# The causal effect of health on social and economic prosperity: Methods and findings

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## Abstract

Socio-economic status (SES) and health go hand in hand: better off people tend to be in better health across the globe, within populations and over the life course. A critical review of both experimental and non-experimental evidence indicates that, in some contexts, health has a causal impact on SES. Fetal health and health in early life appear to affect health and economic prosperity in later life. Changes in health during adulthood also affect work outcomes and wealth. Overall, the strongest evidence for a causal impact of health on economic success is reflected in the impact of improved nutrition on economic productivity. There is little scientific evidence on the dynamic interplay between health and behavioral choices that affect economic outcomes over the longer term. Unparalleled opportunities exist for making major contributions to knowledge that have the potential to have a lasting impact on the well-being of populations across the globe.

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## Introduction

Poverty and poor health tend to go hand in hand. Health and socio-economic status (SES) have been shown to be positively associated in human populations on every continent across the globe, in modern times and also stretching back at least several centuries. In most contexts, the relationship emerges very early in life and persists over the entire life course. Further, the association has been established for a broad array of indicators of both health and SES. Interpretation of these empirical relationships takes center stage in large, contentious and important debates about health, the economy and society.

Arguably, isolating the causal pathways that link health and economic prosperity lies at the heart of these debates. Pinning down causal pathways at the individual level is key for science and policy. Research on the impact of health behaviors, health investments and unanticipated health events on the economic and social well-being of individuals, their families and their communities has provided valuable insights into a broad array of important questions in the social sciences. These include, *inter alia*, understanding routes into and out of poverty, coping strategies in the face of adversity, dynamics within families and within communities, inter-temporal trade-offs and forward looking, savings, wealth and credit constraints. From the perspective of policy design, it is important to measure the extent to which growth in income causes health improvements so that those benefits are taken into account when resources are allocated to programs that are intended to promote economic growth. Moreover, if health contributes to economic prosperity, these benefits ought to be included in evaluations of the impact of resources allocated to the health sector. These might include the provision of public health services, immunization, maternal and child health programs, subsidized medical care and health insurance programs as well as public health information campaigns and training health personnel.

There are at least two reasons why interpretation of the association between health and SES is complex. First, causality almost surely runs in both directions. In low resource populations, as income increases, individuals and families typically invest in better diets, improved sanitation and allocate more resources to health care. In this case, income or SES is driving health. On the other hand, if a healthier worker is less susceptible to disease, more alert and more energetic, then he or she will probably be more productive and possibly work longer hours. The worker will thus command higher earnings. In contrast, individuals in poorer health may reduce their hours or be unable to work at all and they may have to dip into their savings to pay for costly treatments or care. In these cases, it is health that is driving SES.

A second, related reason that interpreting correlations between health and SES is difficult arises because other, unobserved factors likely drive both health and economic prosperity. For example, part of health is inherited as are other attributes related to economic prosperity. The role of genes – and the role the environment plays in their expression – is not fully understood. While the technology for measurement of these factors is changing very rapidly, they are typically not observed in empirical studies and may drive changes in both health and SES. Further, families that choose to invest in the health of their children may also value other investments that would improve their own and their children's lives. These may include investments in other dimensions of their children's human capital. To the extent that indicators of these investments and the behaviors, tastes and attitudes that underlie them are not included in the empirical models, they will be reflected in unobserved heterogeneity that drives both health and SES in the models.

Taking into account factors that are not readily observed (or, more precisely, not treated as observed in most models) is important for at least two reasons. First, demonstrating that health does not have a causal impact on income cannot be interpreted as indicating that the correlation

between income and health should be attributed to a causal effect of income on health. The association may be attributed to these third, unobserved factors.

Second, the literature has typically referred to unobserved factors as including, for example, tastes, information and the like. Some of these are amenable to measurement – such as information about health practices, adherence to drug regimens or propensities to save for the future. Providing scientific evidence on the role they play, if any, in behavioral choices that explain the association between health and economic prosperity is a potentially important line of research. Moreover, some of these factors can be influenced by policy. For example, campaigns that provide information about health and the health consequences of behaviors have been successful in effecting lifestyle changes that promote better health. Tobacco use and exercise provide excellent case studies.

This paper focuses on key issues involved in isolating causal pathways that link health to SES at the micro-level. Rather than attempt to provide an exhaustive description of the methods and evidence, we focus on a small number of studies in the literature to illustrate the motivation for the approach taken, the ideas underlying the methods and assess their contributions to science. We highlight some of the lines of future inquiry in this literature that are likely to be especially profitable and describe some methodological advances that have the potential to yield new evidence that will substantially influence both science and policy design. For a fuller description of the methods, see Burns, Keswell and Thornton (2009) and the discussion of methods in Deaton (2009) and Banerjee and Duflo (2009). For a broader perspective on the substantive issues in the literature on health and SES, see reviews by Strauss and Thomas (1996, 2008).

**Measurement of Health and SES**

While relationships between a broad array of markers of health and different indicators of SES have been established, the magnitude and direction of the correlation varies with both measures of health and of SES. There are good reasons to expect this variation and it is important to take the nature of both the health and SES markers into account when interpreting the empirical evidence.

For example, short-term episodes of illness that have no longer-term ramifications for health or functioning may result in changes in how time is allocated (to rest and recuperation, say) and, thereby, possibly to earnings during the period of illness although even this will depend on the nature of the work and the payment mechanisms. Those paid by the piece or on an hourly or daily basis are likely to have lower earnings whereas workers with paid sick leave may not. Re-allocations of time in response to episodes of illness may be temporary and there may be substitution when the individual's health improves to make up for lost time in some activities (such as work).

More persistent or chronic health conditions may have broader effects on SES. These conditions may result in permanent changes in time allocation, productivity and therefore earnings as well as consumption, savings and wealth as individuals draw on resources for treatment or because of reduced earnings. Conditions that affect life expectancy likely impact inter-temporal preferences and therefore decisions about savings and wealth accumulation.

In practice, in many instances, whether a specific health event will turn into a chronic condition may not be immediately obvious and the path from short-term to longer-term health difficulties may be complex and circumspect. Moreover, the cumulative effect of multiple health insults may turn an apparently short-term health problem into a chronic condition. For example, the literature suggests the deleterious consequences on day-to-day functioning of a traumatic

event such as an earthquake or exposure to a violent attack, are dramatically magnified when an individual has had prior exposure to traumatic events.

The impact of a health condition may also have long-reaching effects on economic and social success if the condition results in a shift in the individual's lifetime trajectory. For example, health insults during the fetal period or a health condition that emerges during early childhood may affect cognitive development or school performance which, in turn, has lasting effects on academic achievement and economic success in later life. There is an extensive literature on the deleterious consequences of macro- and micro-nutrient deprivation *in utero* and in early childhood. Studies indicate that deprivation during this critical period, when many of the biological systems in the body are being developed, affects both physical health and cognitive functioning.

In some instances, behavioral responses to chronic conditions may mitigate negative consequences on economic prosperity. These might include, for example, changes in occupation or changes in life-style choices. Families may also play a role in mitigating the impact of poor health of one of their members. The extent to which these sorts of responses are feasible may depend on SES – better educated people may have more options for changes in occupation and life-style than people who rely on physically demanding labor for income.

Health innovations in later life are likely to have yet different impacts on the economic security of an individual. The impact will vary with the extent to which there is a public safety net or the individual has a private safety net either through accumulated wealth or relying on family and others for support.

It is important to keep in mind that there is likely to be considerable heterogeneity in not only the magnitude and direction of the association between health and SES – but also its interpretation. These issues have received inadequate attention in the literature. In part, this

reflects the fact that relatively few health measures have been used in studies relating health to SES which, in turn, reflects the constraints of data.

Much of the earliest work in this literature examined the links between self-assessed health and SES. As an example, many surveys ask each respondent to provide an assessment of his or her own health status on a scale that ranges from, say, excellent to poor. The responses are interpreted as global indicators of general health status (GHS) which has been shown to be predictive of mortality and health problems in later life. However, GHS is also a reflection of factors other than health. These include the reference group against which the respondent compares his or her health as well as the related issue of each respondent's perception of "excellent" health. Moreover, it has been shown that GHS is worse among people who have better access to health services: in studies that randomly assigned participants to different prices of health care, those who faced lower prices used more care and reported themselves to be in worse health than the group that faced higher prices. This fact might contribute to the observation that, in low resource contexts, higher income people tend to have higher expectations for their health. In many low income countries, the association between GHS and income is negative: richer people report themselves as being in worse health than poorer people. The measurement of health status is a rapidly evolving line of scientific investigation which is resulting in nothing short of a revolution in the population health field.

More generally, in recent years there have been dramatic improvements in the quality and quantity of data to explore the relationships between health and SES. On the horizon, there are exciting innovations in the technology for measuring a broad array of health markers which involve minimally-invasive and non-invasive methods that can be implemented in the field at low cost. Anthropometric measures such as height, weight, waist, arm and head circumference, have been included in studies for many years. Indeed historical data on height and weight have been creatively used to draw inferences about standards of living in the distant past.

Complementary measures are becoming increasingly feasible with studies measuring fat, bone density, heart rate and blood pressure in the home. Rapidly evolving technologies using, for example, point-of-care monitors have enabled population-based surveys to measure a broad array of health makers in the home. Many studies have used finger sticks that take a few drops of blood and measured levels of micronutrients (such as hemoglobin), one of more markers in a lipid profile (such as total cholesterol), indicators of inflammation (such as C-Reactive Protein), HIV status and indicators of diabetes risk (such as glycosolated hemoglobin, HbA1c). Other studies have assayed blood drops dried on filter paper, taken venous blood, cheek swabs, saliva, hair to identify the presence of short-term and longer-term health conditions as well as measure DNA.

Paralleling improved measurement of health, there have been dramatic improvements in the quality and quantity of linked socio-economic and health data. This includes long-term longitudinal surveys that follow individuals over the life course as well as rich administrative data that can be linked with health and socio-economic surveys.

