



## Personal Histories and Poverty Traps

PARTHA DASGUPTA

The persistence of poverty in a world that has otherwise and elsewhere enjoyed enormous income growth since World War II remains a puzzle. It wasn't absurd to imagine, as many development economists did in the 1980s, that growth in income in poor economies would trickle down to lift even the poorest out of the mire, but it hasn't happened. Today, the World Bank estimates that more than 1.3 billion people live on less than \$1.25 a day, the Bank's rough-and-ready measure of absolute poverty.

### Motivation

In speaking of an "economy," I cast a wide net. The economy could be a village, a district, a province, a nation, or even the whole world. A household could be poor in a village that is otherwise prosperous, just as a village could be poor even if the country is not, or a country could be languishing with a per capita income of 800 international dollars in a world where more than a billion people enjoy an average income of over 35,000 international dollars. It is frequently argued that in such a situation outside help is needed if the poor are to lift themselves out of poverty. Others question this argument. But all would seem to agree that the form any such help should take can be determined only when the unit to be assisted is identified (a household or an village or entire country) and the pathways by which lives get shaped are well understood.

When development economists talk of poverty, they have absolute poverty in mind (the 1.3 billion mentioned above). But social scientists in Europe and the United States also worry about poverty in their lands. Because context matters, social activists are quick to point out that poverty means different things to different

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Partha Dasgupta is Frank Ramsey Emeritus Professor of Economics at the University of Cambridge and Professional Research fellow at the University of Manchester.

In preparing this keynote lecture, I benefited greatly from conversations and correspondence with Patrick Bateson, Peter Gluckman, and Trevor Robbins.

*Annual World Bank Conference on Development Economics 2011, Global*

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people—that poverty is multidimensional. But if there is something common in a wide-ranging notion, it is not senseless to use one name for it.

The question is whether there *is* something of significance in common. One feature that could be thought to be common is *persistence*. That absolute poverty persists along family lines in rural communities in poor countries is not a controversial claim, even though few longitudinal studies among urban populations prove the claim. Some studies suggest that even in high-income countries poverty is inherited, in that people don't move in and out of poverty periodically (Creedy and Kalb 2006). But I have been unable to find reliable work covering a wide range of places that has determined whether there are lock-in effects, in the sense that the poor on average remain poor and do not enjoy periodic spells of prosperity and the well-off on average remain well-off and do not periodically become poor.

It is the job of theorists to predict what the data would reveal if someone were to look for them. Over the years, I have tried to understand the twin presence of poverty and wealth in poor countries by studying a variety of metabolic and socioecological pathways that would lead to persistent poverty (Dasgupta 1993, 1997, 2000, 2003, 2009). The processes giving rise to those pathways operate at different speeds and at various, often overlapping, spatial scales. And they are highly nonlinear, involving positive feedback. In some cases, the positive feedback is a reflection of fixed costs. For example, the maintenance energy in human metabolic processes is substantial (see below), as are the overhead labor hours in running a household in a world where water cannot be obtained by turning on a tap, where energy is not available at the flick of a switch, and where cooking is a vertically integrated activity. The common feature in all these processes is that the innumerable class of inputs required daily by humans are *complements* of one another. My theme here is the role those complementarities play in dividing populations. The theory I sketch shows why we should expect deep poverty to have a strong tendency to persist across generations.

I am concerned with the absolute poverty experienced by what is commonly referred to as the “bottom billion.” Along the way, I shall connect with recent findings by James Heckman and his colleagues on the complementarities that divide populations even in wealthy societies (e.g., Cunha and Heckman 2007; Cunha, Heckman, and Schennach 2010).

## Framing Poverty

In studying absolute poverty, it is necessary to go beyond income to the access people have to basic amenities. When you do that, you discover that in low-income countries only 68 percent of people have access to clean water and 39 percent to sanitation facilities; the corresponding figures for high-income countries are 99 percent for both (table 1). Such amenities are the universal determinants of human well-being. If instead you were to study figures for the constituents of well-being, you would discover that in low-income countries 28 percent of children under five years old are wasted and 44 percent are stunted. The corresponding figures in even upper-middle-income countries are 4 percent and 14 percent, respectively (table 2).

**TABLE 1. Access to Clean Water and Sanitation, 2006**

	Access	
	Clean water (%)	Sanitation (%)
Low-income countries	67	39
South Asia	87	33
(India)	89	28
Sub-Saharan Africa	58	31
China	88	65
High-income countries	99	99
World	86	60

Source: World Bank 2010, table 2.18.

**TABLE 2. Prevalence of Child Undernutrition, 2000–08**

	Percentage of children under 5		
	Wasted	Stunted	Low birth weight ( $\leq 2.5$ kg)
Low-income countries	28	44	15
South Asia	41	47	27
(India)	44	48	28
Sub-Saharan Africa	25	43	14
China	7	22	2
United States	1.3	4	8
World	23	35	15

Source: World Bank 2010, table 2.20.

These numbers tally with general impressions. The geographic distribution of absolute poverty makes for curious viewing of the world's map, as does the character of that poverty. Globally, the proportion of those who are underweight at birth is 14 percent, which is about the same as the figure for low-income countries. The corresponding figure in the United States, 8 percent, looks disquietingly high (table 2). In numbers, the bulk of the world's poor, when identified in terms of income, are still found in China and South Asia: 47 percent of children in South Asia are stunted and 27 percent are underweight at birth, whereas the corresponding figures in Sub-Saharan Africa are 43 percent and 14 percent, respectively (table 2). And yet, the proportion of people without access to clean water in South Asia is 33 percent, whereas the corresponding figure in Sub-Saharan Africa is 31 percent (table 1). I do not have a satisfactory understanding of some of the puzzling differences in the statistics, but elsewhere I have sought a partial explanation in terms of differences in the socioecological environments in South Asia and Sub-Saharan Africa (Dasgupta 1993, 2000, 2003).

