

Personal Histories and Poverty Traps*

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Contents

1 Motivation

2 Framing Poverty

 2.1 Description

 2.2 Analysis

3 Role of Complementarities

4 Adult Health and Productivity

5 Childhood Experiences

6 Prenatal Experiences

7 Morals

Appendix

References

Tables 1-3

Figure

The persistence of poverty in a world that has otherwise and elsewhere enjoyed enormous income growth since the War 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. Even today over 1.3 billion people are estimated by the World Bank to live under \$1.25 a day, the Bank's rough and ready measure of absolute poverty.

1 Motivation

In speaking of an economy, I cast a wide net here. 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 over 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 the argument. No matter, all would seem to agree that the form any such help should take is something that can be determined only when the unit to be assisted is identified (a household or village or an entire country) and the pathways by which lives get shaped is well understood.

When development economists talk of poverty, they have absolute poverty in mind (the 1.3 billion just mentioned). But social scientists in Europe and the US also worry about poverty in their lands. As the context matters, social activists are quick to point out that poverty means different things to people, that poverty is multi-dimensional. 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 there are few longitudinal studies among urban populations that would clinch the claim. There are studies suggesting that poverty even in high income countries 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 will 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 socio-ecological pathways that would harbour persistent poverty (Dasgupta, 1993, 1997, 2000, 2003, 2009). The processes giving rise to those pathways operate at different speed and at various, often overlapping, spatial scales. And they are highly non-linear, involving positive feedback. In some cases the positive feedback is a

reflection of fixed costs. For example, the maintenance energy in human metabolic processes are substantial (see below), as are the overhead labour hours in running a household in a world where water cannot be obtained by turning 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 those processes is that an innumerable class of inputs required daily by we humans are *complements* of one another. The role those complementarities play in dividing populations is the theme of this paper. The theory I sketch here shows why we should expect deep poverty to have a strong tendency to persist across generations.

I am concerned with the (absolute) poverty that is experienced by what's today 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 go to divide populations even in wealthy societies (e.g., Cunha and Heckman, 2007; Cunha, Heckman, and Schennach, 2010).

2 Framing Poverty

In studying absolute poverty, it has proved 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 per cent of people have access to clean water and 39 per cent to sanitation facilities; the corresponding figures for high income countries are nearly 100 per cent 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 per cent of children under-5 are wasted and 44 per cent are stunted. The corresponding figures in even upper middle-income countries are 4 per cent and 14 per cent, respectively (Table 2).

Those numbers tally with one's general impression. 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 under-weight at birth is 14 per cent, which is about the same as the figure for low-income countries. Compared to that the corresponding figure in the United States, namely 8 per cent, looks disquietingly high (Table 2), a point to which I return below (Section 6). In numbers, the bulk of the world's poor, when identified in terms of income, are still to be found in China and South Asia. 47 per cent of children in South Asia are stunted and 27 per cent are underweight at birth, whereas the corresponding figures in sub-Saharan Africa are 43 per cent and 14 per cent, respectively (Table 2). And yet, the proportion of people without access to clean water in South Asia is 33 per cent, whereas the corresponding figure in sub-Saharan Africa is 42 per cent (Table 1). I don't 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 socio-ecological environments in South Asia and sub-Saharan Africa (Dasgupta, 1993, 2000, 2003).

The world of the poor is also one where fertility is high. The total fertility rate (TFR) in

low income countries is 4.2 as against a world average of 2.5 (Table 3).¹ Being in excess of 2.1, the global TFR is still above the long-term replacement level. In South Asia TFR has fallen to 2.9, but in sub-Saharan Africa it is even now a high 5.1, with a number of countries experiencing TFRs round 7. To see how great the cost of high TFRs are for women, consider that in Africa a successful birth involves at least two years of pregnancy and breast-feeding. 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 breast-feeding 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, nutritional anaemia playing a central role in this. In sub-Saharan Africa one woman dies for every 110 births. In contrast the maternal mortality rate in Europe today is 1 per 20,000 (Table 3).

Contemporary data from over 180 countries indicate that 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's 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 ones that encourage high fertility rates. Income and fertility are both "endogenous" variables.

2.1 Description

Although absolute poverty is usually defined to be a state of affairs where a person has very little income, a large contemporary literature has arrived at the following thought:²

"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, experience high birth rates."

And to that we should add that the poor often reside in fragile ecosystems (MEA, 2003). Even absolute poverty is multi-dimensional.