In combination, these data sources provide opportunities to make major advances in understanding the complex interplay between different dimensions of health and SES over the life course. In the following sub-sections, variation over the life course will be used as an organizing principle in an assessment of the state of the field regarding the causal impact of health on socio-economic prosperity. It will be helpful to precede that discussion with a brief review of some of the main conceptual issues that arise in empirical research in this field.

**Conceptual and empirical framework**

To highlight the challenges confronting scientists in this area of inquiry, consider a simple model of behavior in which an individual chooses to allocate resources to goods and services over his or her life course given a budget constraint, limited time and the technology of

the production of human capital, part of which is health. Investments in health may have an immediate pay-off or the investments may reap returns in the future. Time may be allocated to work, investment in human capital or to leisure activities. In this model, productivity in any period, $y$, will depend on health, $\theta$, other dimensions of human capital (such as education, training and experience) along with other observed characteristics of the individual, $x$, such as age and gender, and unobserved characteristics, $\varepsilon$. The latter may include abilities, attitudes and tastes, aspirations and expectations as well as indicators of health that are not measured in $\theta$ (and $x$) but affect y through a function f(.):

$$y = f(\theta, x, \varepsilon) \qquad [1]$$

If $\theta$ were randomly assigned to an individual, in principle, it would be straightforward to interpret $\partial y/\partial \theta$ as an estimate of the causal impact of changing this dimension of health on the outcome of interest, y. In this case, by assumption, $\theta$ will not be correlated with the observed characteristics, $x$. Therefore, whether those characteristics are included or excluded in the model will have no impact on the estimated magnitude of the causal effect, $\partial y/\partial \theta$. However, to the extent that $x$ and $\varepsilon$ are correlated, the inclusion of $x$ in the model will affect the precision with which $\partial y/\partial \theta$ is estimated.

While the interpretation of $\partial y/\partial \theta$ is conceptually straightforward under the assumption that $\theta$ has been randomly assigned, it is not obvious that it is interesting. For example, improving the health of an individual in ways that increase the person's expected life may not result in a change in economic productivity today but may result in an extension of the time the person can expect to be productive. This may result in a change in the level of investment in health and human capital, asset accumulation and portfolio allocation. The impact of the improvement in health may be revealed only in the longer term. Thus a focus on shorter-term indicators and indicators of labor market outcomes may be misleading. Similarly, an

improvement in health that results in greater productivity in the work place may not be rewarded by employers in terms of wages and so may only be reflected in promotions after the productivity increase has been sustained. These are important concerns and underscore the importance of developing a theoretical underpinning for any empirical implementation of the general framework [1].

That said, the key advantage of randomization is that, by design, $\theta$ is not correlated with $\varepsilon$. There are, however, many ways in which that condition may be violated. First, while conceptually straightforward, inducing random variation in health of individuals in a population is not straightforward. A standard approach to randomization is to split a study population into a group who receive a treatment such as something that improves health and a control group who receive something that is observationally equivalent but has no impact on health. It is standard practice for no one in the study to know who has been assigned to the treatment or control group. Comparing the outcome, *y*, before and after the intervention, $\Delta y^T$ , provides a measure of the impact of the treatment on those who received the treatment. Changes in *y* among the controls, $\Delta y^C$ , measures changes that would have occurred without the treatment and so the difference in the changes, $\Delta y^T - \Delta y^C$ , is the causal impact of the treatment on *y*.

If assignment to the treatment or control group is known to the subjects, it is not only possible but very likely that the assumption that variation in health, $\theta$, is not correlated with unobserved characteristics, $\varepsilon$, is violated. In that case, the primary benefits of randomization are dissipated and, in general, the estimate of $\partial y/\partial \theta$ will be biased. There are many plausible examples of why this may happen. A subject who knows that he has been assigned to the treatment may try harder to improve the outcome, y, in order to help the study. Or the subject may compensate for being in the treatment by adjusting investments in health. If the treatment improves health, a subject assigned to the control group may seek out the treatment – or a similar

treatment from other sources. If a subject is assigned to the control group, the subject may be loath to continue to participate in the study and attrition will be greater among this group. This will not be a problem if attrition is unrelated to both observed health, $\theta$, and unobserved characteristics, $\varepsilon$, that affect the outcome, $y$. It is very difficult to know if attrition is selective on $\varepsilon$.

The assignment to treatment and control group may be known because the study informs the subjects or cannot keep the assignment from the subjects (such as changes in prices of health services or insurance coverage), because changes in health are observed and subjects learn about their status and that of others in the community (such as a nutrition intervention) or because the treatment involves adopting a behavior (such as a change in exercise or diet). If the intervention calls for a change in behavior by the respondent, different behavioral responses that are correlated with $\varepsilon$ will also contaminate the interpretation of the estimate of $\partial y/\partial\theta$. Studies have sought to address the observability of assignment to treatment or control group by stratifying the study population into communities and randomizing at the community level. This has the advantage that everyone in a community receives either the treatment or the placebo and so inter-personal comparisons within the community provide no information on whether the subject is assigned to treatment or control. However, unless it is implemented in a large number of communities, the study is likely to have low statistical power.

While randomization is an important and potentially extremely useful tool for measuring causal effects, it is but one method. Many studies of the impact of health on socio-economic outcomes rely on observational data. In general, the assumption that $\theta$ and $\varepsilon$ in the model [1] are uncorrelated is not plausible and it is important to adopt an empirical strategy that takes this into account.

One approach has been to exploit a policy reform, the implementation of a health program or some other change in the health environment that can be treated as outside the

control of the study subjects. These quasi-experimental designs seek to replicate the experimental design as closely as possible by identifying two groups of people who are in all respects identical except that one group was exposed to the change and the other group was not. There have been some very clever studies which have exploited variation of this sort to isolate the causal effect of health programs on socio-economic outcomes. Clearly, it is critical that the "treatment" and "control" groups are identical in all respects other than the specific change that is being investigated. That is not straightforward. In many cases, programs are targeted towards some sub-group -- say to areas or people who need them most. Similarly, if a program such as improved health services is successful, it is likely to attract users from outside the immediate catchment area. Selective program placement or selective response to program placement by users of the program substantially complicates comparisons of indicators of those exposed with those not exposed. More generally, this concern may arise in studies that rely on variation due to spatial, temporal or cohort differences in exposure to health innovations.

If reforms will be implemented, there are likely to be substantial returns to introducing them on a phased basis. For example, introducing a program on a pilot basis provides early information about the effectiveness of the program and might offer policy-makers opportunities to evaluate different models rather than implement a single program with little information about its effectiveness. Say a pilot is to be conducted in fifty villages. If a hundred villages that are effectively identical are identified and the program is randomly assigned to fifty of the villages, it is in principle straightforward to measure the impact of the program by comparing changes in the villages that received the pilot treatment with the other villages that did not.

An alternative approach to estimation of [1] with observational data attempts to model the process that links $\theta$ and $\varepsilon$. For example, if the unobserved characteristics, $\varepsilon$, that are thought to be correlated with the indicator of health, $\theta$, are assumed to be time invariant – such as an individual's propensity to invest in human capital and other longer-term investments – then it

may be possible to sweep out this unobserved fixed effect with a parameter that is estimated in the model and retrieve the causal effect of health on the outcome of interest, *y*. An alternative approach is to use instrumental variables to separate that part of $\theta$ that is not correlated with $\varepsilon$, call it $\hat{\theta}$, and then estimate the effect of that component of health on the outcome of interest. The key in this case is that it is necessary to identify instruments that are not correlated with unobserved heterogeneity, $\varepsilon$, but are good predictors of health. In some cases, it may make sense to combine fixed effects and instrumental variables approaches in order to pin down the causal effect of health on socio-economic outcomes.

Some studies have used prices of health care services as instruments. In this literature, prices are typically broadly defined to include the financial costs of services, availability, waiting times and quality of services. The validity of these instruments depends on assumptions about why prices, access and quality vary. Prices paid by individuals who are choosing among alternative service providers are likely to be correlated with unobserved characteristics that affect both health and socio-economic outcomes; in those cases, prices will not be valid instruments. The array of prices in the market from which the individual chooses may be appropriate instruments although why there is variation in prices, access and quality over time or space may not be random. For example, public health services may be more heavily subsidized in areas that are more remote, where health status is lower and people are poorer. In that case the prices are likely to be correlated with unobserved characteristics in the model [1] and the estimated effects will be biased.

Taking into account the dynamic interplay between health and SES further complicates the model [1]. The importance of understanding the potential dynamic pathways was discussed in the context of the design and interpretation of randomized treatment-control studies. The same issues apply *a fortiori* to studies that are based on observational data. Whereas in studies that induce random variation in health status control the timing of those changes and the timing is

known to the scientist, observational studies are more complicated. If changes in health take time to manifest themselves or if those changes are difficult to measure, it is possible that observed improvements in SES will precede observed improvements in health even when it is, in fact, health that drives SES. The reverse may also be true and, of course, there may be unobserved factors that drive changes in both health and SES but the timing of the impact of those factors may be different for SES and health. Relying on the timing of changes in health and SES to infer causality is likely to be fraught with problems in the absence of a clear understanding of the mechanisms that underlie the relationships.

Empirical research based on randomized treatment-control designs or observational data rely on assumptions. It is important to evaluate the credibility of those assumptions and, whenever possible, provide empirical evidence that evaluate the assumptions or the implications of the assumptions. This might include providing evidence on whether treatment and control groups in experimental and quasi-experimental studies are identical in all observed dimensions, whether instruments are good predictors of the covariates that are thought to be endogenous and whether the instruments are valid in the sense of not being correlated with unobserved heterogeneity in the models.