In the world of the poor, fertility is high. The total fertility rate (TFR) in low-income countries is 4.2, compared with a world average of 2.5 (table 3).<sup>1</sup> Being in

**TABLE 3. Maternal Burden 2008**

	Total fertility rate	Maternal mortality (per 100,000)
Low-income countries	4.0	790
Sub-Saharan Africa	5.1	900
South Asia	2.9	500
China	8	45
High-income countries	1.8	10

Source: World Bank 2010, table 2.19.

excess of 2.1, the global TFR is still above the long-term replacement level. In South Asia, the TFR has fallen to 2.9, but in Sub-Saharan Africa it is a high 5.1, with a number of countries experiencing TFRs around 7. To see how great the cost of high TFRs is for women, consider that in Africa a successful birth involves at least two years of pregnancy and breastfeeding. In a country where the TFR is, say 7, about half of a woman's reproductive years would be spent either carrying a child in her womb or breastfeeding it. And we have not allowed for unsuccessful pregnancies. In those circumstances, employment outside the home is not an option.

An absence of reproductive health facilities in poor countries has meant that maternal mortality rates are high. In several poor countries, maternal mortality is the largest single cause of death among women in their reproductive years, and nutritional anemia plays a central role. In Sub-Saharan Africa, one woman dies for every 110 births. In contrast, the maternal mortality rate in Europe is one death per 20,000 births (table 3).

Contemporary data from more than 180 countries indicate that gross domestic product (GDP) per capita is negatively correlated with TFR (Schultz 2006). Much has been made of that in the demographic literature and by the media. The problem is that the relationship is a correlation, nothing more. It is no good using the correlation to recommend that countries should raise incomes if they wish to reduce fertility; the underlying reasons why household incomes are very low could also be the factors that encourage high fertility rates. Income and fertility are both endogenous variables.

### *Description*

Although absolute poverty is usually defined as a state of affairs in which a person has very little income, a large contemporary literature has arrived at the following conclusion: "In the world of the poor, people don't enjoy food security, are stunted and wasted, don't live long, can't read or write, don't have access to easy credit, are unable to save much, aren't empowered, can't insure themselves well against crop failure or household calamity, don't trade with the rest of the world, live in unhealthy surroundings, are poorly governed, and experience high birth rates."<sup>2</sup>

We should add that the poor often reside in fragile ecosystems (Millennium Ecosystem Assessment 2003). Even absolute poverty is multidimensional.

We will call the passage above *description*. Although we can all agree on it, it offers little guidance for action. It doesn't say what is a cause and what is an effect; it doesn't distinguish between proximate and deep causes; it doesn't say what is a variable and what is a parameter in the environment in which the poor reside; and it doesn't say whether variables can be interpreted in samples to move together over time (time series data) or across parameter values at a point in time (cross-sectional data). Above all, the passage doesn't help us identify the pathways that lead to a state of affairs where *description* holds.

### *Analysis*

*Description* suggests that poverty and riches have multiple causes; however, the temptation to seek monocausal explanations for the twin presence of poverty and wealth in our world is so powerful that even development experts haven't always been able to overcome it. But mutual causation has implications for interpreting data. Of course, people's lives are subject to many processes. One category—creating metabolic pathways—works at the level of the individual person. The pathways are based on physiological links connecting (1) undernourishment and a person's vulnerability to infectious diseases, (2) nutritional status and physical and mental development among children, and (3) nutritional status and work capacity among adults.

Another class of processes, operating at a spatially localized level, is site-specific. It involves a combination of ecological and socioeconomic pathways, giving rise to reproductive and environmental externalities. These processes are influenced by the local ecology. The theory based on them acknowledges that the economic options open to a poor community in, say, the African savannahs are different from those available to people in the Gangetic plains of India. Although policies and institutions shape the forces people face, the local ecology also shapes them.

Among ecological and socioeconomic processes, some involve positive feedback among poverty, population growth, and degradation of the local natural resource base. But poverty, population growth, and environmental degradation are not the prior causes of each other; over time, each influences and is influenced by the others. The two broad categories of positive feedback are able to coexist in a society because, except under conditions of extreme nutritional stress, nutritional status doesn't much affect fecundity.<sup>3</sup>

Those who are caught in poverty traps don't necessarily spiral down further. For most of them, there is little room below to fall into—many are already undernourished and susceptible to diseases. Modern nutrition science has shown that relatively low mortality rates can coexist with a high incidence of undernutrition and morbidity. To be sure, many people die from causes traceable directly to their poverty. But large numbers continue to live under nutritional and environmental stress. Moreover, people tend not to accept adverse circumstances lying down. So it is reasonable to assume that they try their best to improve their lot. In some situations, human responses to stress lead to successful outcomes. However, because I am talking about poverty traps, I will identify conditions under which the coping mechanisms people adopt are not enough to lift them out of the mire. Turner and Ali (1996), for

example, have illustrated the possibility by showing that in the face of population pressure in Bangladesh, small landholders have periodically adopted new ways of doing things to intensify agricultural production. The authors have shown, however, that this has resulted in an imperceptible improvement in the standard of living and a worsening of the ownership of land, the latter probably owing to the prevalence of distress sales. These are the kinds of findings that the perspective I explore here anticipated and was designed to meet.