We will call the above passage *Description*. Although no one would have ever doubted *Description*, it offers little guidance for action. It doesn't say what is a cause and what is an

¹ TFR is the number of live births a woman would expect to give if she were to live through her child-bearing years and to bear children at each age in accordance with the prevailing age-specific fertility rates. If TFR were 2.1 or thereabouts, in the long run population would stabilize.

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*.

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 those that are 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 to identify the pathways that lead to a state of affairs where *Description* holds.

2.2 Analysis

Description suggests that poverty and riches have multiple causes. Nevertheless the temptation to seek mono-causal 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. They are based on physiological links connecting (i) undernourishment and a person's vulnerability to infectious diseases, (ii) nutritional status and physical and mental development among children, and (iii) 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 socio-economic pathways, giving rise to reproductive and environmental externalities. Those 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 plain of India. Although policies and institutions shape the forces people face, the local ecology shapes them too.

Among ecological and socio-economic processes, some involve positive feedback between poverty, population growth, and degradation of the local natural-resource base. But neither poverty, nor population growth, nor environmental degradation is the prior cause of the others: over time each influences, and is in turn influenced by, the others. The two broad categories of positive feedback are able to co-exist in a society because, or so it has been found, except under conditions of extreme nutritional stress, nutritional status doesn't much affect fecundity.³

Those who are caught in poverty traps don't necessarily spiral down farther. 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 co-exist with a high incidence of undernutrition and morbidity. To be sure, many die owing to 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 own lot. There are situations where human

³ For a more detailed account, see Dasgupta (2000).

responses to stress lead to successful outcomes. However, as this paper is about poverty traps, my idea is to 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 land-holders have periodically adopted new ways of doing things so as to intensify agricultural production. The authors have shown, however, that this has resulted in only 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 of land. These are the kind 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 behaviour is pro-natalist, the private returns to having large numbers of children are high in contrast to the social returns. Similarly, where communities degrade their natural-resource base, the collective endeavours to maintain the base are unable to withstand the pressure of private malfeasance. And so on.

3 Complementarities

There is a wide range of cases where the complementarities among the drivers of metabolic and socio-ecological pathways manifest themselves as fixed costs. When an individual maintains nutritional balance, somewhere in the region of 60-75 per cent of her energy intake is spent on maintenance, which is a fixed cost of being alive. The remainder is used for work and discretionary activities.⁴ 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 a commonplace today to say that it's not much use providing class rooms to children if there are no teachers to teach them, or that it's no good providing class rooms and teachers if children come to school hungry (they are then unable to concentrate), or that it's not much use providing class rooms 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 or more of the other factors were in short supply. It's easy to recognise complementarities in the case of class rooms and school teachers because both must be available at the same time. It's less easy to recognise complementarities when they operate sequentially, stretching back to the distant past of a person's

⁴ See WHO (1985) for estimates of mean protein-energy requirements among the genders, occupations, and age groups.

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 the productivity of labour effort, ideas, capital, and of land and natural resources are all low and remain low. The lives of the poor are filled with *problems* each day. The flip side is where the 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 the productivity of labour effort, ideas, capital, and of land and natural resources are all high and continually increasing. Success in meeting each challenge reinforces the prospects of success in meeting further challenges.

So, the processes that shape our lives harbour 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*: very similar persons diverge cumulatively to face very different life chances. Horizontal inequity is a manifestation of broken symmetry; and poverty traps are an extreme form of horizontal inequity. The Appendix provides a formal illustration by means of a stylized example.

Of the many complementary factors that shape our lives, I want to focus on one broad class that displays the stranglehold a person's early life can have on their ability to function satisfactorily in later years. Those processes range from malnutrition and infectious disease at the very earliest stages of life to the non-acquisition of socio-economic competencies in early childhood. In order to do that, I work backward, from adulthood through childhood to the pre-natal stage of life, and thence to a person's mother's status. I need to re-trace 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 his distant past, one that includes his mother's status before he was conceived, and perhaps even before that, is to show how small shocks could have had marked cumulative effects in his subsequent development. That is the sense in which two very similar individuals can face very different life experiences. Complementarities are the cause of broken symmetry.

