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**Comment** Amitabh Chandra

In 1996, Lant Pritchett and Lawrence Summers produced a highly influential paper that was boldly titled “Wealthier is Healthier.” Their work along with the important contributions of their predecessors, notably Thomas McKeown and Robert Fogel, has advocated the sanguine view that increases in national income will improve population health. There is no disputing the striking cross-sectional association between income and conventionally used measures of health such as infant mortality and life expectancy (see figure 1 of Pritchett and Summers 1996). Indeed, since the pioneering work of Preston (1975), who first illustrated the striking relationship between life expectancy and income, social scientists have been primed to believe that countries will move along these curves as their incomes increase; so seductive is the power of graphical regularities. The key question for any scientist interested in population health is the degree to which this association is causal. If Preston curves shift upward or rightward over time, then the cross-sectional relationship is completely spurious.

Motivated by this ambitious agenda and armed with a cornucopia of data, largely collected by their own tenacious efforts, Anne Case and Angus Deaton have written a magnificent paper, which provides a nuanced interpretation for the aforementioned observation that wealthier is healthier. Few questions in economics or, for that matter, in all of social science can claim to be as fundamental to human well-being as this one. First, they consider their macroeconomic evidence: for most countries, the historical record points to a fragile relationship between decennial change in gross domestic product (GDP) growth per capita and improvements in life expectancy. In India and China, which together account for over two-thirds of the human experience since 1950, the preceding relationship is essentially zero (and often negative) for ten-year changes that occurred in the late twentieth century. We can bicker about whether these regressions should be weighted or whether they should report jackknifed coefficients, but we should not miss the substantive point: it is astonishing to note that even for countries that lie on the “steep” part of the Preston curve, those who have not yet transcended the epidemiological transition, the relationship between per capita incomes and improvements in health is tenuous.

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Economic growth appears to move the entire Preston curve to the right, raising incomes, but with no effect on measured population health. This result has important implications for the literature on the health effects of globalization. I do not know the magnitude of the relationship between globalization and its effects on economic growth (as proxied by changes in per capita income), but let us assume that this channel is large and robust. In the context of the Case-Deaton results, the role of per capita income is estimated to be too minor to actually improve population health in the short run. Critics of globalization may cheer at the impotence of the wealthier is healthier mechanism, but their glee is premature; Case and Deaton are the harbingers of unpleasant news for both champions and critics of globalization. If we hypothesize that globalization is detrimental for population health, either by increasing inequality and relative deprivation or by reducing per capita incomes, then the evidence for that proposition is also extremely weak.

Even over forty years' changes, the relationship between average per capita income growth and health is weaker than one may have expected (figure 9.3). This is a result with enormous policy relevance: virtually every theory of development would predict that nations with higher average growth rates over thirty to forty years would be able to realize improvements in health. This relationship may operate through improved investments in nutrition, public health, or women's education. The conspicuous lack of a relationship in both decennial and longer-term changes challenges the empirical basis for providing aid initiatives that are biased toward targeting economic growth. Rather than assume that economic growth causes governments to push policy levers that affect health, might it be wiser to offer incentives to push these levers directly? What levers are these? What is the historical record on such policies, and what are the determinants of successful policies that improved population health? Addressing this question is beyond the scope of this paper, but should provide an active research agenda for development economists.

### **“Wealthier is Healthier” and Its Discontents**

Much of the belief (among economists) that “wealthier is healthier” has its intellectual antecedents in work of Prichett and Summers (1996; henceforth, PS) who studied the relationship between increases in real income, infant mortality, and life expectancy. For many reasons, the popularity of this argument is overstated: (1) PS found an inverse relationship between the first two variables, but not between the first and third. This is surprising: given that life expectancy at birth is largely determined by infant and child mortality (not in the post HIV era, but that is not of relevance for the time period studied by PS), the lack of a relationship causes a skeptical reader to ponder the robustness of their principal result. Clearly, more research into this puzzle is warranted. (2) It is not clear that outcomes such

as infant mortality are the sufficient statistics for population health. The incidence of chronic conditions in the adult population such as diabetes, kidney disease, tuberculosis, and asthma will not affect life expectancy tabulations for several decades. (3) Higher growth rates may mask the fact that economic gains from higher growth rates may accrue to a small fraction of the population; if 5 percent of people get 95 percent of the increase in income, then it would be difficult to see a relationship between per capita and health (or the result is being driven for those whom incomes increase). In other words, if the increase in income is unbalanced across the population, population health may increase less than the health of those who benefit from income gains.