**Early Life Health and Later Life Outcomes**

Investigation of the links between early life health – including fetal health – and later life outcomes is an active area of scientific inquiry. Research by Barker and his colleagues has highlighted the key role of maternal nutrition during pregnancy and argue that inadequate nutrients and oxygen during fetal development may result in adaptions in organ development to assure survival while a fetus and possibly immediately after birth. This fetal "programming" appears to result in elevated risk of health problems throughout the entire the life course (Barker 1994, Victora and others, 2008).

For example, low birthweight may result in reduced growth during infancy as the body is programmed while a fetus to survive on low levels of nutrients. This “thrifty phenotype” which develops as an adaption to adverse nutrition *in utero* results in permanent metabolic and endocrine changes. These changes would be beneficial if food availability continued to be scarce after birth. However, if food is plentiful, the same metabolic changes may be disadvantageous. They have been associated with elevated risks of obesity, dyslipidemia and glucose intolerance in later life (Hales and Barker, 1992). Thus, being born small and thin but becoming overweight during adolescence or adulthood are thought to put an individual at greatest risk of heart disease and type 2 diabetes in later life. Furthermore, the biological literature suggests that it is not only the overall nutritional status of the mother that will affect fetal health but it is also the timing of nutrition and health insults that affect the growth and development of the child. For example, inadequate nutrients during the period that arteries are formed is associated with elevated risk of heart disease because of hardened arteries in mid and later life. (Godfrey and Barker 2000.)

Much of the experimental evidence on the longer-term effects of maternal nutrition deprivation relies on animal models. There are, however, several very innovative studies that exploit quasi-experimental designs to measure the effects among humans. For example, Roseboom and others (2001) summarize a rich body of work that examines the impact of the Dutch famine on health and mortality of prime age adults. Towards the end of the second World War, several Dutch cities that were thought to be supporting the Resistance were subjected to severe food rationing for around 6 months. Following people who were *in utero* during that period and comparing them with others who were born at the same time in other cities in Holland and were not subjected to the same food rationing provides a treatment and control group whose health can be compared in later life. Under the assumption that the treatment and control groups were not different in any respects that relate to health in later life, the authors report that nutrition

deprivation early in the fetal period results in elevated risk of glucose intolerance and heart disease among adult males and females.

It is likely that maternal nutritional status is not the only factor that affects fetal outcomes. Finch and Crimmins (2004) highlight the importance of infection during the fetal period. They argue that maternal infection and stress result in inflammation which has detrimental consequences for the development of the fetus which affects the health and well-being of the offspring through the entire life course. For example, inflammation *in utero* is thought to be implicated in the development of plaque in the arteries and therefore heart disease and that these effects are independent of diet and life style choices.

Quantifying the magnitude of these effects is particularly salient for developing countries with the coexistence of infectious diseases and the onset of the nutrition transition. On the one hand, the epidemic of obesity is reaching beyond the developed countries and spreading throughout the globe (Popkin 1993). On the other hand, levels of infection and thus inflammation are much higher in poorer societies suggesting these populations may be carrying a particularly heavy burden of disease into the future.

Establishing a biological pathway through which early life health insults causes later life health conditions is an important first step. This does not mean that every human who is exposed to such insults is destined to poor health in later life. Indeed, many maternal and reproductive health programs seek to minimize the risk of being exposed to such insults or mitigate their impact. The impact of effective interventions is perhaps nowhere more impressive than in examples that have resulted in low birth-weight babies attaining survival rates that are no different from those in the broader population and having no cognitive deficits relative to that population.

A key implication of this research is that the long arm of fetal and early childhood health may extend not only to health in later life but may also extend to economic and social prosperity

over the life course. As an example, studies that use population-based data have documented that in the absence of interventions, low birth weight is predictive of lower school attainment and less success in the labor market. While low birth weight likely reflects poor intra-uterine growth, it is not clear that these associations can be given a causal interpretation. Low birth weight is the outcome of many factors including low levels of resources, inadequate access to health services and maternal behaviors, all of which may be related to other investments in infants and children. To establish that variation in fetal health causes variation in economic success, it is necessary to identify changes in fetal health that are arguably exogenous and not affected by behavioral choices of parents.

Some recent studies attempt to address this concern. One of the best examples is Almond (2006) who examines the longer-term effects of being conceived in late 1918, during the Spanish flu pandemic. The Spanish flu was caused by an unusually severe strain of the H1N1 virus and resulted in very high levels of morbidity and mortality. It is estimated that over half a million people died in the United States and that about one in four people suffered from the virus (Crosby 1989). The virus was first detected in March 1918 and although the pandemic persisted through to the middle of 1920, its impact on the health of the United States population was concentrated on those who were *in utero* in the last half of 1918. Whereas, influenza usually kills the youngest and oldest, the Spanish flu killed prime age adults and fell particularly hard on males. This has been attributed to undetected tuberculosis among these men, rendering them particularly vulnerable. (Noymer and Gerenne, 2000.)

Using data from the 1960 United States census, Almond compares the health, education and labor market outcomes of people who were born in 1919 with those born prior to the onset of the pandemic and at least nine months after the pandemic ended. As shown in panel A of Figure 1, he finds that, relative to other cohorts, males who were born in 1919 (and thus *in utero* during

the most virulent phase of the pandemic) complete significantly fewer years of schooling. They are also less likely to have graduated from high school and earn less at age 40.

Almond attributes these effects to the impact of maternal infection on the fetus. It is possible that they reflect other influences such as higher food and other prices at the time of the pandemic, lower incomes because of reduced labor supply at the time of the pandemic or stress associated with the pandemic. It is possible that the results reflect the impact of the pandemic on child-bearing decisions of parents. As the influenza emerged in early 1918, some parents may have chosen to delay births. It is also possible that it reflects other changes in the society that occurred simultaneously: World War I ended in November, 1918 and the process of demobilizing 4 million men from the United States army began in February, 1919. Children born to those men may be different from those who were born prior to their return from Europe.

To investigate this issue, panel B of Figure 1 presents an indicator of SES of parents for the same cohort of births in panel A. Data are drawn from the 1930 census and the SES index is based on an occupation-income score. It is significantly lower for children born in 1919 relative to those born in 1920 which suggests that either lower SES parents were more likely to have children in 1919 than higher SES parents or that children of higher SES parents in the 1919 birth cohort were more likely to have died by age 11. The latter seems unlikely. There is a strong inter-generational correlation in education and SES and so the fact that the 1919 birth cohort has less schooling in adulthood reflects, at least in part, differences in family background. It would appear premature to attribute the entire gap between the 1919 and other birth cohorts in economic prosperity as an adult to the impact of health insults during the fetal period due to the influenza pandemic. While Almond also exploits variation across states in mortality attributed to the pandemic, those estimates are also potentially contaminated by behavioral responses to the pandemic. See Thomas (2008) for a discussion.

Related work examines the impact of being born during the 1959-1961 Chinese famine on human capital and economic prosperity in mid-life. Using the 1991 wave of the China Health and Nutrition Survey (CHNS), Chen and Zhou (2007) compare outcomes of cohorts born during the 1950s and 1960s. The impact of the famine is identified by using regional variation in the death rate in 1960 that is attributed to the famine. They find that adults who were born in 1959 or 1960 and survived to 1991 would have been 3 cm taller if they had not been exposed to the famine. They also report reduced hours of work of individuals in this cohort. Family income per capita is lower but the magnitude and significance of these effects differ across the cohort depending on the specific measure of income. It is unclear how to interpret evidence on family earnings which reflects the contributions of family members from other birth cohorts.

As with the influenza pandemic, it is possible that the estimated effects of the Chinese famine reflect the impact of income and price variation due to the famine. As the authors note, interpretation of the estimates is complicated by at least two additional concerns. First, location of birth is not known in CHNS and so the authors assume that respondents were born in the same location that they were interviewed 40 years later which, they conjecture, is a reasonable assumption in China during this period. This highlights an important advantage of Almond's work which uses census data with information on location of birth.

Second, survivors of the famine may be selected on health and human capital. On one hand, it is likely that the most frail would have died in the harsh environment at the time of the famine (in which case estimated effects are probably downward biased). On the other hand, there is evidence that fertility responded to the famine and birth rates declined substantially soon after the onset of the famine. The direction of bias in this case is unclear.

One assessment of the empirical magnitude of these influences is provided by comparing birth cohorts born around the time of the 1974 floods in Bangladesh with cohorts born before and after the floods. Flooding is commonplace in the Ganges delta. However, in terms of severity,

there had been no parallel to the floods of 1974 for almost twenty years and it is estimated that over 20,000 people were killed by the floods. Table 1 presents data from the 1996 Matlab Health and Socioeconomic Survey (MHSS) which collected population-representative information on individuals living in Matlab, one district in Bangladesh. The fraction of the population born between 1970 and 1978 is reported in the first column for each year of birth. The floods resulted in a substantially reduced birth cohort in 1975 – which is consistent with the idea that fetal health was affected by the floods. The second column reports differences in the fractions of males in each birth cohort that are unable to read. The 1970 birth cohort is the excluded category. Cohorts born in 1975 are nearly 10 percentage points less likely to be illiterate than those born in the early 1970s or in the late 1970s. These are very large differences since around 16% of males born in the 1970s are unable to read. Results are very similar when we compare males who are both illiterate and innumerate in the third column. (There is no evidence of a secular trend during the 1970s in literacy rates. In contrast, comparisons of school attainment and height are complicated by such trends.)

The fact that the cohort of males exposed to the floods has higher levels of human capital suggests that these estimates are dominated by selection rather than the deleterious impact of the floods on fetal health. There are two additional pieces of evidence suggesting this is a legitimate interpretation of the evidence. First, the 1974 and 1976 birth cohorts are also more likely to be literate and numerate than cohorts born before or after them. The 1976 cohort was not directly exposed to the flood.