4 Adult Health and Productivity

By undernourishment I mean a combination of inadequate nutrition 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 (Pollitt and Amante, 1984; Ferro-Luzzi, 1985; Collins and Roberts, 1988) they mean the maximum power (i.e. maximum work per unit of time) someone is capable of offering. Laboratory methods for estimating that in a person

include getting him to run a treadmill and pedal a bicycle ergometer. The most compelling index of a person's physical work capacity is his maximal oxygen uptake, usually denoted by the ungainly expression, $\dot{V}O_2$ max. It is the highest rate of oxygen uptake a person is capable of attaining while engaged in physical work at sea level. The reason maximal oxygen uptake provides us with the measure we need is that it is dependent on the body's capacity for a linked series of oxygen transfers (diffusion through tissues, circulation of haemoglobin, pulmonary ventilation). $\dot{V}O_2$ max measures (cardio-respiratory) fitness; the higher is its value, the greater is the capacity of the body to convert energy in the tissues into work (Åstrand and Rodahl, 1986). That capacity depends on the (metabolically) active tissue mass, which is very nearly the same as muscle cell mass. The latter is on occasion called the cell residue. Clinical tests suggest that $\dot{V}O_2$ 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, over 80 per cent of the difference in $\dot{V}O_2$ max between mildly and severely malnourished people were traced to differences in their muscle cell mass (Barac-Nieto *et al.*, 1980). It is therefore useful to have a measure of $\dot{V}O_2$ max per unit of muscle cell mass. A rough approximation to this is provided by the maximal aerobic power, which is $\dot{V}O_2$ max per unit body weight. As muscle cell mass and lean body mass are related, we do not lose much in not being particular as to which of the two we identify as the chief determinant of $\dot{V}O_2$ max.⁵

We are however trying to identify the determinants of physical work capacity. It is necessary for a person to enjoy good current nutritional status if he is to perform well at strenuous physical work; but it 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 his $\dot{V}O_2$ max is higher. Broadly speaking, taller and heavier (but non-obese) people have greater physical work capacity. $\dot{V}O_2$ max also depends on the level of habitual physical activity ("training", in sports parlance), but I ignore the latter's role here.⁶ Maximal oxygen uptake depends as well on the concentration of haemoglobin in the blood. I ignore that too in what follows.⁷ $\dot{V}O_2$ max is usually expressed in litres per minute (l/min). To obtain a sense of orders of magnitude, we note that the figure 6 l/min is about as high as can be, while 2 l/min and a bit below are the sorts of figure observed among chronically malnourished people.

⁵ The mass of muscle tissue and muscle constitutes about 40 per cent of body weight, and 50 per cent of the lean body mass.

⁶ Unskilled labourers in poor countries may often be slight and weak, but they are never out of shape; it is sedentary workers who often are.

⁷ A classic article on iron-deficiency anaemia and its effect on physical work capacity is Basta *et al.* (1979).

$\dot{V}O_2$ max measures the maximum volume of oxygen the body is capable of transferring per minute. Excepting for very short bursts this maximum cannot be reached. It transpires that the highest level of oxygen transfer a person is capable of sustaining over an extended period of 8 hours or so is of the order of 35-40 per cent of his $\dot{V}O_2$ max. More generally, there is a relationship between the rate at which a person works (expressed as a fraction of his $\dot{V}O_2$ 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

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

In equation (1), b (> 0) is a constant. Barac-Nieto *et al.* (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 per cent of $\dot{V}O_2$ max in their sample was on average 97 minutes, with a coefficient of variation of 12 per cent. This means $b = 0.0023/\text{min}$. 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 that the figure means is that, as a very rough approximation, we may 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 ($\dot{V}O_2$ 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 $PT = KV\exp(-bT)$, which attains its maximum value at $T = 1/b$. I conclude that if it is aggregate work we are interested in, the duration of work should be $1/b$. If $b = 0.0023/\text{min}$, we have $1/b = 7.2$ hours. I do not know if among healthy people in western industrialized countries, a 7-hour day has been arrived at from such a consideration as this.