To some degree, the popularity of this theory is also a consequence of economists' intellectual predilection that higher incomes *must* improve health: either through access to better healthcare, or through the provision of better public health, population health *must* progress. The first channel may or may not be protective of health. While it is probably not damaging, higher incomes for richer countries may translate into marginal investments in health care. Better dental care, use of antihistamines, and more frequent office visits are utility enhancing but have not been shown to affect long-term measures of health. More intensive use of technologically advanced medicine has been shown to be inversely associated with "quality care" in the United States (Baicker and Chandra 2004). As Jack Wennberg and his colleagues at Dartmouth have argued for decades, "more is not better" (Fisher et al. 2003a,b).

Conscious of these concerns, Case, Deaton, and their collaborators collect data from three poor populations in India and South Africa. They obtain on assets, anthropometrics, self-reported health, depression, anxiety, blood pressure, and a rich set of health conditions. This is an extraordinarily challenging enterprise and one that established a new standard for empirical work in economics: it is no longer sufficient for us to claim "no data" as an excuse for less rigorous empirical work. The results from this new micro data set are striking. Despite having income levels in the ratio of 4:2:1 between South African communities and Udaipur, and ownership of durable goods in the ratio of 3:2:1, the correlation across the prevalence of twenty-two symptoms is 0.84 (figure 9.9). I was struck by three facts: South Africans are more likely to report missing meals, there is anecdotal evidence of child malnourishment, and the prevalence of obesity among South African women is striking. In addition to casting doubt at the wealthier is a healthier hypothesis, they also portend worsening outcomes in years to come. While it has been suggested that the association between obesity and mortality is decreasing over time as medical science improves its understanding of obesity (Flegal et al. 2005), this view appears to be idiosyncratic to the second and third waves of the National Health and Nutrition Examination Survey (NHANES) relative to the first. Attempts to

replicate this result in other data sets have failed (Calle et al. 2005). It is probably best to interpret the presence of obesity (as measured by a body mass index [BMI] greater than 30) as doubling the risk of death (all cause) relative to those with BMI of 23.5 to 24.9 (Calle et al. 1999). With this interpretation, being wealthier is certainly not healthier: a point that is further reiterated when we note the significantly higher obesity rates among African American women who are undoubtedly richer than their South African counterparts.

Let us explore three potential explanations why the maxim “wealthier is healthier” may not be as accurate as previously thought. With these explanations, I am not arguing that “wealthier is unhealthier.” Rather, I am interested in pushing the observation that (1) income is neither necessary nor sufficient for improved population health, and (2) even though increasing income may be protective of population health, there may be aspects of this increase that *may* undo some the protective gains in certain populations.

### Public Health

Efficacious public health polices may be the drivers of large gains in population health, but may be the consequence of good governance, foreign aid, and knowledge transmission, none of which require higher income as a prerequisite. In the framework of Preston curves, such policies move the Preston curve upward; population health improves without any change in incomes. To understand public health’s role more concretely, we would want to understand the history of efficacious public health programs that targeted smallpox and cholera in 1960s and early 1970s in India. Were these campaigns the consequence of political willpower, the presence of an extensive public health service, or generous funding from the World Health Organization (WHO)? Similarly, in March 2006, the media was abuzz with reports that global measles deaths had fallen 48 percent over a five-year period. This improvement was attributed to a massive WHO-coordinated immunization program in regions of sub-Saharan Africa, where measles and its attendant maladies—pneumonia and diarrhea—are responsible for hundreds of thousands of deaths. The majority of these deaths have been preventable since 1960, when an economical and highly potent vaccine for measles was invented. This decline in child mortality is clearly not associated with improvements in sub-Saharan incomes, and one would like to know the details of how and why this particular initiative was so successful forty years after the discovery of the innovation that made it feasible. Finally, the iodization of salt is championed as being one of the great success stories of public health, but we do not have a single high-quality evaluation of this intervention. What was its effect in reducing the incidence of goiter (and consequently, retardation and perhaps educational attainment)? One key testable prediction from the hypothesis that public health interventions improved health is that the variance of

