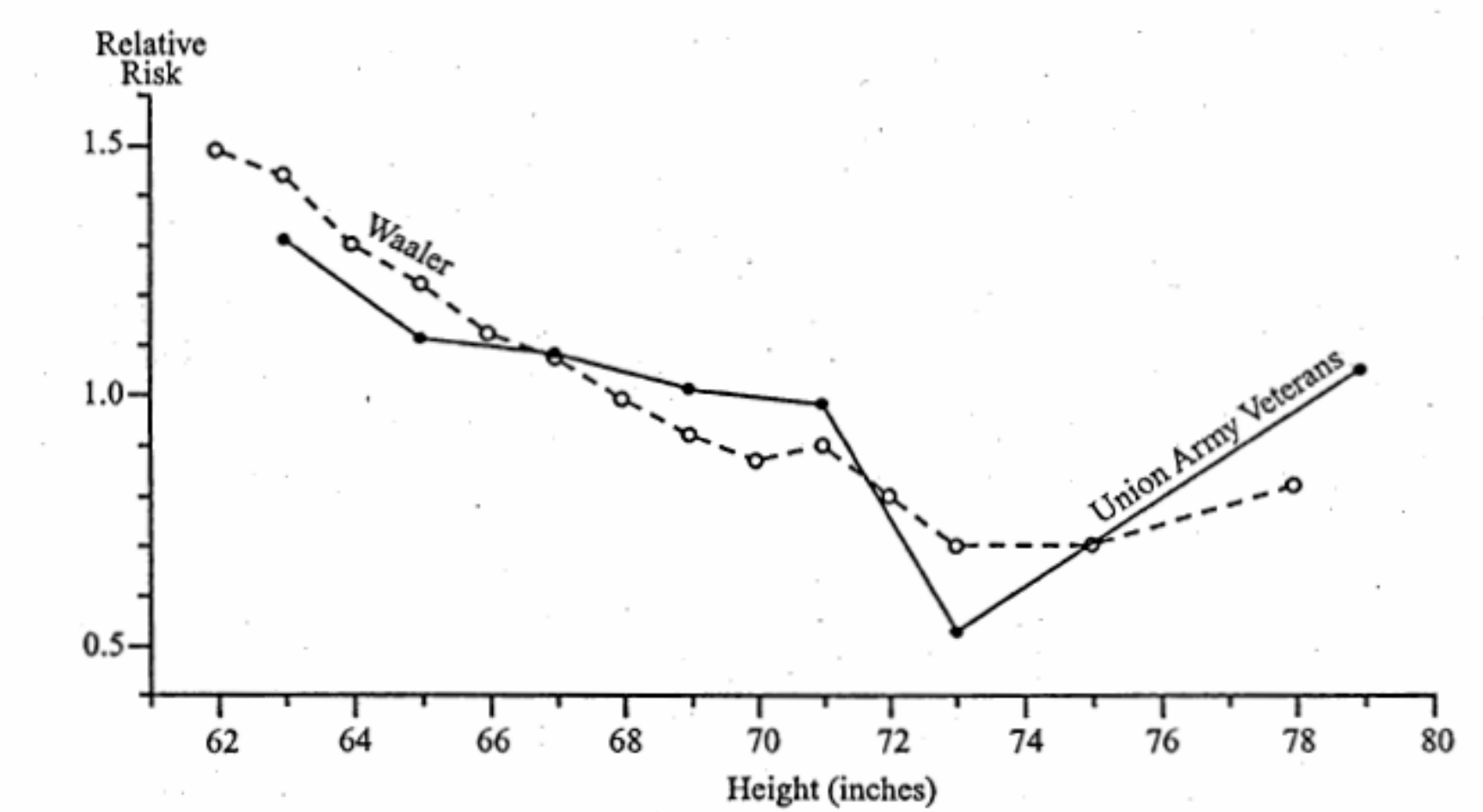


Figure 6: Height in Centimeters of Adult Females in Ghana in 1987-1989

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

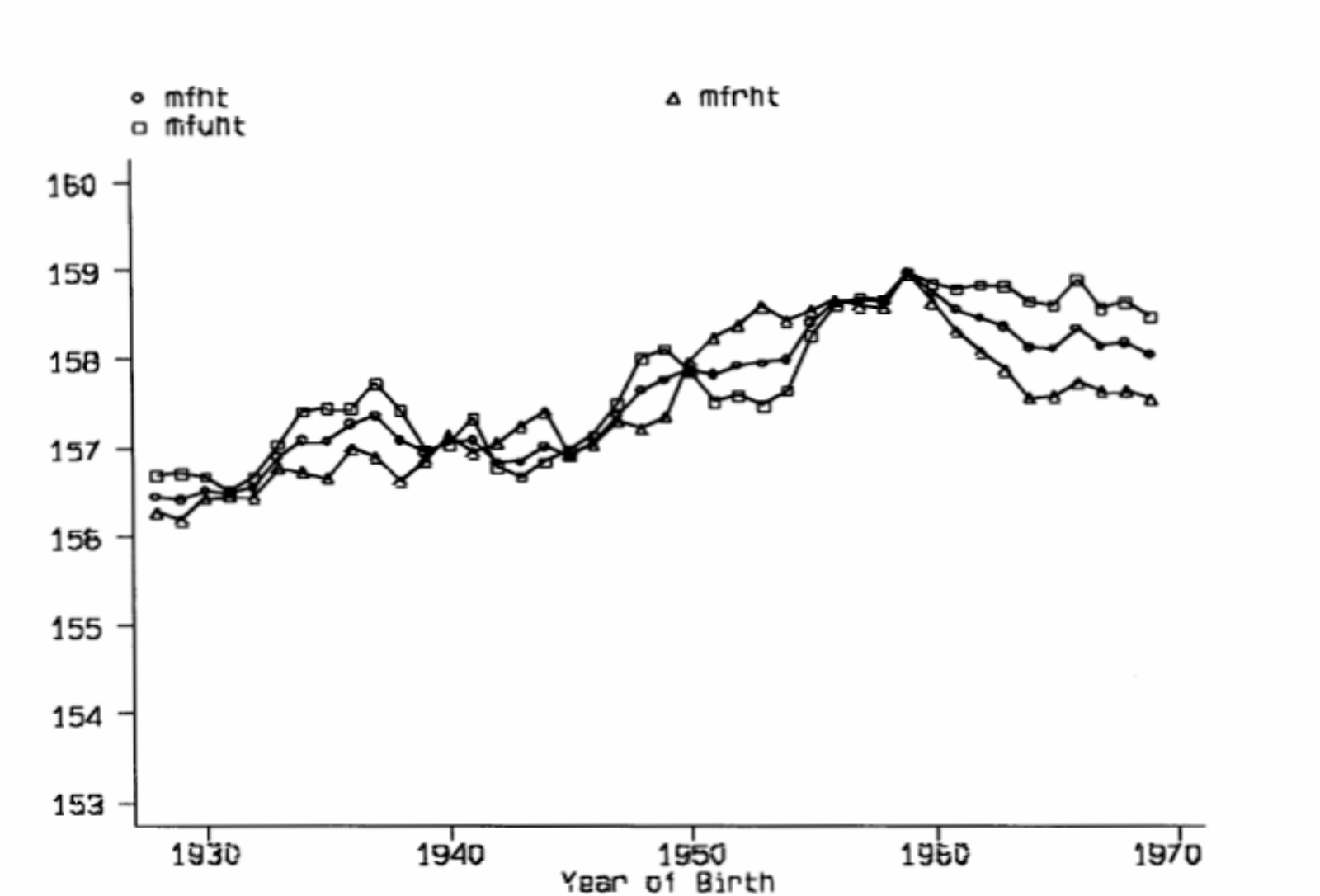


Figure 7: Height in Centimeters of Adult Females in Brazil in 1989