For strenuous work, those with a low $\dot{V}O_2$ 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 on nutritionally-normal control subjects among sugarcane cutters, loaders, and agricultural workers from Colombia (Spurr, 1990). Nutritional status was assessed on the basis of, among other things, weight-for-height, skinfold-thicknesses, total body haemoglobin, and daily creatinine excretion. While, roughly speaking, the first three indices reflect current nutritional status, the fourth picks up nutritional history to an extent (*cet. par.* taller people have greater muscle cell mass). A step-wise multiple regression analysis with the data revealed that $\dot{V}O_2$ max is positively related to weight-for-height, total-haemoglobin count, and daily creatinine excretion; and it is negatively related to skinfold-thicknesses. The chronically

undernourished subjects ranged from "mild" to "intermediate" to "severe". Values of their $\dot{V}O_2$ max were in turn, approximately 2.1 l/min, 1.7 l/min, and 1.0 l/min. The average $\dot{V}O_2$ max of the nutritionally normal sugarcane cutters was 2.6 l/min. As evidence in this field go, this is about as clear as any we should hope to find for the thesis that undernourished people suffer from depressed levels of $\dot{V}O_2$ max.

Consider an activity whose oxygen cost is 0.84 l/min. The nutritionally normal group could sustain it at 0.32 of $\dot{V}O_2$ max, whereas the remaining three groups would have to sustain it at 40 per cent, 50 per cent and 80 per cent, respectively, of their $\dot{V}O_2$ max. At those paces 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. One would expect though 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. There is now a wide body of evidence linking 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, $\dot{V}O_2$ max) to be significant determinants of productivity, as measured by daily tonnage of sugarcane delivered. Measuring productivity (W) in units of tons-per-day, $\dot{V}O_2$ max (as before, V) in litres per minute, height (H) in cms., and denoting by F the percentage of body weight in fat, their most-preferred specification was:

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

In related work, Immink *et al.* (1984) found stature (and thus lean body mass and $\dot{V}O_2$ max) to be positively correlated with the quantity of coffee beans picked per day, the amount of sugarcane cut and loaded, and the time taken 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 (1996) reported that height has a strong, positive effect on market wages. That is consistent with the findings of Immink *et al.* (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, for an adult, height is not a variable; so there is less ambiguity about the direction of causality. However, it has been more usual for investigators to study the links between current nutritional status and productivity. In a sample of factory workers (producing detonator fuses) in India, Satyanarayana *et al.* (1977) found weight-for-height to be the significant determinant of productivity. Strong effects of weight-for-height on both productivity and wages have been found among agricultural workers in South India by Deolalikar (1988). 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 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 found to be 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 were to assume that the workers were in energy balance, we could interpret differences in daily intakes as mirroring a combination of differences in nutritional status and the energy expended in the tasks that were accomplished. Strauss (1998) also found that the body mass index (BMI) is positively correlated with wages among Brazilian labourers.

We noted earlier that the energy required for maintaining human life is substantial, in that only 25-40 per cent of a person's daily energy intake is spent on "discretionary" activities (work and leisure). Maintenance costs, or what are formally called resting metabolic rates, are higher for taller people of equal BMI. That's 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 eliminate undernutrition easily. The reason is that 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 needed to obtain the food they require if they are 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. So, over time undernourishment can be both a cause and consequence of someone falling into a poverty trap (Dasgupta and Ray, 1986). Moreover, because undernourishment displays hysteresis (there is a further positive feedback between nutrition and infection), their analysis implied that we should expect poverty to be dynastic. The conjecture is that once a household falls into a poverty trap, it proves especially hard for descendants to emerge out of it. Why?

5 Childhood Experiences

One way a person can economize on her energy expenditure is by reducing physical activities. Mild-to-moderately wasted pre-school 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 non-stunted 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 refraining from brushing the flies off their faces, we should infer that it is energy they are conserving. Among pre-school children the first line of defence against low energy intake would appear to be reduced physical activity. Such behavioural modes of adaptation are not deliberately arrived at, we are "wired" to so adapt. Little children by the wayside no more consciously husband their precarious hold on energy than bicyclists solve

differential equations in order 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 6 months of age between those who received nutritional supplements and the control group. Supplemented children made greater contact with the "floor", slept less during the day, spent greater time outdoors, and began playing almost 6 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 her grip. In normal circumstances children develop these fundamental motor patterns by the age of 6 or 7 years. It is through such movement patterns and skills that many childhood experiences, especially learning and interpersonal relationships, are mediated. During infancy and early childhood, interactions between the mother and child are of critical importance in this development. This is where the cost of anaemia and low energy intake on the part of mothers makes itself felt. Since housework and production activity 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 the mother is an important figure in a child's cognitive and motor development in all societies.