Externalities associated with people's coping strategies can amount to significant differences between private and social returns to various economic activities. Where reproductive behavior is pro-natalist, the private returns of having large numbers of children are high in contrast to the social returns. Similarly, where communities degrade their natural resource base, collective endeavors to maintain the base are unable to withstand the pressure of private malfeasance. And so on.

## Complementarities

In a wide range of cases, the complementarities among the drivers of metabolic and socioecological pathways manifest themselves as fixed costs. When an individual maintains nutritional balance, somewhere in the region of 60 percent to 75 percent of his or her energy intake is spent on maintenance, which is a fixed cost of being alive. The remainder is used for work and discretionary activities.<sup>4</sup> Nutritionists refer to those metabolic fixed costs as "maintenance costs" and sometimes as "resting metabolic rates." About a third of maintenance costs can be traced to the energy expenditure associated with the innumerable brain activities that are synchronized in ways complex adaptive systems generally organize themselves. Those activities are complementary to one another—destroy key steps of a neural pathway, and the brain's overall performance worsens discretely.

Complementarities have been much studied in education. It is common today to say that it's not much use providing classrooms for children if there are no teachers to teach them; or that it's no good providing classrooms and teachers if children come to school hungry and are unable to concentrate; or that it's not much use providing classrooms and teachers and free school meals if the children have been damaged by iodine deficiency during infancy. The return on investment in each of those factors would be low if any of the other factors were in short supply. It's easy to recognize complementarities in the case of classrooms and teachers, because both must be available at the same time. It's less easy to recognize complementarities when they operate sequentially, stretching back to the distant past of a person's life. Complementarities across time give rise to irreversibilities in human development.

Another implication of complementarities is that in the world of the poor, each item in *description* reinforces the others, implying that productivity in labor effort, ideas, capital, land, and natural resources is low and remains low. The lives of the poor are filled with problems every day. On the flip side, the same factors give rise to virtuous feedback, meaning that the rich suffer from no such deprivation. People in the rich world face what today are called "challenges." An implication of the

complementarities and the positive feedback they give rise to is that in the world of the rich, productivity in labor effort, ideas, capital, land, and natural resources is high and continually increasing. Success in meeting each challenge reinforces the prospects of success in meeting additional challenges.

So, the processes that shape our lives harbor multiple stability regimes. Some display progress even as others do not. The presence of multiple stability regimes means that in certain regions of the space of personal characteristics, the processes violate the *principle of horizontal equity*, so that very similar persons diverge cumulatively to face very different life chances. Horizontal inequity is a manifestation of a divided society, and poverty traps are an extreme form of horizontal inequity. (See the appendix for a stylized example.)

Of the many complementary factors that shape our lives, I want to focus on one broad class that illustrates the stranglehold a person's early life can have on his or her ability to function satisfactorily in later years. Those processes range from malnutrition and infectious disease at the very earliest stages of life to the nonacquisition of socioeconomic competencies in early childhood. I work backward from adulthood through childhood to the prenatal stage of life, and from there to the mother's status. I need to retrace people's lives because if, say, you place a malnourished person next to a healthy person, they won't look similar at all. You would then ask, where is the horizontal inequity that supposedly characterizes poverty traps? The point in tracing a person back to the distant past, one that includes the person's mother's status before conception and perhaps even before that, is to show how small shocks could have had marked cumulative effects in the person's subsequent development. That is the sense in which two very similar individuals can face very different life experiences. Complementarities are the cause of societal breaks.

## Adult Health and Productivity

By undernourishment, I mean a combination of inadequate nutritional intake and exposure to a disease environment. Stunting is a reflection of long-term undernourishment, while wasting is a manifestation of short-term undernourishment. Each significantly limits the capacity for physical work, where strength and endurance are needed.

When nutritionists talk of physical work capacity (Collins and Roberts 1988; Ferro-Luzzi 1985; Pollitt and Amante 1984), they mean the maximum power (i.e., maximum work per unit of time) someone is capable of offering. Laboratory methods for estimating maximum power include having a person run on a treadmill and pedal a bicycle ergometer. The most compelling index of a person's physical work capacity is maximal oxygen uptake, usually denoted by the ungainly expression  $O_2$  max. It is the highest rate of oxygen uptake a person is capable of attaining while engaged in physical work at sea level. Maximal oxygen uptake depends on the body's capacity for a linked series of oxygen transfers (diffusion through tissues, circulation of hemoglobin, pulmonary ventilation). It measures cardiorespiratory fitness—the higher the value, the greater the body's capacity to convert energy in the tissues into

work (Åstrand and Rodahl 1986). That capacity depends on the metabolically active tissue mass, which is nearly the same as muscle cell mass (sometimes called the cell residue). Clinical tests suggest that  $O_2$  max per unit of muscle cell mass is approximately constant across well-nourished and marginally undernourished people (Viteri 1971). Even among undernourished persons, the difference is not thought to be great. In one set of studies, more than 80 percent of the difference in  $O_2$  max between mildly and severely malnourished people was traced to differences in their muscle cell mass (Barac-Nieto et al. 1980). It is therefore useful to have a measure of  $O_2$  max per unit of muscle cell mass. A rough approximation of this is provided by the maximal aerobic power, which is  $O_2$  max per unit of body weight. As muscle cell mass and lean body mass are related, we do not lose much by not being particular as to which of the two we identify as the chief determinant of  $O_2$  max.<sup>5</sup>