Second, it is possible to assess the extent to which families with children who were born between 1974 and 1976 and who survived to 1996 are selective on characteristics that are associated with investments in human capital by re-estimating the models with a household fixed effect. Results are in the last two columns of the table. The differences in human capital across the birth year cohorts are substantially reduced and there is no evidence that the 1974-1976

cohorts are different from the other cohorts. We conclude that, at least in the case of the Bangladesh floods, inferences about the causal effect of health insults during the fetal period need to take into account behavioral responses by parents to the floods and their aftermath.

Selective fertility and fetal or infant mortality that is selective on characteristics that are related to investments in health and human capital is a potential concern for the interpretation of results that are based on comparisons across cohorts. In the Chinese case, there is evidence that the number of live births declined dramatically soon after the onset of the famine. Evidence based on children born in the United States in 1919 is also potentially contaminated by selective fertility. The general point is that individuals, couples and families likely take into account changes in the environment –including changes in the disease environment—when making decisions about the future.

Related work by Field, Robles and Torero (2009) uses a public health intervention in Tanzania to measure the impact of reductions in the extent of *in utero* iodine deficiency on cognitive development of children. Fetal brain development is retarded in the presence of iodine deficiency and many countries across have the globe have sought to increase the availability of iodine in the diet through supplementation. In Tanzania, a campaign in the 1970s distributed iodized oil capsules across the country. Field and her collaborators exploit spatial variation in the timing of the distribution of the capsules in conjunction with information on birth date to identify those children who were *in utero* at the time the capsules were distributed in the area in which they are living. Comparing these children with older and younger children living in the same community (who were not exposed to the iodine oil capsules while *in utero*), they find that iodine supplementation results in the average child completing an additional 1/3 of a year of schooling. They conclude this reflects the impact of iodine on cognition rather than through improved general health. Combining widely available data on child health and education outcomes, in conjunction with detailed information on geography and the timing of public health

interventions, the study provides scientific evidence on the impact of micro-nutrient deficiency *in utero* on later life outcomes.

The emerging body of literature on the health and economic impact of insults *in utero* is extremely important. A related line of inquiry assesses the extent to which investments in the health and well-being of reproductive age women – and especially pregnant women – contribute to the health and economic well-being of the next generation. There are other potential benefits from investments in the reproductive health of women. Greater control over fertility typically leads to fewer children ever born and possibly greater investments in each child (Schultz 2007). Moreover, there may be benefits in terms of better health of reproductive-age women. Greater control over timing of births and improved health potentially improve labor market opportunities for women and the resources available to them. To the extent that this also elevates women's control over resources within a family, the literature suggests that there may be further investments in the health and well-being of children as well as in the accumulation of wealth. (Thomas 1990; Rubalcava, Teruel and Thomas, 2008.)

These issues are explored in a series of studies that use observational data and exploit variation in the availability of reproductive health services across Indonesia. The *bidan desa* or village midwife program seeks to place a trained nurse in every village in Indonesia in an effort to reduce maternal mortality and improve birth outcomes. The program was implemented in the middle of the 1990s and the cost of the midwife is absorbed by the government for the first three years as long as the midwife remains working in the village to which she is assigned. During this time, she is encouraged to develop a private practice.

Evaluation of the program is not straightforward. Comparisons of health of women before and after a mid-wife joins a community would reflect both the impact of the midwife and any other changes over time. From the outset, midwives were allocated to the poorest communities that did not already have a midwife and so comparisons of the health of females in

a community that had a mid wife with the health of females in a community without a mid-wife would reflect the combined influence of the presence of the mid-wife and differences across the communities.

To identify the effects of the midwives, Frankenberg and Thomas (2000) suggest that if mid-wives focused on the health of reproductive age females and had no direct influence on the health of males, then differences between communities would be reflected in the health of males. They use two waves of the Indonesia Family Life Survey (IFLS) to estimate the change in health of females in communities that did not have a midwife by the second wave and compare this change with the change among males in the same communities. This provides an estimate of health changes in "control communities". The difference in changes in health of females and males in communities in which a mid-wife joined the community between the survey waves provides an estimate of the differential health changes in "treatment communities". Comparing the differences in treatment communities with the differences in the control communities provides an estimate of the impact of the village midwife on the health of reproductive age females. This estimation strategy takes into account the fact that midwives are allocated purposively in communities where they are likely to have their biggest impact. The strategy also takes into account secular changes in health within each community as long as those changes are common for males and females. Frankenberg and Thomas (2000) focus on body mass index (BMI), an indicator of general health and nutritional status, and report a large and significant beneficial impact on the BMI of females from having a midwife join a community.

The strategy assumes the mid-wife has no direct or indirect impact on the health of males. Indirect benefits may arise if women learn about better health care practices that affect other family members or if women seek advice from the midwife about the health of others in their families. If these benefits are positive, the estimated effects of the midwife will be biased

downwards. However, if the presence of a midwife results in reduced resources being allocated to improve the health of males and children, the strategy would render upward biased estimates.

Frankenberg, Suriastini and Thomas (2000) address this concern by examining the impact of a midwife on child health. In this case, the identification strategy combines the advantages of the longitudinal design of IFLS with insights from the nutrition literature on the biology of linear growth. Specifically, health and nutrition interventions have their greatest impact on the length of children during the first few years of life. Adult height is largely determined by around age three or four. Thus, if a midwife joins a community when a child is age five or six, the services provided by the midwife are unlikely to have much impact on the child's growth trajectory. In contrast, if the midwife is present from the time the child is conceived, the child will be fully exposed to the potential benefits of the midwife.

Consider a "treatment" community in which a midwife joins the community immediately after the first wave of the survey. Comparing the height of children age eight in the first wave with children the same age in the second wave provides an estimate of the changes in height that can be attributed to secular improvements and are unlikely to be associated with the presence of the midwife. In contrast, the difference in height of children age three and under in the second wave and children of the same age in the first wave provides an estimate of the impact of being fully exposed to the midwife relative to having no exposure to her. However, this difference is contaminated by secular changes that might have occurred during this period. Thus, comparing the differences in heights of young children between the waves with the differences in heights of older children between the waves (which capture secular changes) provides an estimate of the impact of the midwife on the longer-term nutritional status of young children.

It is possible that there are cohort-specific growth trajectories. To assess whether this is important, an alternative strategy compares changes in heights of young children relative to older children in "treatment" communities with similar changes in heights of young and older children

in “control” communities which never had a midwife. Further, by comparing changes in height of older children in treatment and control communities, it is possible to evaluate the validity of assumptions in the model. A child who is fully exposed to a midwife is almost ½ a standard deviation taller than a similar child who is not so exposed which is not only significant but larger than the urban-rural gap in heights of Indonesian children. Related research documents that midwives are associated with improved birth outcomes including increased birthweight and fewer complications at birth. (Frankenberg, Buttenheim, Sikoki and Suriastini, 2008). Taken together, this research indicates that the introduction of improved reproductive health services in rural Indonesia has results in improved health of women and children.

These studies, based on observational data, are able to estimate effects that can be interpreted as causal because of the research designs which develop credible control groups with which treatments are compared. The impact of improved reproductive health services on economic outcomes is more tenuous with this strategy although the evidence suggests that improved reproductive health services is associated with elevated rates of labor force participation in the market and higher earnings among females in Indonesia.

Joshi and Schultz (2007) make more progress on this front by examining the impact of a family planning intervention that was randomly assigned to villages in Matlab district, Bangladesh. Specifically, starting in 1977, family planning services were provided on a door-to-door basis, in conjunction with maternal and child health services, in treatment villages. Control villages continued to receive services at the local clinics. Studies have documented large and significant declines in fertility (ICDDR,B 2004). Joshi and Schultz exploit the experimental design to assess the impact on health and economic prosperity of women exposed to the treatment and to also measure the effects of the program on the women’s children.

Drawing on survey data collected 20 years after the initiation of the program, the Matlab Health and Socio-economic Survey (MHSS), they report significant improvements in the health

of exposed women, higher rates of employment out of the home and greater economic productivity in those activities. The households in which these women reside are wealthier and more likely to have access to water within the family compound. The children of exposed women are more likely to survive to age 5 and they are more likely to be immunized. There is some evidence that their nutritional status is improved although there are only modest gains in schooling relative to children of women in control areas.

Frankenberg and Thomas (2007) highlight the potential impact of the program on female savings and empowerment. Specifically, they exploit a feature of the MHSS which, following IFLS, collects information on assets at the individual level within each household. Reduced fertility associated with exposure to the family planning outreach will reduce the number of children on whom a woman can call at older ages and so there is a greater incentive to accumulate wealth to draw on in later life. Moreover, if control over assets translates into control over decisions about resource allocations, then as women gain control over the fertility they may also be empowered to take control over other domains of the family economy. Women who were exposed to the family planning outreach treatment have significantly greater levels of assets relative to women not in the treatment areas. Treated women tend to accumulate gold and goats – assets that are traditionally controlled by women. Males in the treatment communities did not save more than males in control communities.

The combination of data from randomized trials and observational sources paints a picture of reproductive health programs yielding benefits not only in terms of greater control over fertility but also improved health and economic outcomes of women as well as greater accumulation of resources for the future. Further, there is some evidence that the programs yield benefits in terms of improved health of children. We turn next to a discussion of the evidence on the impact of child health on economic prosperity.

**Child Health and Economic Success**

A substantial body of literature has documented that adult height is correlated with not only improved health but also economic prosperity as indicated by higher rates of participation in the labor force and, among those who work, higher hourly earnings. These relationships have been established in the historical literature following pioneering work by Fogel (2004) and his collaborators. They have also been documented in the literature on developing countries (Strauss and Thomas 1998). Adult height is largely determined during early childhood and clearly economic prosperity during adulthood cannot have a causal impact on linear growth during childhood. It is, therefore, tempting to interpret these associations as indicating a causal effect of early childhood nutrition on economic prosperity during adulthood.