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

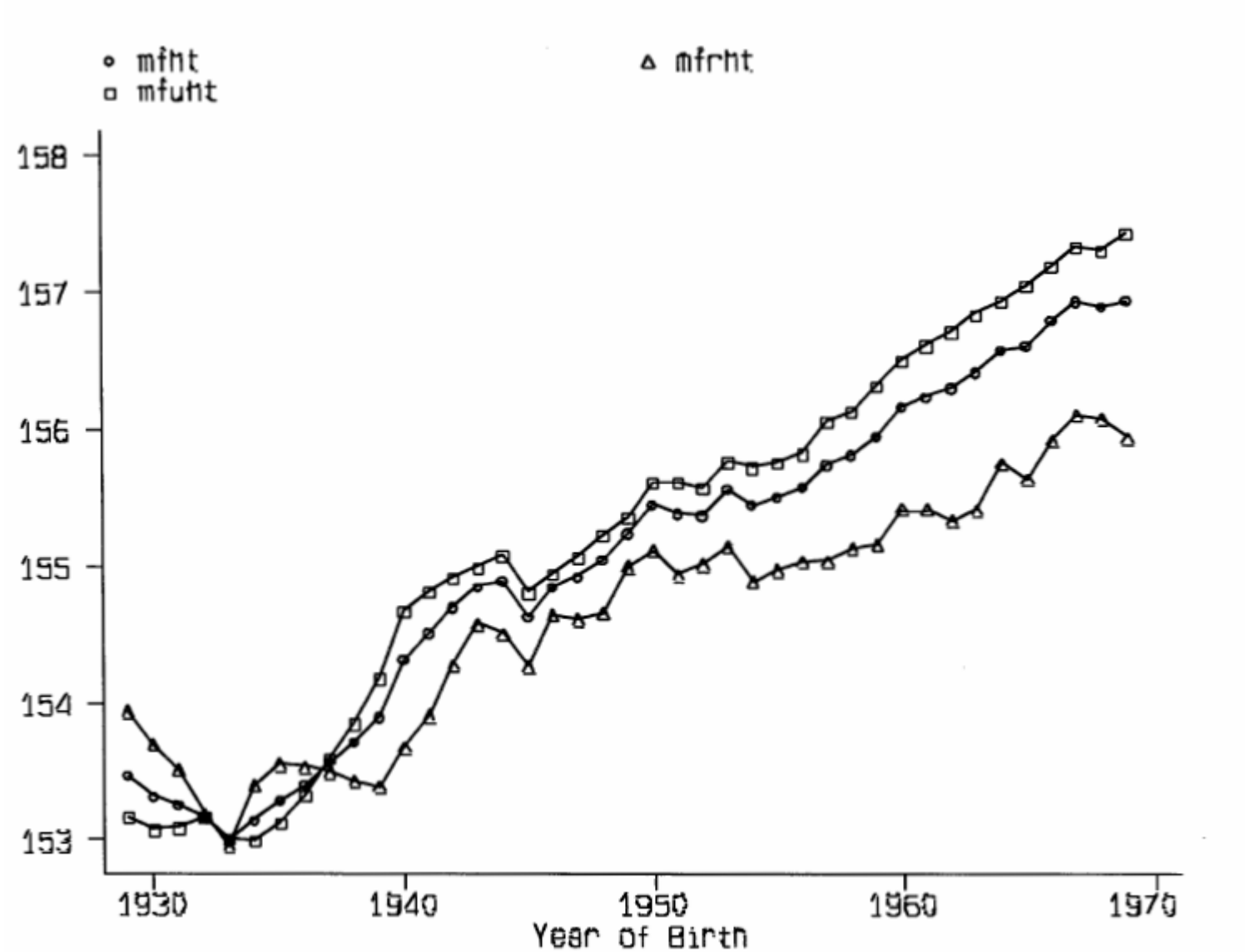


Figure 8: Comparison of Relative Mortality Risk by BMI among Men 50 Years of Age, Union Army Veterans around 1900 and Modern Norwegians.
Source: Costa and Steckel (1997)
Note: In the Norwegian data, BMI for 79,084 men was measured at ages 45-49, and the period of risk was 7 years. BMI of 550 Union Army veterans was measured at ages 45-64, and observation period was 25 years.