We are, however, trying to identify the determinants of physical work capacity. A person must enjoy good current nutritional status in order to perform well at strenuous physical work, but that isn't sufficient, because one can be healthy but stunted. Of a pair of people with the same body mass index (BMI), the taller person typically possesses greater muscle cell mass; so the  $O_2$  max is higher. Broadly speaking, taller and heavier (but nonobese) people have greater physical work capacity.  $O_2$  max also depends on the level of habitual physical activity (training, in sports parlance), but I ignore this factor here.<sup>6</sup> Maximal oxygen uptake depends as well on the concentration of hemoglobin in the blood. I also ignore that in what follows.<sup>7</sup>  $O_2$  max is usually expressed in liters per minute (l/min). To obtain a sense of orders of magnitude, note that 6 l/min is about as high as this measure can be, while 2 l/min and below are the numbers observed among chronically malnourished people.

$O_2$  max measures the maximum volume of oxygen the body is capable of transferring per minute. Except for very short bursts, this maximum cannot be reached. The highest level of oxygen transfer a person can sustain over an extended period of eight hours or so is of the order of 35 percent to 40 percent of the  $O_2$  max. There is a relationship between the rate at which a person works (expressed as a fraction of his  $O_2$  max) and his endurance in maintaining that rate of work. The negative-exponential function has been found to be a good approximation, even among undernourished subjects (Åstrand and Rodahl 1986); so, writing the duration of work by  $T$ , we have

$$\text{percentage of } O_2 \text{ max} = \exp(-bT). \quad (1)$$

In equation (1),  $b$  ( $> 0$ ) is a constant. Barac-Nieto and colleagues (1980) have found  $b$  not to be significantly different among people suffering from degrees of malnourishment ranging from mild to severe. The endurance time for 80 percent of  $O_2$  max in their sample was, on average, 97 minutes, with a coefficient of variation of 12 percent. This means that  $b = 0.0023/\text{min}$ . The suggestion is not that this is a human constant; nor is it claimed that the energy cost of a task does not vary with the rate at which it is performed. All the formula means is that, as a very rough approximation, we can distinguish people's capacity for physical activities in terms of their physical work capacity, which I define below.

Let  $P$  denote physical work capacity, and  $V$  the maximal oxygen uptake ( $O_2$  max). From equation (1) we conclude that

$$P = KV\exp(-bT), \quad (2)$$

where  $K$  is a positive constant. The total quantity of work a rested individual is capable of performing is then  $PT = KV T \exp(-bT)$ , which attains its maximum value at  $T = 1/b$ . I conclude that if we are interested in aggregate work, the duration of work should be  $1/b$ . If  $b = 0.0023/\text{min}$ ,  $1/b = 7.2$  hours. I do not know whether, among healthy people in western industrialized countries, a seven-hour day has been arrived at from such a consideration as this.

For strenuous work, those with a low  $O_2$  max need to be close to their physical work capacity. That means their hearts must beat at a fast rate. They are then overtaxed and incapable of maintaining the pace of work for long. This is reflected in equation (2). Consider as an example the well-known series of studies by G. B. Spurr and his colleagues on chronically malnourished adult males and nutritionally normal control subjects among sugarcane cutters, loaders, and agricultural workers in Colombia (Spurr 1990). Nutritional status was assessed on the basis of, among other things, weight-for-height, skinfold thicknesses, total body hemoglobin, and daily creatinine excretion. Roughly speaking, the first three indexes reflect current nutritional status, while the fourth picks up nutritional history to an extent (e.g., taller people have greater muscle cell mass). A stepwise multiple regression analysis with the data revealed that  $O_2$  max is positively related to weight-for-height, total hemoglobin count, and daily creatinine excretion; it is negatively related to skinfold thicknesses. The chronically undernourished subjects ranged from mild to intermediate to severe. Approximate values of their  $O_2$  max were, respectively, 2.1 l/min, 1.7 l/min, and 1.0 l/min. The average  $O_2$  max of the nutritionally normal sugarcane cutters was 2.6 l/min. This is about as clear as any evidence we can hope to find for the thesis that undernourished people suffer from depressed levels of  $O_2$  max.

Consider an activity whose oxygen cost is 0.84 l/min. The nutritionally normal group could sustain it at 0.32 of  $O_2$  max, whereas the remaining three groups would have to sustain it at 40 percent, 50 percent, and 80 percent, respectively, of their  $O_2$  max. At those rates, the nutritionally normal group could work for 8 hours, and the three malnourished groups for 6.5 hours, 5 hours, and 1.5 hours, respectively.

All this bears on physical work capacity and endurance, not physical productivity, although one would expect that they are closely related for unskilled manual work. And they are. For tasks such as sugarcane cutting, loading and unloading, and picking coffee, it is possible to measure physical productivity directly in terms of the amount done. Indeed, payment for such work is often at a piece rate. A wide body of evidence links nutritional status to productivity in these occupations. In their work on Colombian sugarcane cutters and loaders, Spurr and his colleagues (Spurr 1990) found height, weight, and lean body mass (roughly,  $O_2$  max) to be significant determinants of productivity measured by daily tonnage of sugarcane delivered. Measuring productivity ( $W$ ) in units of tons per day,  $O_2$  max (as before,  $V$ ) in liters per

minute, and height ( $H$ ) in cms., and denoting the percentage of body weight in fat by  $F$ , their most-preferred specification was:

$$W = 0.81 V - 0.14 F + 0.03 H - 1.962. \quad (3)$$

In related work, Immink and colleagues (1984) found stature (and thus lean body mass and  $O_2$  max) to be positively correlated with the quantity of coffee beans picked per day, the amount of sugarcane cut and loaded, and the time it took to weed a given area.