That would be premature. Factors that affect early childhood growth – such as genes as well as parental resources and their tastes for investing in the human capital of their children – might also affect economic prosperity during adulthood. In that case, height and educational attainment and possibly cognitive achievement will be positively correlated (Case and Paxson 2008). Genetic influences, cognition and tastes are seldom included in empirical models that relate adult height to economic prosperity and, without taking them into account, it is difficult to interpret the associations between attained adult height and economic prosperity.

Observational studies that relate height or weight of children to school performance suffer from similar problems although attempt have been made to treat the joint determination of child health and child schooling with an instrumental variables approach or by including family fixed effects in the models. Prices of inputs into the production of height are potential instruments; they may include (implicit or explicit) prices of local health services and prices of specific foods, relative to other prices. Estimation is complicated by the fact that child nutrition reflects both a stock and flow component and so the instruments necessarily capture variation in the environment to which the person was exposed during the fetal period and in the first few

years of life. As noted above, the use of local variation in prices and services is complicated if programs and subsidies are allocated taking into account child health, or if people move to areas because of improved services. (Schultz, 2002.)

Alderman, Hoddinott and Kinsey (2006) exploit variation in resources during the first years of life to measure the impact of early childhood nutrition on adult stature and schooling attainment. They exploit variation in resources due to droughts and civil war in Zimbabwe and treat this variation as unanticipated "shocks". It is not clear that this variation is entirely unanticipated and that parents do not respond to it by moving.

The authors find that early childhood nutrition has a large and significant impact on years of completed schooling indicating that public health interventions that assure infants and young children are well nourished are likely to have a direct impact on the human capital and economic prosperity of a population. (See Glewwe and Miguel, 2008, for a review of this literature.)

Numerous animal experiments and some small-scale randomized interventions with children have implicated both macro-nutrients and several micro-nutrients such as iron and zinc in cognitive functioning. Some of these studies have linked supplementation to school performance or economic outcomes. See Pollitt and others (1993) for a review.

Among the most influential studies of the impact of child nutrition on economic well-being is a randomized intervention conducted by INCAP in four villages in rural Guatemala between 1969 and 1977. Two villages were randomly assigned to receive a treatment of a fortified drink, Atole, which was high in energy and protein and contained iron and niacin. The other two villages were provided with sugared water, Fresco. Children from birth to age 7 along with pregnant and lactating mothers were eligible to receive the drink on a daily basis at a community center in each village. The study population was poor and undernourished. Prior to the intervention, about four of every five of the children age 2 years old in the villages were stunted (height for age z score < 2) and about one-third were wasted (weight for age z score < 2).

Children exposed to the treatment are taller at 36 months relative to children in the same villages who were not exposed to the treatment during their first few years of life. To isolate the impact of the supplementation, these differences across cohorts are contrasted with differences across the same cohorts in control villages. This is an important result as it establishes that a nutrition intervention in early life can have long-lasting effects on the health and well-being of a population. (Martorell and others, 2005.)

Maluccio and others (2006) use a similar approach to measure the impact of nutrition supplementation on cognitive achievement. In a follow-up study conducted in 2002-2004, when the study subjects were adults, abstract reasoning skills were measured with a Ravens progressive colored matrices test. Subjects who were exposed to the treatment during the first three years of life performed better on the test relative to those not exposed in early life. There is a small impact on the completed schooling of females and also on their language skills.

Related research explores the impact of the nutrition supplementation on economic productivity in adulthood. Using the same follow-up survey, Hoddinott and others (2008) use the same empirical strategy of comparing the cohort that was fully exposed to the supplement with earlier birth cohorts in the treatment villages and with similar cohorts in the control villages. They find that, among males who were working, supplementation results in hourly earnings being between 40% and 60% higher than they would otherwise have been. This is an extremely large effect. Elevated productivity was largely offset by reduced hours of work. Annual earnings are estimated to be about 7% higher among treated males relative to controls although this difference is not significant (which may reflect inadequate power due to small sample sizes). Among females, the authors find treatments tend to earn less per hour, work few hours and have lower annual earnings than controls. None of these differences is significant.

In sum, females benefit more in terms of school performance than males. But, among those who earn income, there is a 50% premium in hourly earnings among males who are treated

relative to controls. Compensating reductions in hours of work result in much smaller effects on annual earnings. There are no effects on the labor market outcomes of females.

Taken together, these results on schooling and labor market outcomes are puzzling. It is possible that they reflect a complex set of choices related to schooling, marriage and labor market participation that differs between males and females. It is also possible that the estimated effects are contaminated by attrition that differs between treatments and controls and also between males and females in ways that are related to labor market and schooling outcomes. The authors compare the characteristics of those respondents who were re-interviewed in the 2002-2004 follow-up with those that were not re-interviewed in the follow-up and conclude this is an unlikely explanation for the results. Since these comparisons are, by necessity, based on the observed characteristics of respondents when they were young children living in the study villages, it is not clear that they are very informative. As noted above, the key concern is that attrition may be related to unobserved characteristics that are correlated with health status and schooling and labor market outcomes. For example, if the children who are most likely to attrit are those that are the most ambitious, invest in their health and education, move to towns, Guatemala City or beyond the borders of Guatemala and get better jobs, then the estimated effects will likely be downward biased. However, if successful movers are more likely to remain connected to their families in the original villages and it is those young adults who have not done well in the labor market who attrit from the sample, then the estimated effects will be upward biased. Attrition is a vexing problem that has the potential to substantially complicate interpretation of evidence in longitudinal studies. (Thomas, Frankenberg and Smith, 2001.)

Putting attrition aside, the results on the longer-term impact of this early child nutrition intervention suggest that improvements in the nutritional status of children in the first few years of life result in improved human capital outcomes (as indicated by better health in later life, improved cognitive functioning and, among females, more schooling) and elevated economic

productivity (among males in the labor force). However, since there is little evidence that annual earnings are higher among treated cohorts, the benefits of improved childhood nutrition do not appear to translate into greater economic prosperity at the aggregate level.

The vast majority of the literature on the link between childhood nutrition and economic prosperity in developing countries has, appropriately, focused on under-nutrition. However, in advanced countries, rates of pediatric obesity are high and the evidence indicates obesity among young children is an emerging concern in developing countries passing through the nutrition transition (Popkin 1993; de Onis and Blossner 2000). Rapid changes in nutrition levels across cohorts in these countries may presage serious health problems as these children enter mid-life. Recall, the thrifty gene hypothesis suggest that it is children who are low weight at birth but overweight in adolescence that are at greatest risk of cardiovascular disease and diabetes in later life. Low birthweight is far more common in lower income countries and more likely among children whose mothers were poorly nourished as children as indicated by their height as adults (Victora and others 2008). There is very little scientific evidence on whether and how childhood obesity affects social and economic success in later life. This is an important gap in the literature.

Although nutrition has played a central role in the literature on health and development, communicable diseases remain a leading cause of death in many low income settings. Putting aside HIV, many of the diseases are preventable at modest cost. As an example, intestinal helminth infections, or worms, are prevalent in many parts of the developing world and can be effectively treated with readily available helmintic drugs. High worm loads results in iron deficient anemia and protein-energy deficiency which have, in turn, been associated with reduced cognitive development. The evidence from randomized trials that measure the impact of pharmacological interventions to treat helminth infections on cognitive performance of treated subjects is inconclusive (Dickson and others, 2004).

Successfully treating infectious diseases in a community is likely to have significant externality benefits. Miguel and Kremer (2004) take this as their starting point to estimate the causal effect of treating worms among school-age children using a random assignment treatment-control design in rural Kenya. In the first year of the study, 25 primary schools were randomly assigned to receive an orally-administered anti-helmintic drug; those schools and an additional 25 schools received the treatment in the second year. All 75 primary schools received the treatment in the third year. There are externality benefits: children who were not treated, but lived in the treated areas, benefit from the reduced worm loads in those communities. School attendance increased substantially as indicated by a 25% reduction in school absenteeism at schools after treatment. However, performance on school tests did not respond to the treatment. The authors conjecture that this may reflect the effects of congestion. It may also reflect on what is learnt in school and what is tested in examinations – which may itself explain the high levels of absenteeism among primary school children. It is also consistent with there being little impact of helminth infection on cognition (Dickson, 2000).

**Adult Health and Economic Success**

The evidence discussed thus far suggests that early life health likely affects later life economic productivity. The most convincing evidence highlights the role of early life nutrition and indicates that there is a large impact on hourly earnings. We turn next to assess whether changes in health during adulthood affects economic prosperity.

A temporary bout of poor health is likely to be accompanied by reduced time working and possibly lower productivity over the short run. Whether these effects translate into longer-term impacts on earnings is less clear. Individuals may compensate by allocating additional time to work when they are in better health. Family members may allocate their time to substitute for a member while he or she is in poor health (Thirumurthy, Graff Zivin and Goldstein, 2008).

Chronic diseases, and longer-term bouts of illness, are more likely to have permanent effects on economic resources. For example, studies have documented declines in the productivity of individuals working on tea estates in Kenya during the eighteen months prior to AIDS-related death or exit from the labor force (Fox and others 2004).

Determining whether changes in adult health have a causal impact on economic and social success is a substantial challenge. To the extent they exist, identifying the domains of health that are most important and the magnitudes of their impacts on different dimensions of economic prosperity poses even greater challenges. Convincing evidence will be founded on a model of behavior that takes into account investments in health over the entire life course, expectations about future health and labor market prospects, time allocation in the short and longer run as well as the broader role of family members in these decisions.

Whereas energy supplementation during the first two or three years of life has long-term benefits, experimental studies that involve calorie supplementation of male adults indicate modest effects on worker productivity. For example, sugar cane cutters in some villages in Guatemala were randomly assigned to receive calorie supplements while similar works in other villages were not. There were no differences in the productivity of the workers from the two villages (Imminick and Viteri, 1981). In contrast, construction workers in Kenya who were randomly given energy supplements were slightly more productive in road digging than the controls (Wolgemuth and others, 1982).