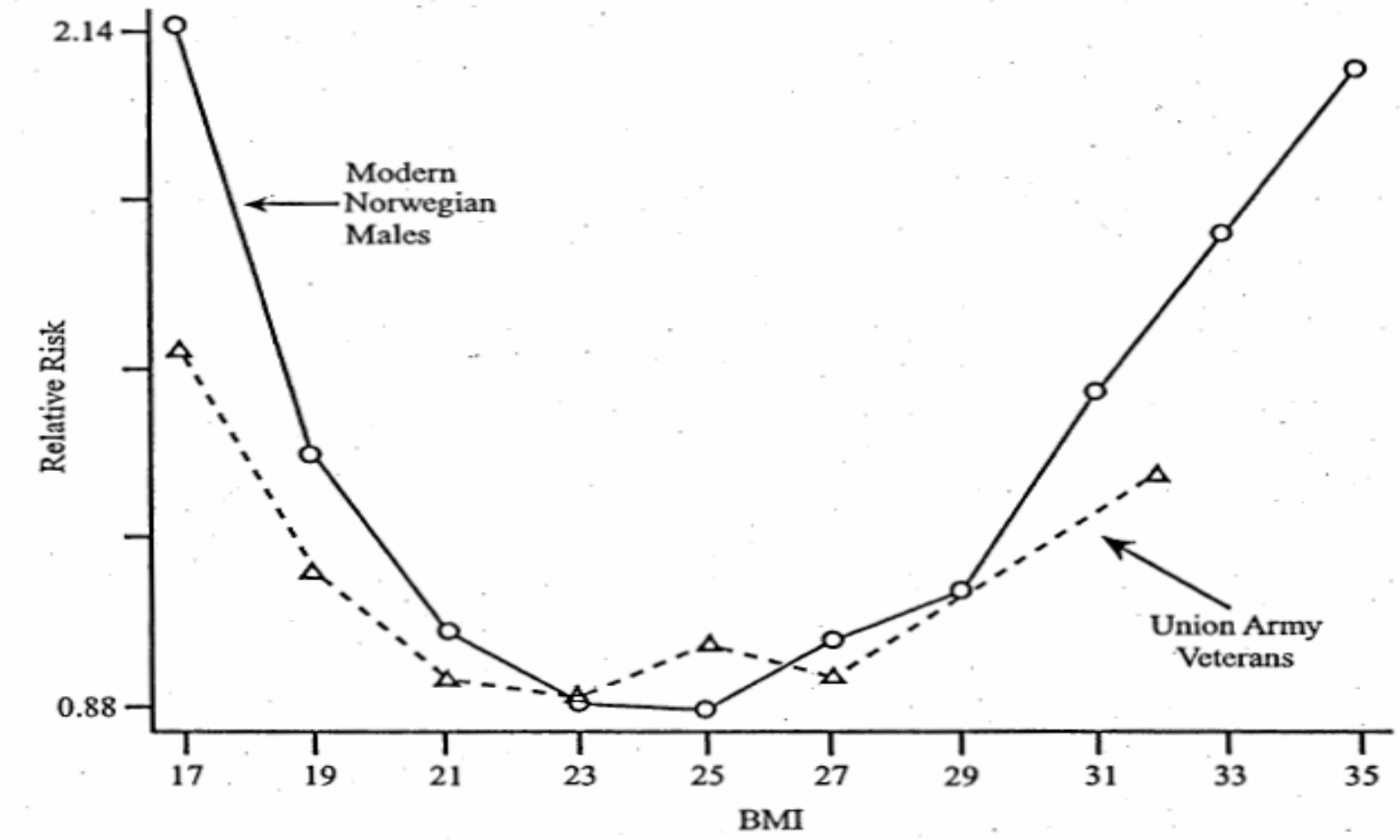