I turn now to economic investigations. In their study of a sample of both men and women workers in urban Brazil, Thomas and Strauss (1997) reported that height has a strong positive effect on market wages. That is consistent with the findings of Immink and colleagues (1984) and Spurr (1990), because wages would be expected to bear a positive association with productivity. The relationship between height and productivity is significant because height is not a variable for an adult, so there is less ambiguity about the direction of causality. However, investigators have usually studied the links between current nutritional status and productivity. In a sample of factory workers producing detonator fuses in India, Satyanarayana and colleagues (1977) found weight-for-height to be the significant determinant of productivity. Deolalikar (1988) found strong effects of weight-for-height on both productivity and wages among agricultural workers in South India. The elasticity of farm output with respect to weight-for-height was estimated to be approximately 2, and the elasticity of wages in the region was 0.3–0.7, where the lower value reflects the effect in peak seasons and the higher value in slack seasons, when the tasks are different. In a study of farm workers in Sierra Leone, Strauss (1986) found that energy intake has a positive effect on productivity up to about 5,200 kcal per day. He also found that a worker who consumed 5,200 kcal per day was twice as productive as one who consumed 1,500 kcal per day. Strauss did not report on differences in nutritional status among workers. But if we assume that the workers were in energy balance, we could interpret differences in daily intake as mirroring a combination of differences in nutritional status and the energy expended in the tasks that were accomplished. Thomas and Strauss (1998) found that BMI is positively correlated with wages among Brazilian laborers.

I noted earlier that the energy required for maintaining human life is substantial and that only 25 percent to 40 percent of a person's daily energy intake is spent on discretionary activities—work and leisure. Maintenance costs (resting metabolic rates) are higher for taller people of equal BMI. That is the cost side of healthy persons. On the other hand, they are more productive. It can be shown that because maintenance costs are substantial, markets aren't able to easily eliminate undernutrition, because the undernourished are at a severe disadvantage in their ability to obtain their daily requirements. Since their capacity to work is impaired, they are unable to offer the quality of work necessary to obtain the food they need to improve their nutritional status. Maintenance costs imply that it isn't possible for everyone in an economy that in the aggregate is poor to attain reasonable nutritional status. Thus, over time, undernourishment can be both a cause and a consequence of falling

into a poverty trap (Dasgupta and Ray 1986). Moreover, because undernourishment displays hysteresis (there is a further positive feedback between nutrition and infection), Dasgupta and Ray's analysis implies that we should expect poverty to be dynastic. The theory is that once a household falls into a poverty trap, it is hard for descendants to emerge from it.

### Childhood Experiences

One way a person can economize on energy expenditure is by reducing physical activities. Mild to moderately wasted preschool children under free-living conditions have been observed to spend more time in sedentary and light activities than their healthy counterparts. They have been found to rest longer and to play more often in a horizontal position. A Jamaican study found stunted children in the age group 12–24 months to be significantly less active than their nonstunted counterparts. The energy saved was comparable to the energy cost of growth at that age. At an extreme, when we observe little children in poor countries lying expressionless on roadsides and not even brushing the flies off their faces, we can infer that they are conserving energy. Among preschool children, the first line of defense against low energy intake would appear to be reduced physical activity. Such behavioral adaptation is not learned; humans are wired that way. Little children by the wayside no more consciously husband their precarious hold on energy than bicyclists solve differential equations to maintain balance.

Chavez and Martinez (1979, 1984) reported that among infants from poor households in rural Mexico, differences in activity levels were marked from about six months of age between those who received nutritional supplements and the control group. Supplemented children made more contact with the ground, slept less during the day, spent more time outdoors, and began playing almost six months earlier. The thesis here is that low nutrition intake depresses activity and isolates the infant or child from contact with the environment and from sources of stimuli of vital importance to both cognitive and motor development. It is significant that the control group in the Chavez-Martinez study was only moderately undernourished.

Motor development is the process by which a child acquires basic movement patterns and skills, such as walking, running, jumping, hopping, throwing, kicking, and holding something. In normal circumstances, children develop these fundamental motor patterns by the age of six or seven years. It is through such movement patterns and skills that many childhood experiences, especially learning and interpersonal relationships, are mediated (Grantham-McGregor 1990). During infancy and early childhood, interactions between the mother and child are of critical importance in this development. This is where the cost of anemia and low energy intake on the part of mothers makes itself felt. Since housework and production activities are mandatory, reducing discretionary and child-rearing activities offers the mother a way of maintaining her energy balance. To be sure, societies

differ in the way people other than the mother are involved in a child's upbringing, but in all societies the mother is an important figure in a child's cognitive and motor development.