The nutrition literature has highlighted the important role that specific micronutrients play in the etiology of disease and functioning of the human body. Iron is a good example. Not only is iron deficiency prevalent throughout the developing world, particularly in south and southeast Asia, but studies have demonstrated there is a causal effect of iron deficiency on reduced work capacity suggesting there may be a direct link between iron deficiency and economic productivity (Haas and Brownlie, 2001).

Iron plays an essential role in oxidative energy production. Iron deficient anemia affects physical activity through two main pathways. As hemoglobin levels decline, the maximum amount of oxygen the body can use (aerobic capacity) declines. As iron stores are depleted, the amount of oxygen available to muscles declines, reducing endurance and causing the heart to work harder for the same activity. Iron deficiency is also associated with, *inter alia*, greater susceptibility to disease, fatigue and reduced cognitive development.

Rigorous clinical trials with animals and humans demonstrate a causal relationship between iron deficiency and reduced maximum aerobic capacity ($VO_2max$) which changes by about 25-30% as subjects are made anemic or receive adequate iron supplementation. Iron deficient anemia is also associated with reduced endurance at below maximal work rates.

Demonstrating iron deficiency impedes maximal capacity and endurance does not reveal the economic consequences of iron deficiency in daily life. Those consequences may be more closely aligned with energy efficiency (the amount of physiological energy required to perform a given task). In fact, evidence from randomized treatment-control studies of Chinese female cotton mill workers and Sri Lankan female tea plantation workers suggest that elevated productivity resulted in changes in time allocation. Specifically, both studies found no evidence of greater productivity per hour among iron-deficient subjects who received iron supplements relative to subjects who were not supplemented. However, in both studies, treated women re-allocate time away from work and towards non-work activities (Li et al., 1994; Edgerton et al., 1979). Results from a random assignment treatment-control iron supplementation study of male rubber workers, however, indicates that treatments who were anemic at the initiation of the study were able to tap around 20% more rubber after supplementation, relative to anemic controls (Basta et al, 1979). Interpretation of this result is complicated by the fact that attrition from the study exceeded 50%; if those subjects who did not benefit from the study were more likely to attrit, the estimated benefits will be biased upwards.

The Work and Iron Status Evaluation (WISE) is designed to examine the immediate and longer-term impact of providing iron supplements to older adults in Central Java, Indonesia (Thomas et al., 2006.) In the population, about one-quarter of older men and a third of older women are iron deficient (as indicated by low hemoglobin). Older males are randomly assigned to receive a weekly iron supplement for slightly over a year or an identical appearing placebo. Everyone in the man's household was similarly assigned to treatment or control (in the small fraction of households with more than one older male, a random male was selected to determine assignment to treatment or control for the entire household).

Respondents who were iron deficient prior to the intervention and who were assigned to the treatment have higher levels of iron in the blood and are able to cycle for longer on an ergocycle at the end of the intervention, relative to comparable control subjects. Iron deficient treatments are better off in terms of physical health, psycho-social health and economic success. Relative to similar controls, treated subjects are more likely to be working, lose less work time to illness, are more energetic, more able to conduct physically arduous activities and their psycho-social health is better.

About half the male workers in the study are self-employed (primarily rice farmers) and the other half are paid a time wage. There is no evidence that hours of work responded to the treatment for time-wage workers or the self-employed. Among males who earned a time wage, there is no evidence of changes in productivity as indicated by their hourly earnings. Of course, if their wages are set by an employer, it is not obvious the worker will reap the benefits of greater productivity. This is not true for the self-employed. Males who were iron deficient and self-employed at baseline reported 20% higher hourly earnings after 6 months of supplementation relative to similar controls. Since there was no change in their hours of work, this translates into 20% higher income from labor.

Wage workers who received the treatment reduced the amount of time spent sleeping by around 40 minutes per day and re-allocated this time to leisure. Self-employed workers made no such adjustments. A picture emerges of iron deficiency having a causal impact on work capacity

and energy needed to complete tasks. The self-employed who benefited from the treatment allocate the additional energy to their fields and work harder, produce more and earn more per hour of work. In contrast, wage workers were able to channel the greater energy to reduced sleep and more time allocated to leisure. After twelve months of supplementation, there is evidence that some of the treated iron-deficient males who were working for a time wage at baseline had shifted to self-employment or taken up an additional job.

While WISE demonstrates iron deficiency has a causal impact on time allocation and economic productivity, it also highlights the importance of taking behavioral responses to the experiment itself in assessing the impact of the treatment. Results from he Chinese and Sri Lankan studies reinforce this point. They indicate that iron deficiency had little impact on productivity but did affect how individuals allocate their time.

If iron supplementation improves the health and well-being of individuals, we expect they will not only be more productive at work but in other domains of their lives. Also, they may respond to these changes in many ways such as changing the nature of their work and time spent on work. The results echo the evidence from the INCAP study which indicated that treated subjects compensated for increased hourly earnings by allocating less time to work. It is critically important that all of these responses are taken into account if we are to understand the impact of health improvements on economic growth. This limits the contributions to the literature of experiments that are designed to isolate specific inputs and outputs in the health and productivity relationship and do not capture the broader ramifications of the health intervention.

Health care costs have skyrocketed and far outpaced inflation in many countries in recent decades. Much of the increase in advanced economies has been attributed to the development of technology that improves health and extends life (Cutler 2004). At the same time, calories have become inexpensive relative to other goods although food prices are subject to considerable variation from year to year and have increased dramatically in recent months. These changes in

prices, along with differences in prices across space, provide potentially powerful sources of variation to identify the impact of changes in health of adults on social and economic success.

The assumptions under which identification will be valid are not obviously satisfied, at least in general. First, the relative price changes should have no direct impact on the economic prosperity of an individual. In the case of food prices, this rules out the agricultural sector including all farmers along with workers in food-related industries. Workers in the health sector – and allied industries – are similarly affected. Second, variation in prices reflects the combination of a number of factors that conspire to bring demand in line with supply in a general equilibrium. To the extent that those factors also influence economic prosperity, it will be difficult to pin down the impact on health status. This is a special challenge when relying on only temporal or spatial variation in prices. Third, measurement of markers of health in survey data that are both affected by variation in prices and also affect economic success raises additional challenges.

These issues may be side-stepped by relying on experimental or quasi-experimental variation in prices. For example, subjects enrolled in the RAND Health Insurance Experiment (HIE) were randomly assigned to one of 14 fee-for-service health plans starting in 1974. About 1,800 subjects were assigned to a plan that provided free care while the remaining 2,800 subjects were assigned to plans that varied in terms of the co-payments (from 25% to 95% of fees) and total out-of-pocket expenses. Almost three-quarters of the subjects were enrolled in the experiment for three years. The rest participated for five years.

Newhouse and others (1993) describe the experiment and key results. Use of health care declined as the price increased with subjects in the free-care treatment using health care services most intensively. The evidence on health outcomes is mixed. Newhouse and colleagues conclude that the health of the average person enrolled in the HIE was not adversely affected by reduced use of health services. They note, however, that the health of the poor who were sick improved more among those who faced lower prices. For example, among the poor who were enrolled in the free

care plan, relative to other plans, mortality was lower, blood pressure was improved more, vision was corrected more often, dental health was better and the incidence of anemia was reduced more. All of these indicators were measured through clinical testing or from blood samples.

In stark contrast, self-reported general health of this group deteriorated during the period! Specifically, of 11 self-reported health indicators, 9 were reported to have worsened among those who received free care relative to those who incurred some cost when seeking health care – although only one of these differences is significant. The clinical and biomarker evidence indicates these people were, if anything, in better health and so one might suspect that, as a result of having greater exposure to the health care system, these people learnt more about their health than they would have if they had used services less frequently and it is this information that is reflected in the worse self-assessed health outcomes. These results provide a practical example of some of the difficulties associated with health measurement in population-based surveys.

Dow and others (2008) side-step these problems with measurement of health and examine the impact of randomly-assigned variation in the price of health care on labor outcomes. They find that females who received free health care are significantly more likely to be working at the end of the intervention than females who had to pay for health services. The difference in participation rates is about 4 percentage points. Males who had not completed high school are about 8 percentage points more likely to be working if they received free care relative to those who paid for care (although this effect is only significant at a 10% size of test).

A similar experiment conducted in Indonesia involved changes in the prices of health services. To measure the impact of increasing user fees, prices of basic services at public health centers were raised in randomly selected "treatment" districts while prices were held constant (in real terms) in neighboring "control" districts. Subjects were interviewed before prices were increased in 1991 and again two years later. Paralleling the results for the HIE, respondents in the areas where prices had increased used less health care and also reported their general health as

having improved relative to subjects in the control areas where prices had not been changed. However, more specific indicators of health indicated higher prices resulted in worse health. Specifically, respondents in areas where prices increased reported they had more difficulties doing physically demanding tasks, spent more days away from work and had more days in bed because of illness. These results further underscore the challenges associated with health measurement.

Turning to work outcomes, areas where prices of health care increased, employment rates increased among males and females relative to those in areas where health prices did not change. These effects are largest among the least educated. The most plausible interpretation of both the HIE and Indonesian results is that the average treatment effects on labor supply indicate a causal role of improved health on the allocation of time to the labor market.

Evidence regarding the impact of health on economic productivity in the Indonesia study is less clear. Wage growth was lower among workers in the market sector living in the areas where health care prices were raised relative to the control areas. The estimated effects are large (around 15%) but significant at only a 10% size of test.

Quasi-experimental evidence supports the conclusions that health affects employment outcomes. During the 1960s and early 1970s, Canada introduced national health insurance. Exploiting the fact that the introduction of the system was phased across provinces and occupations, Gruber and Hanratty (1995) find that employment and wages increased as workers were covered by national health insurance. The authors conclude that labor demand rose because workers were more productive, either because of increased job mobility and better matching of skills or because their health improved as a result of being covered by health insurance.