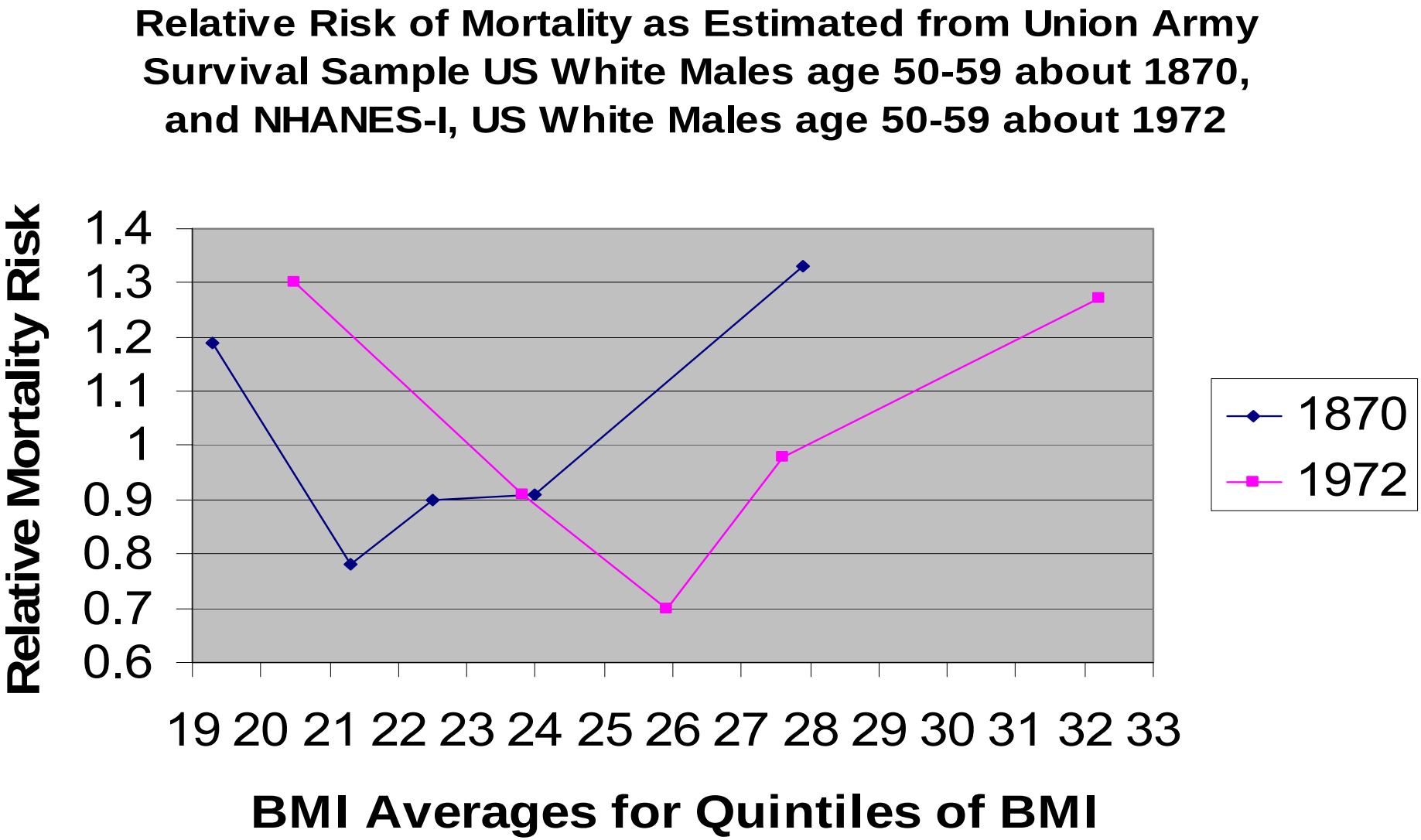