Long-term malnutrition among infants is especially associated with cognitive development. Dietary deficiencies of iron and iodine in the first two years of life are known to create problems that cannot be reversed by adequate diet in later years (Benton 2010). Under conditions of severe undernourishment (marasmus or marasmic kwashiorkor), both motor development and cognitive development are hampered in infants. Severe malnutrition affects development of the brain, which experiences rapid growth starting at around 10 weeks of pregnancy and continuing in spurts to about three or four years of age (Benton 2010). Fetal iodine deficiency is well known to damage the central nervous system. Equilibrium reactions ("righting reflexes") are functions of the cerebellum and play an important role in the development of motor control. Some of the damage is extremely difficult to reverse and may indeed be irreversible (Kar, Rao, and Chandramouli 2008; Walker 2005). For example, even after six months of nutritional rehabilitation of infants hospitalized for severe malnutrition, Colombo and Lopez (1980) observed no recovery in their motor development (see also Celedon and de Andraca 1979). It is possible that anatomical changes that have been observed are retardation rather than permanent injury, but this is not known with any certainty.<sup>8</sup>

Among schoolchildren, matters are somewhat different. Peer pressure tends to counter the instinct for reducing physical activities, especially among boys. But even for school-aged children, reduced activity is a line of defense. Studies indicate that in school-aged children the low energy expenditure associated with nutritional deficiency can be traced to low body weight; their basal metabolic rates are low. In addition, the development of lean body mass among undernourished children is retarded, which has a detrimental effect on their capacity to work as adults. Marginally malnourished boys don't appear to experience lesser muscle function. Their low capacity for work is due to the fact that their lean body mass is low.

On a wider front, malnutrition and infection have been found to have a pronounced detrimental effect among schoolchildren on such cognitive processes as attention and concentration. Much evidence exists showing that children who suffer from nutritional deficiencies and infections perform poorly in aptitude tests. In extreme cases, nutritional deficiencies affect the central nervous system (Levitsky and Strupp 1995). In less extreme cases, the matter isn't one of brain function; frequent absence and attrition affect learning as well (Bhargava 1994; Pollitt, 1990).

Intertemporal complementarities also exist along nonmetabolic pathways. In a wide-ranging study, Cunha and Heckman (2007) developed a theoretical framework to accommodate the fact that ability gaps between individuals and across socioeconomic groups appear at an early age for both cognitive and noncognitive skills. Studies have shown that enhancements of family environments improve the early development of cognitive as well as socioemotional competencies among children (e.g., perseverance, confidence, motivation, self-control). These competencies are retarded in adolescence if they are not acquired in early childhood (see Cunha, Heckman, and Schennach 2010, and the references there).

## Prenatal Experiences

The nature versus nurture or genes versus environment controversy has been recognized as meaningless (Bateson and Martin 1999; Ehrlich 2000). Many important changes to gene expression occur during the first weeks of pregnancy. The DNA experiences epigenetic changes in the first week in particular. These changes determine the pattern of gene expression that not only controls the next stage of the fetus's development but also many of the person's attributes throughout life. The mother's long-term nutritional status determines how she mobilizes nutrients to support fetal development. So the experiences that shape an adult start before birth and perhaps even before the mother's birth. In pioneering work, David Barker and colleagues (Barker et al. 1989a, 1989b, 2002) found that rates of ischemic heart disease in England and Wales were more closely related to mortality conditions that prevailed when heart patients were born than to recent conditions. The hypothesis is that maternal conditions in the prenatal period have an important impact on the emergence of later cardiovascular disease.

What are the signatures of prenatal experiences? Although it would be astonishing if a single scalar index at birth could summarize prenatal experiences, a substantial body of work has shown that birth weight is a reasonable indicator of prenatal conditions.<sup>9</sup> In addition, studies that were based not only on birth weight confirm that food deprivation in the womb affects adult metabolism and cardiovascular health; in fact, it has been found to have adverse effects even on age-associated declines in cognitive functions.<sup>10</sup>

What mechanisms would determine the association between prenatal conditions and the cardiovascular-metabolic cluster of chronic diseases? Barker and colleagues (2002) suggested that insufficient energy during fetal development triggers biased apportioning of the available energy to brain development. Maternal stress may be communicated to the fetus via alterations of placental blood flow and changes in energy available for fetal growth, compromising the development of other organs, including kidneys, pancreas, and adipose tissue. For example, small babies have fewer nephrons in their kidneys, fewer beta cells in their pancreases, and lower fat cell numbers than their peers who are larger at birth. However, many of the deleterious adult outcomes of small birth size appear to be related to altered insulin sensitivity and activity of the hypothalamic-pituitary-adrenal (HPA) axis. Both these systems are important modulators of energy metabolism. A good deal of attention has been paid in recent years to pathways that involve the extent to which insulin sensitivity and the reactivity of the HPA axis are established in utero; the potential for maternal nutritional status to affect those aspects of metabolic physiology; and the cellular mechanisms by which the effects are mediated (Ellison 2010). Between 24 and 42 weeks of gestation, the developing brain is particularly vulnerable to nutritional deficiency, owing to the rapid development of vital neurological processes, including synapse formation. And yet, at that time, the developing brain also demonstrates its greatest degree of plasticity (Georgieff 2007).

What accounts for that aspect of fetal development? One possibility is that beginning as early as the first weeks after conception and continuing into early infancy, the

fetus reads key features of its environment and prepares to adapt to an external world that can vary dramatically in its level of safety, self-sufficiency, and danger. When early experiences prepare a developing child for conditions involving high levels of stress or instability, the body's systems retain that initial programming and put the stress response system on a quick response and high alert status. Under those circumstances, the price of short-term survival could be longer-term health. This is called the "fetal programming hypothesis," and it has generated much interest in phenotypic plasticity and the mechanisms that govern it.<sup>11</sup>