While the introduction of national health insurance was not designed as an experiment, this study exploits the fact that some people were covered by the system before others. The plausibility of the results rests crucially on the extent to which this "natural experiment" approximates random assignment. The authors provide a compelling argument in favor of this interpretation.

Designing health policy innovations so that they are phased in and rigorously evaluated during the phase-in period will not only contribute to improved policy design and implementation. It also has the potential to contribute to identifying the broader impacts of the policy innovations beyond health care and health outcomes to economic and social indicators. It is unfortunate that this is a relatively rare design.

Mohanan (2009) uses a quasi-experimental design to examine the impact of bus accident injuries on household consumption, assets and debt. Drawing study subjects from among people who travel on buses, he compares those who sustained an injury in a bus accident with those who did not and find that debt is the principal mechanism used by households to mitigate the impact of the injury. This is a clever study which calls into question other research that suggests individuals are able to smooth consumption in the face of health shocks (Gertler and Gruber, 2001). See also Genoni (2009) who follows the approach used by Gertler and Gruber but comes to the same conclusion as Mohanan.

Turning to the literature based on observational data, the challenge is to identify instruments that are, first, predictive of health status and, second, valid in the sense of not being correlated with unobserved factors that affect the economic or social outcome of interest in the model. The experimental results suggest that prices of health inputs (including foods) may satisfy the first condition for measures of productivity. Labor supply effects are harder to identify in this context since health inputs will affect real wages and thus the value of time.

Studies have explored the relationship between body mass index BMI and labor market outcomes. BMI, which depends on net energy intake and varies through the life course, captures both longer and shorter run dimensions of nutrition. It is related to $VO_2$max and, thus, aerobic capacity and endurance independent of energy intake (Spurr 1983). Whether this pathway is one through which health importantly influences productivity is not obvious since many jobs do not

require sustained physical effort. Treadmill tests suggest that excess (fat) weight affects the efficiency at which energy is transferred to work output (Cuerton 1992).

Using data on workers in the rural Philippines, Haddad and Bouis (1991) find no effect of BMI on earnings of agricultural works. However, using the same sample, Foster and Rosenzweig (1993, 1994) highlight the difficulties of observing health and argue that employers use BMI as a marker for health. They contrast workers paid by time with those paid on a piece-rate basis and find that BMI affects the wages of only time-rate workers. This suggests that it is not health (or at least robustness) that is rewarded in the labor market but rather there is a benefit from conveying the impression of being in good health. It is not clear why employers would be consistently fooled into paying a worker more because he is heavier (but not more productive) or why employers would not learn about the productivity of workers.

Thomas and Strauss (1997) explore this issue among male and female workers in urban Brazil. Using relative food prices as instruments, BMI has a positive impact on the hourly earnings of both employees and the self-employed suggesting that BMI is more than just an easily observed marker of health used by employers. Since the effect of BMI on hourly earnings is largest among the least educated, who are the most likely to do manual labor, the authors conjecture that BMI may be a marker for strength. Similar evidence is reported for African workers by Glick and Sahn (1998) and Croppenstedt and Muller (2000).

There is little empirical evidence relating BMI to labor supply. BMI has been shown to affect the proportion of working time that is spent on very physically demanding activities by men. (Pitt, Rosenzweig and Hassan 1990; Bhargaca 1994 and Fafchamps and Quisumbing 1999).

Adams and others (2003) take a different approach to evaluating the direction of causality underlying the associations between health and SES. They examine whether prior SES predicts current health status, conditional on an extensive set of information on health histories. Under the assumptions of their model, if prior SES does not predict health innovations, then a direct causal

pathway from SES to health can be rejected. This is essentially a test for Granger causality. Using data from older adults in the United States, they conclude there is little evidence that SES has a causal impact on health except, perhaps, in the case of psycho-social health which the authors attribute to the fact that Medicare does not cover mental health services. Their conclusions on the impact of health on SES are more tentative although they generally find little evidence that health affects wealth of older adults.

The assumptions underlying these tests are not innocuous. The models include a battery of prior health indicators, each of which is the outcome of choices made over the life course. If those choices are correlated with SES, the assumptions underlying the empirical strategy will be violated. In addition, there are legitimate questions about whether the testing strategy has sufficient power to detect causal effects of SES on health in a broader class of models.

In an effort to assess the importance of this concern, Adda and others (2003) conduct a replication using the same empirical methods with data from the United Kingdom and Sweden, They find results that are remarkably similar to those for the United States including the evidence on psycho-social health. This is troubling since, in the United Kingdom and Sweden, all health services are available for free – including mental health services. The authors conclude that the testing strategy lacks power against plausible alternative models.

It is likely that the dynamics underlying observed health and observed SES are very complex. For example, a person's health may be in decline prior to a diagnosis of, say, heart disease and the decline in health may result in reduced earnings because of reduced hours or lower hourly earnings. Attempts to link the diagnosis to reduced wealth are unlikely to be fruitful. Similarly, a person who perceives their health is in decline and anticipates having reduced earnings in the future may choose to increase their labor supply and thus earnings now.

More generally, the dynamic links between health and economic prosperity have been little studied. This is an important gap in the scientific literature for several reasons. Health is a stock

that evolves over time, and prior health behaviors --and health shocks-- likely influence current economic status. Virtually nothing is known about the speed with which the effects of health transitions at the individual level are transmitted to the labor market in low income settings. Does a period of poor health (or a negative health shock) put a worker on a permanently lower wage trajectory or do the negative consequences of ill-health dissipate as health subsequently improves? The extent of catch up likely depends on the nature of the health problem, the structure of the labor market and characteristics of the worker including age, education and the extent to which the individual has a buffer of resources on which to draw in bad times.

Developing a better understand the dynamic inter-play between health and economic prosperity is likely to shed new light on some of the mechanisms through which the two covary. Apart from the econometric advantages associated with analyses based on repeated observations of the same person over time, "natural experiments" arising from unanticipated variation in a respondent's life is likely to be a powerful resource in this literature.

Thomas and Frankenberg (2000) provide an example. The first column of Table 2 uses data on males age 18 through 68 interviewed in the 1997 wave of IFLS and indicates the elasticity of wages with respect to BMI is 2.0. Results in the second column control height, age, education and location of residence: the elasticity is reduced to 1.0 indicating that current weight reflects, in substantial part, human capital and background characteristics. Prior BMI might serve as a control for these characteristics and so, exploiting the repeat-observation dimension of IFLS, BMI measured in 1993 is added in column 3. It does predict current wages, and soaks up some of the correlation with current BMI. Controlling 1993 BMI, 1997 BMI has the interpretation of weight gain since 1993: its effect is also positive. The interpretation of that correlation is not obvious. It is possible that increased wages were spent on more energy intake (or less energy output) or that changes in both wages and weight arise because of some other unobserved factor.

To explore this futher, we turn to wages measured in 1993. Column 4 essentially replicates the 1997 results reported in column 2. Wages in 1993 are related to BMI in 1993 and to BMI in 1997 (column 5). BMI in 1993 remains significant. However, weight gained between 1993 and 1997 is a significant predictor of wages measured in 1993, prior to that weigth change. This is compelling evidence that BMI and wages are jointly determined (or influenced by other unobserved factors) and that the regressions in Table 1 cannot be given a causal interpretation.

This evidence raises further doubts regarding the validity of the approach to identifying causal effects taken by Adams and others (2003). We conclude that modeling the dynamics underlying the evolution of health and economic prosperity remains an important scientific endeavor.

**Conclusions**

The weight of the evidence indicates that fetal health and early childhood nutrition have a causal impact on human capital accumulation and economic productivity. There is some evidence indicating that changes in adult health affect labor outcomes, particularly productivity. While the exact mechanisms underlying these relationships are not entirely clear, the most convincing evidence is distinguished by the co-existence of carefully conducted experimental studies and observational studies that have documented sizeable effects of nutrition on productivity indicators. Explanations that are founded on an understanding of the underlying biological mechanisms have proved to be a powerful force in support of these conclusions.

Little attention has been paid to the behavioral response to longer-term improvements in health and productivity. The limited evidence suggests that short-term increases in productivity have little immediate impact on time allocation but if the productivity gains persist over the longer-term, individuals reduce their hours at work as would be suggested by theory. If this turns out to be a general result, it is important.

Studies that focus exclusively on productivity and studies that focus solely on annual earnings will provide incomplete descriptions of the relationships between health and labor outcomes. By ignoring the fact that individuals choose to substitute time away from work, these studies will tend to understate the welfare gains associated with health improvements. Those benefits are not reflected in aggregate statistics of economic growth. Scaling estimated productivity effects to the aggregate level will overstate the extent to which the macro association between health and income can be attributed to a causal effect of health on economic prosperity.

A plausible argument can be -- and has been -- made that other dimensions of health likely affect economic prosperity. Relative to the impact of reducing nutritional deficiencies on productivity, these links have not been reliably established. In spite of their prevalence across the globe, there is little evidence on the impact on socio-economic success of obesity and related health conditions, of infection and inflammation, of stress or of injuries. Remarkably little is known about the impact on economic prosperity of psycho-social health problems.

The cumulative effect of multiple health insults on health outcomes in later life has been highlighted in the literature on older adults in advanced economies (McEwan 2000). It has received little attention in the developing country literature in spite of the fact that health insults are more frequent and potentially more damaging in lower income societies and may take their toll in early or mid-life. It remains unclear whether wear and tear on the body due to health insults has implications for economic prosperity.

More generally, there is a paucity of evidence on the dynamics linking innovations in health and innovations in economic prosperity, particularly over the longer term. Studies that take into account the shorter and longer-tem behavioral responses that accompany health innovations are few and far between.

In the literature relating health to SES, inadequate attention has been paid to different domains of SES. The evidence discussed above suggests that the impact of particular health

markers on productivity at work and time allocation may be quite different and that these differences may only emerge over the medium or longer term. Similarly, there are likely to be distinct effects of health on education and cognition in early life and cognitive performance in later life. The impact of health on productivity in home production, choices between consumption and savings – and inter-temporal preferences – are also likely to be different.