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 and cognitive development are hampered in infants. Severe malnutrition affects development of the brain, which experiences rapid growth starting round 10 weeks of pregnancy and continuing in spurts to about 3 to 4 years of age (Benton, 2010). Fetal iodine deficiency is well known to damage the central nervous system. Equilibrium reactions (otherwise called "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 (Walker, 2005; Kar, Rao, and Chandramouli, 2008). For example, even after 6 months of nutritional rehabilitation of a sample 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, of course, possible that even such anatomical changes as have been observed are

retardation rather than permanent injury. But this is not known with any certainty.⁸

Among school children matters are somewhat different. Peer pressure tends to counter the instinct for reducing physical activities. This is likely to be so especially among boys. But even for school-aged children reduced activity is a line of defence. 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. Relatedly, the development of lean body mass among undernourished children is retarded. This has a detrimental effect on their capacity to work when 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 school children on such cognitive processes as attention and concentration. There is much evidence that children who suffer from nutritional deficiencies and infections perform badly in aptitude tests. In extreme cases nutritional deficiencies affect the central nervous system (Levitsky and Strupp, 1995). In less-than- extreme cases the matter isn't one of brain function; frequent absence and attrition affect learning as well (Pollitt, 1990; Bhargava, 1995).

Intertemporal complementarities exist also along non-metabolic pathways. In a wide-ranging study, Cunha and Heckman (2007) have 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 non-cognitive skills. Studies have shown that enhancements of family environments improve the early development of cognitive as well as socio-emotional competencies among children (perseverance, confidence, motivation, self control). Those competencies are retarded in adolescence if they are not acquired in early childhood (see Cunha, Heckman, and Schennach, 2010; and the references there).

6 Prenatal Experiences

The "nature vs. nurture" or "genes vs. environment" controversy has now been recognised to be meaningless (Bateson and Martin, 1999; Gluckman and Hanson, 2008). Many important changes to gene *expression* occur during the first weeks of pregnancy. The DNA experience epigenetic changes in the first week in particular. They determine the pattern of gene expression that not only controls the next stage of the fetus' development but also many of the person's attributes throughout life. The mother's long term nutritional status determines how nutrients are mobilized by her to support fetal development. So, the experiences that shape an adult start even before his birth and perhaps even before his mother's birth. In pioneering work David Barker and

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).

his colleagues (Barker *et al.*, 1989a,b, 2002) found that rates of ischemic heart diseases 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 cardio-vascular diseases.

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.⁹ In addition, studies that were based not only on birth-weight confirm that food-deprivation in the womb affects adult metabolism and cardiovascular health; it has been found even to have adverse effects on age-associated declines in cognitive functions.¹⁰

What mechanisms would determine the association between prenatal conditions and the cardiovascular-metabolic cluster of chronic diseases? Barker *et al* (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. The development of other organs, including kidneys, pancreas, and adipose tissue are thereby compromised. For example, small babies have fewer nephrons in their kidneys, fewer beta cells in their pancreases, and lower fat cell number than their peer 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 given in recent years to pathways that involve the extent to which insulin sensitivity and the reactivity of the HPA-axis is 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

⁹ That birth-weight does not capture many salient aspects of fetal experiences and can in certain ways even mislead, see the summary in Schulz (2010). Ellison (2010) gives a fine overview of the literature.

¹⁰ Bateson and Martin (1999) offers an excellent general account. For studies of the consequences for fetal development during the Dutch Hunger Winter (Winter 1944-45), see Roseboom, de Rooij, and Painter (2006) and Rooij *et al.* (2010).

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.¹¹

Gluckman, Hanson, and Spenser (2005) have proposed that the apparent paradox of adaptive developmental processes yielding pathological results can be resolved if (a) the adaptive processes are aimed at adjusting the organism's physiology to a predicted postnatal environment, and (b) there is a mismatch between the predicted and actual post-natal 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 expect to inhabit. This can lead to paradoxical responses as when a fetus "expecting" a strongly constrained environment enters a world where food is abundant. Obesity, 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 programming that explains why diets rich in protein and calorie have been known to have adverse effects among children of low birth-weight. The authors also suggest that conditions *in utero* may not merely reflect maternal conditions at the time, but also her 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.¹²

Much medical research on prenatal development has been conducted on subjects in high income countries. There the matter isn't usually of maternal energy deficiency. During pregnancy maternal stress, anxiety, or depression have been linked with lower birth-weight, and

¹¹ See the excellent review by Shonkoff, Boyce, and McEwen (2009). They note that the origins of many adult diseases can be found among adversities in the early years of life, establishing "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 evolutionary novel environments (Barker, 1994; Barker *et al.*, 2002).