Gluckman, Hanson, and Spenser (2005) have proposed that the apparent paradox of adaptive developmental processes yielding pathological results can be resolved if (1) the adaptive processes are aimed at adjusting the organism's physiology to a predicted postnatal environment, and (2) there is a mismatch between the predicted and actual postnatal environment. The hypothesis is that the fetus can sense the environment into which it can expect to be born from maternal signals. The availability of food is one such signal; maternal stress reflected in hormonal changes is another; and fluid deprivation and oxygen availability are others. The authors' point is that there are selection advantages in trying to match the physiology we develop in our plastic phase of development to the environment we may inhabit. This can lead to paradoxical responses, as when a fetus that is "expecting" a strongly constrained environment enters a world where food is abundant. Obesity and the onset of type-2 diabetes are familiar phenomena today. In a wide-ranging work, Gluckman and Hanson (2006) call the maladaptive response *mismatch*, a kind of programming that explains why diets rich in protein and calories have been known to have adverse effects among children of low birth weight. The authors suggest that conditions in utero may reflect not merely maternal conditions at the time but also the mother's sensitivity to those conditions. For example, the energy available to the fetus is affected by maternal undernutrition and the sensitivity of the mother's own physiology to variation in the energy available to her. Maternal sensitivity to energy availability, in turn, may be partly a consequence of the conditions *she* faced in utero, which in turn would depend on *her* mother's sensitivity to energy availability, and so on.<sup>12</sup>

Much medical research on prenatal development has been conducted on subjects in high-income countries. There, the issue isn't usually a question of maternal energy deficiency. Stress, anxiety, or depression during pregnancy have been linked to lower birth weight and subsequently even to psychopathology (Fumagalli et al. 2007). Maternal stress has been found to be associated with increased basal HPA-axis activity in the offspring at different ages, including six months, five years, and ten years. Increased activation of the HPA axis causes the adrenal gland to produce glucocorticoids. These are important for normal brain maturation, but elevated levels impair brain development and functioning. Some of these conditions are reversible, and the effects of prenatal stress are often moderated by the quality of postnatal care; others are not (Cottrell and Seckl 2009). Chronic exposure to stress hormones—whether it occurs during the prenatal period or in infancy, childhood, adolescence, adulthood, or old age—has an impact on brain structures involved in cognition and mental health (Evans and Schamberg 2009; Lupien et al. 2009;

Rice et al. 2010). The fact that there are many pathways to low birth weight perhaps explains why the incidence of low birth weight remains high in rich countries (table 2).

## Morals

What morals do we take away from this account? The following seem to be worth commending:

1. The high maintenance costs of good physical and emotional health underlie the existence of poverty traps.
2. From (1) it follows that in low-income countries, absolute poverty is both a cause and a consequence of unequal distributions of assets.
3. High maintenance costs are manifestations of complementarities among the inputs that humans need for survival. Maintenance costs are higher among people who are fortunate enough to flourish.
4. The acquisition of human capital is continuous and cumulative. Formally speaking, investments in human capital are complementary over time. Complementarities across time give rise to irreversibilities in human development. Nutritional insults at the earliest stages of life have a marked effect on a person's subsequent ability to acquire human capital. If governments and international organizations believe human capital formation is important, they should treat all periods of a person's life with respect.
5. From (4) we can conclude that personal history has a long reach, affecting not only the person in question but also any descendants.

These observations bring us back to the point with which I began: that a person's current productivity is a function of the person's nutritional and morbidity history. A reasonable index of a person's productivity over time would be the present-discounted sum of the person's output of work. The reckoning should start from the earliest stages of the person's life. The computation is no doubt very, very hard, but there is no escaping it.

Much international attention has been given to saving lives in times of collective crisis in poor countries. This is as it should be. International agencies have also paid attention to keeping children alive in normal times through public health measures, such as family planning counseling, immunization, and oral rehydration. This too is as it should be. The fact that many poor countries fail to do either does not mean that the problems are especially hard to solve. In fact, they are among the easier social problems—they can be addressed without any major modifications to the prevailing resource allocation mechanism. The much harder problem for intellectual design, political commitment, and administration is to ensure that those who are conceived have a chance for a healthy life. This is a problem whose solution brings no easily visible benefit. But the stunting of both cognitive and motor capacity is a prime hidden cost of energy deficiency and anemia among children and, one step removed, among mothers. It affects learning and skill formation, and thus future productivity. The price is paid in later years, but it is paid.

## Appendix: Poverty Traps and Horizontal Inequity

Poverty traps are a sharper notion than horizontal inequity. To illustrate the differences at the nutrition-productivity interface, we consider a stylized example.

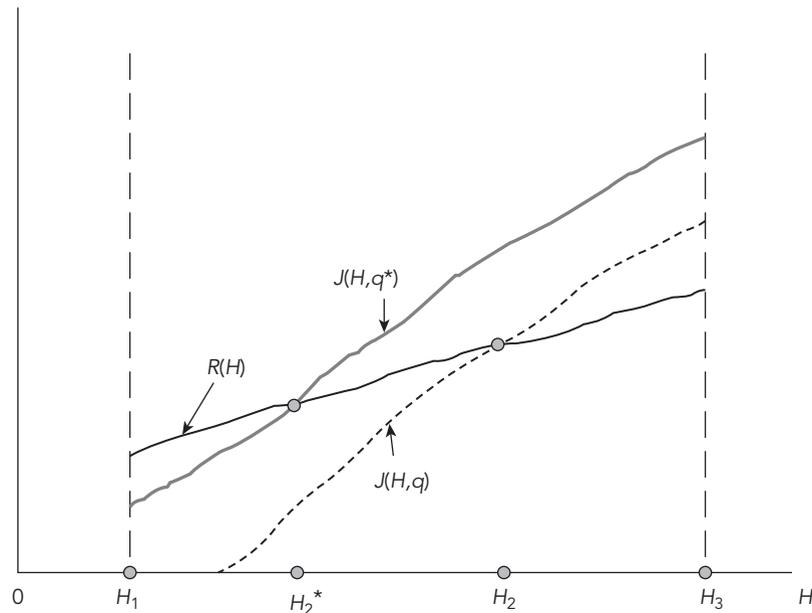
Denote time by  $t \geq 0$ . The present is  $t = 0$ . Consider someone whose nutritional status at  $t$  is a scalar,  $H(t)$ . Let  $J(H, q)$  be a person's income, where  $q$  is a (scalar) parameter reflecting the person's socioecological environment. We suppose that  $dJ(H, q)/dH > 0$  and that  $J$  shifts vertically upward with increases in  $q$ . Let  $R(H)$  denote the person's nutrition requirement (expressed in units of income). We assume  $dR(H)/dH > 0$ , to reflect the fact that a person's resting metabolic rate increases with body size. A person's health, when viewed as a stock, is assumed to obey the deterministic differential equation,