These are all critical gaps in our knowledge. Filling them calls for a sustained program of research that integrates the advantages of randomized treatment-control designs, phased policy interventions and broad-purpose, longitudinal social surveys. Each of these tools, on their, own is unlikely to provide the scientific evidence necessary to make progress in the field and thereby influence policy design.

Sustained longitudinal studies that collect information with repeated observations on the same individual will be necessary to understand dynamic aspects of the links between health and SES over the short and longer-term. Broad-purpose panel surveys, in combination with the changes that will occur in the global economy, have the potential to provide a rich laboratory for understanding the links between health and economic success. Financial crises, the collapse of markers, price spikes and natural disasters are all candidates for this research. However, it is often difficult to separate “shocks” from events that might have been predicted in which case estimated effects of the observed change will be contaminated by behavioral response that anticipate the change. Separately identifying the effects of multiple, related changes is very complex and the level of this complexity likely increases as the magnitude of the “shock” increases.

Relying on these “natural experiments” alone is unlikely to suffice. Conducting pilot studies of the impact of policy changes has the potential to yield important new insights that will improve policy design and affect the state of science. Co-ordinating longitudinal health and socio-economic surveys with phased policy changes and health interventions provides opportunities for more planning in the study design and greater control over the variation that people confront.

There are good reasons to expand the horizons of experimental studies. Specifically, measuring broader socio-economic outcomes than is typically the case, examining behavioral responses to the treatment and following subjects for an extended period will substantially increase the contributions of these studies. For example, an intervention that seeks to eradicate malaria in an area might not only measure its incidence before and after the intervention, but also track changes in economic productivity as well as behavioral responses to the intervention (such as changes in work status, hours of work, time allocation, migration and investments). Since economic and social changes will likely take some time to emerge, subjects will need to be tracked beyond the intervention period. Taking account of the medium and longer term impacts on both health and economic outcomes will provide a more comprehensive assessment of the intervention.

Conducting randomized interventions or phased trials in parallel with large-scale panel studies has the potential to significantly affect science and policy design in cost-effective ways The breadth and quality of research in this area is poised to make a quantum leap by exploiting rapidly evolving technologies for health measurement in a field setting along in conjunction with innovative techniques for combining state-of-the-art survey data and administrative records on individuals, their families and their communities.

Developing the knowledge base necessary to understand the complex linkages between health and economic prosperity is far from straightforward. However, the challenges provide an extraordinary opportunity for health scientists, social scientists and practitioners to collaborate in order to yield new knowledge that may have a lasting impact on the well-being of populations across the globe.

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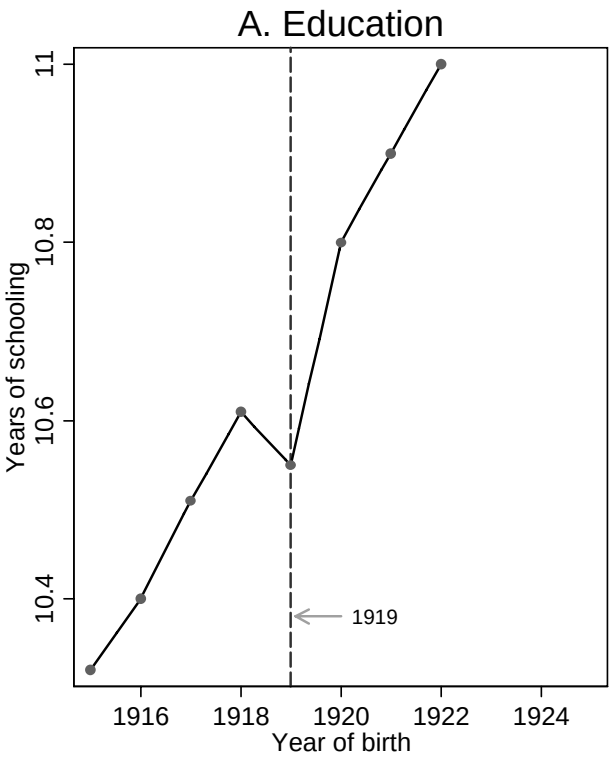
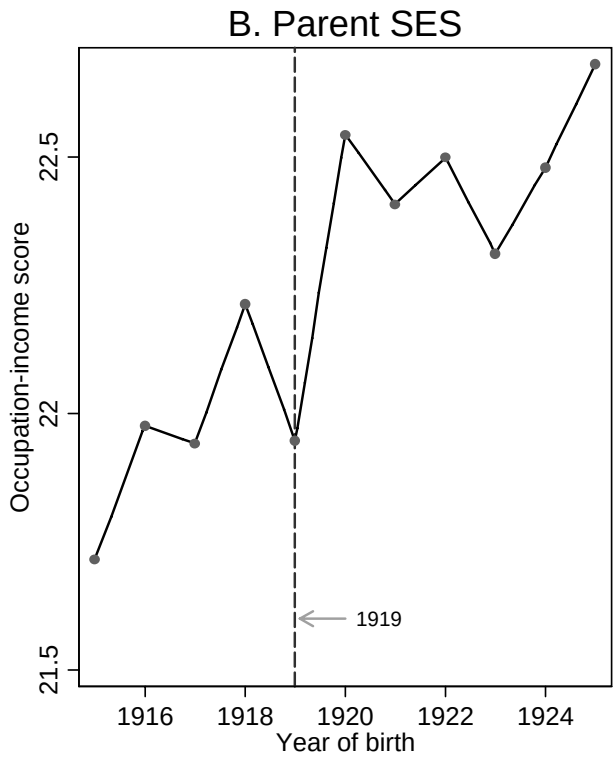
# Figure 1: Fetal health, own education and SES of parents By year of birth




Source: 1960 U.S.Census when 1919 cohort age 41




Source: 1930 U.S. Census when 1919 cohort age 11

## Table 1: Human capital and exposure to the 1974 floods in Bangladesh

| Year of birth | % Population | OLS | | HH Fixed Effects | |
|---|---|---|---|---|---|
| | | Unable to read | Unable to read, write or do maths | Unable to read | Unable to read, write or do maths |
| | [1] | [2] | [3] | [4] | [5] |
| 1971 | 9.2 | 0.9 | 1.1 | -3.2 | -2.8 |
| | [0.5] | [0.2] | [0.3] | [0.6] | [0.5] |
| 1972 | 9.4 | -1.7 | -2.5 | -0.4 | -0.5 |
| | [0.5] | [0.5] | [0.7] | [0.1] | [0.1] |
| 1973 | 11.7 | -1.9 | -2.6 | -6.1 | -5.7 |
| | [0.5] | [0.6] | [0.8] | [1.2] | [1.2] |
| 1974 | 11.4 | -6.3 | -7.2 | -3.8 | -3.3 |
| | [0.5] | [1.8] | [2.2] | [0.7] | [0.7] |
| 1975 | 9.0 | -9.6 | -9.6 | -5.2 | -4.8 |
| | [0.4] | [2.6] | [2.8] | [0.9] | [0.9] |
| 1976 | 13.0 | -8.4 | -9.0 | -3.0 | -3.7 |
| | [0.5] | [2.5] | [2.9] | [0.6] | [0.8] |
| 1977 | 14.4 | -0.2 | -0.2 | -0.2 | 1.7 |
| | [0.5] | [0.0] | [0.1] | [0.0] | [0.4] |
| 1978 | 12.6 | -1.1 | -0.6 | 0.5 | 0.6 |
| | [0.5] | [0.3] | [0.2] | [0.1] | [0.1] |
| Constant | | 16.0 | 14.8 | 15.3 | 13.4 |
| | | [6.1] | [6.0] | [3.9] | [3.7] |

**Source**: Authors calculations using data on respondents born between 1970 and 1978 in the Matlab Health and Socioeconomic Survey. There are 2,064 males. T statistics reported below coefficient estimates. 1970 birth year is the excluded category in regressions reported in columns 2 through 5.

## Table 2: Dynamic relationship between body mass index and hourly earnings of adult males

Indonesia Family Life Survey, 1993 and 1997.

| | **ℓn(hourly earnings) in 1997** | | | **ℓn(hourly earnings) in 1993** | |
|---|---|---|---|---|---|
| | BMI97 only | Add height & education | BMI97 & BMI93 | BMI93 | BMI97 & BMI93 |
| | (1) | (2) | (3) | (4) | (5) |
| Covariates | | | | | |
| ℓn(BMI) in 1997 | 2.03 | 1.03 | 0.82 | . | 0.35 |
| | (0.12) | (0.10) | (0.16) | | (0.17) |
| ℓn(BMI) in 1993 | . | . | 0.34 | 0.96 | 0.78 |
| | | | (0.17) | (0.14) | (0.18) |
| ℓn(height) | . | 2.36 | 2.40 | 2.27 | 2.52 |
| | | (0.34) | (0.44) | (0.40) | (0.44) |
| Years of education (spline) | | | | | |
| 0- 5 | . | 0.05 | 0.04 | 0.05 | 0.05 |
| | | (0.01) | (0.01) | (0.01) | (0.01) |
| 6-11 | . | 0.09 | 0.10 | 0.09 | 0.10 |
| | | (0.01) | (0.01) | (0.01) | (0.01) |
| >11 | . | 0.11 | 0.10 | 0.14 | 0.14 |
| | | (0.01) | (0.01) | (0.01) | (0.01) |
| Age & location controls | No | Yes | Yes | Yes | Yes |
| $R^2$ | 0.06 | 0.29 | 0.30 | 0.30 | 0.30 |

**Source**: Author's calculations using 1993 and 1997 waves of the Indonesia Family Life Survey. Sample includes adult males age 18 through 68 earning income at time of each survey. Standard errors in parentheses robust to arbitrary forms of heteroskedasticity and take into account correlations due to survey clustering. Age is specified as spline; location includes control for each province and urban sector.