outcomes should fall with the successful implementation of these programs. In the spirit of these questions, David Cutler and Grant Miller (2005) study the historical record in America between 1900 and 1936, and argue that the provision of clean water can explain half the decline in total mortality, three fourths of the decline in infant mortality, and two-thirds of the decline in child mortality. Their calculations suggest that every dollar invested in this program yielded 23 dollars in social benefit; even the upper bounds of the “wealthier is healthier” hypothesis do not generate returns of this magnitude. More work on the precise mechanisms by which population health improves would help both governments and aid agencies.

### **The Nutrition Paradox**

I would like to draw attention to the “nutrition-paradox” that we have begun to see in developing countries. Caballero (2005) provides an extremely lucid introduction to this phenomena, which refers to the finding that many households in poorer countries have an underweight *and* overweight family member, and my discussion draws from his article. Doak et al. (2005) demonstrate that 60 percent of families with an underweight member also have someone who is overweight. This trend is not ubiquitous across all countries, but tends to be seen in middle-income countries (i.e. those with annual per capita incomes of approximately \$3000). Monterio et al. (2004a) demonstrate that being poor is protective of female obesity in low-income countries, but is a risk-factor in middle income countries. We do not understand the mechanisms underlying this phenomenon. Any coherent explanation must be able to reconcile (1) the inverse U-shaped relationship between the prevalence of obesity and per capita incomes (note that most of the historical record points to obesity being a disorder that accompanies prosperity), (2) its concentration in women, and (3) the puzzle that other members of the same household are malnourished. Factors such as a decrease in agricultural labor, or increased access to calorically rich foods, would explain the growth of obesity in adults. Why this should be concentrated among women is a question to which I have no answer, unless the factors noted previously apply disproportionately to women. The large prevalence of female obesity noted by Case and Deaton in Khayelitsha is not unique to that part of the world. Monteiro, Conde, and Popkin (2004) study the experience of Brazil and demonstrate that economic growth has reduced the prevalence of underweight women but has replaced it with obesity (in 1997, 9.5 percent of the poorest women were underweight, while 13 percent were overweight; in 1989, those numbers were 9.7 percent and 8 percent, respectively, while in 1975, the corresponding percentages were 17 percent and 4.7 percent).

Explaining the joint prevalence of obese and underweight members of the same family is difficult. If the underweight members of the family are children, and the obese are adults, then we may be able to appeal to an ex-

planation that is grounded in the quality of calories ingested. Caballero (2005, 1515) sums this up succinctly:

Although many of these low-cost commercial foods are energy-dense, they may be nutrient-poor. And nutrient density is particularly important for growing children. For example, on a per-calorie basis, a five-year-old boy needs five times as much iron in his diet as a man. Cheap, energy-dense, nutrient-poor foods may adversely affect the growth of the child but may provide sufficient calories for the adult to gain excess weight.

I agree with his assessment, but we should note that the explanation is speculative and has not been proven. Much more work needs to be done to determine how diets interact with gender and age to produce overweight mothers and underweight children.

In contrast to piecewise theorizing, where one uses multiple theories to reconcile different parts of the data, it may be useful to evaluate the empirical content of the “fetal origins” hypothesis in explaining some of the facts on obesity. This tantalizing hypothesis come in many flavors, but one version that may be particular relevant for interpreting the results in Case and Deaton’s paper is noted by Caballero (2005): might early exposure to an environment with pervasive malnourishment activate genes that are responsible for the body accumulating body fat? This response would be maximizing in an evolutionary context, but devastating for an individual who is exposed to an environment where energy-dense foods are cheaply available. From the work of Daviglus et al. (2004), we know that obesity in young adulthood is associated with a significant increase in the Medicare expenditures on treating cardiovascular disease and diabetes. Despite the potential importance of this theory, most tests of the fetal-origins hypothesis continue to be muddled because of their uncontrolled nature, but the study by Ravelli, Stein, and Susser (1976) does stand out. In this report, 300,000 men who were exposed to the Dutch famine of 1944 had their health assessed at the age of nineteen (as part of military conscription). For those exposed to the famine in the first half of the pregnancy, significantly higher obesity rates were noted. More recent evidence comes from a recent study by David Barker (the principal advocate of the fetal-origins hypothesis). Barker et al. (2005) examine the trajectory of height, weight, and BMI in a sample of Finns who were admitted to a hospital with heart disease. These individuals were more likely to have BMIs that were below average through the age of about two. After this age, a massive increase in BMI was seen (operating through increases in weight, not height). This accelerated gain in BMI is related to the presence of insulin resistance later in life.