Figure 9



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

Table 5

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 & Household Characteristics	Regional Characteristics & Parent Education	Ethnic or Race Groups		Height in cm
Ghana, 1987-89						
Living Standard Measurement Survey	(a)	(b)	(c)	(d)		
Age 25-54						
Males	.0154	.0943	.0934	-.1017	2876	169.
	(4.78)	(3.71)	(4.27)	(3.66)		(6.49)
Females	.0167	.1091	.1522	-.0765	2754	158.
	(4.04)	(4.14)	(6.56)	(2.37)		(6.16)
Brazil, 1989						
Health and Nutrition Survey	(e)	(f)		(g)		
Age 25-54						
Males	.0140	.0775	na	.0701	7808	168.
	(10.3)	(9.34)		(12.4)		(7.20)
Females	.0166	.1086	na	.0862	4047	156.
	(8.13)	(8.12)		(8.11)		(6.63)
USA, 1989-93						
National Labor Force Survey of Youth	(h)	(i)	(j)	(k)		
Age 24-36						
Males	.0056	.0210	.0353	.0183	18,491	178.
	(5.12)	(1.75)	(4.92)	(3.08)		(7.65)
Females	.0043	.0453	.0473	-.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 ~~in Ghana at the primary level (max 6) and secondary or higher~~ by levels, post-schooling years of experience (age-schooling-6), experience squared, and rural residence.

Table 6: Coefficients on Four Indicators of Human Capital Inputs in log wage equation in Ghana, 1987-1989.

Country Gender Variable	(1) OLS	(2) OLS	(3) OLS	(4) OLS	(5) IV	(6) IV
Ghana	Males: Sample Size 3414					
Education	.0521 (11.7)	.0475 (10.7)	.0449 (10.1)	.0437 (9.86)	.0445 (2.46)	.0445 (9.95)
Migration		.388 (7.48)	.360 (6.97)	.348 (6.75)	.218 (2.26)	.295 (5.34)
BMI			.0542 (6.93)	.0530 (6.80)	.0793 (1.95)	.0658 (1.76)
Height				1.48 (5.02)	5.69 (3.45)	5.56 (3.58)
	Females: Sample Size 3400					
Education	.0481 (9.23)	.0425 (8.22)	.0395 (7.69)	.0375 (7.26)	.0356 (2.69)	.0346 (6.56)
Migration		.617 (9.85)	.537 (8.55)	.531 (8.46)	.361 (2.98)	.447 (6.51)
BMI			.0425 (7.72)	.0420 (7.63)	.0981 (4.11)	.0881 (4.32)
Height				1.29 (3.63)	7.48 (3.44)	7.62 (3.80)

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

[a] Other 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 Cols. (1)-(4) and asymptotic t ratio in Cols. (5)-(6).

Table 7

Means and Standard Deviations in Parentheses of Height and Schooling by Country, Selected Age Groups, and Sex

Country Age	Height (cm.)		Schooling (yrs.)	
	Female	Male	Female	Male
Ghana: 1987-89				
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)
Cote d' Ivoire: 1985-87				
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)
Brazil: 1989				
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)	+3.10 (-0.20)	+4.15 (+1.45)	+3.14 (+1.04)
Vietnam: 1992-93				
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)

Figure 10: Model Linking infectious exposure at earlier ages and external environment to inflammation, height, organ damage, morbidity, and mortality at older ages.

Source: Crimmins and Finch (2006)