$$dH(t)/dt = J(H(t), q) - R(H(t)), \quad H_3 > H(t) > H_1,$$

and if, for any  $t^*$ ,  $H(t^*) = H_1$  (resp.  $H_3$ ), then  $H(t) = H_1$  (resp.  $H_3$ ) for all  $t \geq t^*$ .  $H_1$  and  $H_3$  are absorbing states.

Because the resting metabolic rate is positive,  $R(H_1) > 0$  and  $J(H, q) = 0$  in the neighborhood of  $H_1$ . In figure 1,  $J(H, q)$  and  $R(H)$  have been so drawn that they intersect once, at  $H_2$ . The system defined by (A1) has three equilibria:  $H_1$ ,  $H_2$ , and  $H_3$ . Among them,  $H_2$  is unstable, whereas  $H_1$  and  $H_3$  are stable. Someone whose

FIGURE 1. Nutrition-Based Poverty Trap



Source: Derived by author.

initial health status  $H(0)$  is even slightly in excess of  $H_2$  would enjoy improvement in health status, while someone for whom  $H(0)$  is even slightly less than  $H_2$  would be trapped in a deteriorating situation. It follows that there could be people in the neighborhood of  $H(0)$  who are similar but who face widely differing fortunes. The example exposes the limitations of studies that view the quality of life at a single point in time. Similar people wouldn't remain similar if they were to experience widely different life histories. The principle of horizontal equity could not be applied to them at later times. This is one reason why the principle, as traditionally formulated, has little ethical bite. When assessing an economy, the lives of its citizens should be viewed as a whole, not studied at a frozen moment in time.

What might  $q$  reflect? It could reflect factors that are exogenous to the economy, such as rainfall, as well as factors that are exogenous to the person but endogenous in the economy, such as the effectiveness of property rights, the extent to which government and communities have in place effective support systems, the degree to which markets are open to the person, and the person's nonlabor assets, including education. And we can add the extent to which the person has reasons to trust others and to which others trust the person.

If public policies improve  $q$ ,  $J$  would move up vertically. If the schedule were to rise sufficiently high ( $q = q^*$ ), it would not intersect  $R(H)$ , and  $H_3$  would become the sole (stable) equilibrium point of the system defined in (A1). Welfare support (be it communitarian or state-based) and income guarantees would be another set of mechanisms by which  $J(H, q)$  could be lifted. These are among the various pathways by which nutrition-based poverty traps have been eliminated in a number of countries.

## Notes

1. TFR is the number of live births a woman would expect to give if she were to live through her childbearing years and to bear children at each age in accordance with the prevailing age-specific fertility rate. If TFR were 2.1 or thereabouts, population would stabilize in the long run.
2. See, for example, Sen (1999); Narayan et al. (2000); Banerjee, Benabou, and Mookherjee (2006); Banerjee and Duflo (2007); and—since its inception in 1990—every annual edition of the United Nations *Human Development Report*.
3. For a more detailed account, see Dasgupta (2000).
4. See WHO (1985) for estimates of mean protein-energy requirements among the genders, occupations, and age groups.
5. The mass of muscle tissue and muscle constitutes about 40 percent of body weight and 50 percent of lean body mass.
6. Unskilled laborers in poor countries are often slight and weak, but they are never out of shape; sedentary workers are often out of shape.
7. A classic article on iron-deficiency anemia and its effect on physical work capacity is Basta et al. (1979).
8. The study of the effect of malnutrition on mental development is fraught with difficulties of interpretation. On this, see the chapter by S. M. Grantham-McGregor in Waterlow

- (1992). For wide-ranging reviews of the consequences of chronic energy deficiency, see Schurch and Scrimshaw (1987).
9. For more on the fact that birth weight does not capture many salient aspects of the fetal experience and can in certain ways even mislead, see the summary in Schulz (2010). Ellison (2010) provides an excellent overview of the literature.
  10. Bateson and Martin (1999) offer an excellent general account. For studies of the consequences for fetal development of the Dutch Hunger Winter (1944-1945), see Roseboom, de Rooij, and Painter (2006) and Rooij et al. (2010).
  11. See the excellent review by Shonkoff, Boyce, and McEwen (2009). They note that the origins of many adult diseases can be found adversities in the early years of life, which establish “biological memories” that weaken physiological systems and produce latent vulnerabilities to problems that emerge well into later adult years.
  12. The question whether fetal programming is adaptive remains controversial. Some see these effects as disruptions of optimal development with permanent consequences—developmental pathologies that may be more frequent in novel evolutionary environments (Barker 1994; Barker et al. 2002).

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