While our understanding of the physiology underlying the fetal-origins hypothesis continues to improve, persuasive tests of this hypothesis in hu-

man populations continue to be evasive but clamor for the attention of empirical economists. For those interested in embracing this challenging enterprise, Harding (2001) provides accessible summaries for the principal role of in utero (fetal) and neonatal nutrition in influencing the adult disease. McMillen, Adam, and Muhlhausler (2005) and McMillen et al. (2006) discuss the considerable evidence from rat and sheep populations that shed light on this hypothesis.

### Relative Deprivation

An alternative explanation for why healthier may not be healthier may lie in the role of the relative deprivation hypothesis. Here the idea is that a person's health and well-being is affected by his or her relative deprivation—the degree to which the health of person  $i$  deteriorates as the income of person  $j$  increases. The causal mechanism that leads to adverse outcomes is the greater (alleged) incidence of depression, stress, and the willingness to engage in risky behaviors. Evaluating this hypothesis is a difficult task, for one needs high quality data on health outcomes, a concrete definition of who constitutes an agent's reference group (is it people in one's neighborhood, coworkers at a firm, or individuals with similar demographics?), and knowledge of what margin of a reference group's prosperity (earnings, income, wealth) affects an agent's well-being. Few data sets provide measures that allow this question to be competently executed, but that does not mean that it does not have merit. Historically, support for explanations grounded in relative deprivation has been the purview of epidemiologists (although the concept is noted in Dusenberry 1949), and I would hazard the guess that economists have a natural antipathy to it, for it wreaks havoc with the applicability of the Pareto principle that guides much of our thinking on welfare calculations. In fairness to economists, it is not the case that the epidemiological literature is particularly convincing; much of the empirical work would not meet economists standards for identification (see Deaton and Lubotsky [2003] for an example of economists challenging the premise that income inequality per se is associated with higher mortality).

Despite these concerns, the fact that Swedes with a PhD have substantially lower mortality than those with a master's degree is unlikely to be explained by conventional explanations such as greater incomes, better health care, or better maternal nutrition (Erikson 2001). A recent paper by Banks et al. (2006) notes that white Americans are in substantially worse health than the English, even after accounting for differences in health insurance. While some interpret this result as providing support for the importance of preventative health (a belief that flies in the face of the scant evidence on the returns to preventative medicine in a developed-country context), it is also possible that even higher incomes and health care expenditures in America improve health, but greater levels of relative deprivation undo some of these



gains. Once again, this is pure speculation on my part. More careful work would evaluate this explanation rigorously. More recent work by economists has begun to do precisely this. Eibner and Evans (2005) provide a summary of much of the literature and push our understanding of the empirical evidence in support of this hypothesis. They use individual death records to establish a compelling association between specific measures of relative deprivation and all-cause death, self-reported health, BMI, and prevalence of engaging in risky behaviors. Interestingly, one's location in the income distribution, that is, "rank," seems not to matter. Luttmer (2005) notes that agents are less happy when the earnings of their neighbors increase; relative consumption may enter the utility function in addition to the absolute level of consumption. If the relative deprivation hypothesis is true, then it has massive implications for the wealthier is healthier hypothesis; a country that is able to realize a 10 percent income shock for every citizen, while preserving one's rank in the distribution, would still see the health of those at the bottom worsen (a person who earned \$40k now earns \$44k, whereas someone who earned \$100k now earns \$110k; the initial difference of \$60k is now \$66k) as relative deprivation increases.

The research program underlying this paper poses questions of absolute importance. Obtaining the correct answer has the power to improve the lives of billions, but it is equally humbling to ponder the lethality of the incorrect answer. Much is known, but much more still needs to be known. We are grateful to Case and Deaton for showing us how to know it.

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