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 6 months, 5 years, and 10 years. Increased activation of the HPA-axis produces glucocorticoids by the adrenal. The latter are important for normal brain maturation, but elevated levels impair brain development and functioning. Some of this is reversible, since the effects of prenatal stress is often moderated by the quality of postnatal care; others are not (Cottrell and Seckle, 2008). Chronic exposure to stress hormones, whether it occurs during the prenatal period, infancy, childhood, adolescence, adulthood or aging, has an impact on brain structures involved in cognition and mental health (Evans and Schamberg, 2009; Lupien *et al.*, 2009; Rice *et al.*, 2010). That there are many pathways to low birth-weight perhaps explains why the incidence of low birth-weight in rich countries remains high (Table 2).

7 Morals

What morals do we take away from the account I have collated here? The following seem to me to be worth commending:

1. 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 consequence of unequal distributions of assets.

3. High maintenance costs are manifestations of complementarities among the inputs that we 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 marked effect on a person's subsequent abilities 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 should conclude that personal history has a long reach, affecting not only the person in question but also her descendents.

These observations bring us back to the point with which I began, that a person's current productivity is a function of her nutritional and morbidity history. A reasonable index of a person's productivity over time would be the present-discounted sum of her 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 within poor countries. This is as it should be. Attention has also been paid by international agencies toward keeping children alive in normal times through public health measures, such as

family-planning counselling, immunization and oral rehydration. This too is as it should be. That many poor countries fail to do either is not evidence of the problems being especially hard to solve. In fact they are among the easier social problems: they can be fielded even while no major modification is made to the prevailing resource allocation mechanism. Much the harder problem, in intellectual design, political commitment and administration, is to ensure that those who are conceived have a chance at a healthy life. It is also 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 anaemia among children and, at one step removed, among mothers. It affects learning and skill formation, and thereby 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 socio-ecological 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. (A1)

Because the resting metabolic rate is positive, $R(H_1) > 0$ and $J(H,q) = 0$ in the neighbourhood 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 initial health status $H(0)$ is even slightly in excess of H_2 would enjoy improvement in his 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 neighbourhood 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 of 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 in 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 (i) factors that are exogenous to the economy, such as rainfall, and factors that are exogenous to the person but are endogenous in the economy; such as (ii) the effectiveness of property rights, (iii) the extent to which government and communities have in place effective support systems, (iv) the degree to which markets are open to the person, and (v) the person's non-labour assets (including education). To them we ought to add (vi) the extent to which the person has reasons to trust others and others trust him.

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.

References

- Åstrand, P.O. and K. Rodahl (1986), *Textbook of Work Physiology* (McGraw Hill, New York).
- Banerjee, A.V., R. Benabou, and D. Mookherjee, eds. (2006), *Understanding Poverty* (New York: Oxford University Press).
- Banerjee, A. and E. Duflo (2007), "The Economic Lives of the Poor", *Journal of Economic Perspectives*, 21(1), 141-168.
- Barac-Nieto, M., G.B. Spurr, H.W. Dahners, and M.G. Maksud (1980), "Aerobic Work Capacity and Endurance During Nutrition Repletion of Severely Undernourished Men", *American Journal of Clinical Nutrition*, 33(11), 2268-2275.
- Barker, D.J.P. (1994), *Mothers, Babies, and Disease in Later Life* (London: BMJ Publishing).
- Barker, D.J.P., C. Osmond, J. Golding, D. Kuh, and M.E Wadsworth (1989a), "Growth *in utero*, Blood Pressure in Childhood and Adult Life, and Mortality from Cardiovascular Disease," *British Medical Journal*, 298, 564-567.
- Barker, D.J.P., B.D. Winter, C. Osmond, B. Margetts, and S.J. Simmonds (1989b), "Weight in Infancy and Death from Ischaemic Heart Diseases," *Lancet*, 2(86663), 381-383.
- Barker, D.J., J.G. Eriksson, T. Forsen and C. Osmond (2002), "Fetal Origins of Adult Disease: Strength of Effects and Biological Basis," *International Journal of Epidemiology*, 31, 1235-1239.
- Basta, S.S., Soekirman, D. Karyadi, and N.S. Scrimshaw (1979), "Iron Deficiency Anemia and the Productivity of Adult Males in Indonesia," *American Journal of Clinical Nutrition*, 32(4), 916-925.
- Bateson, P. and P. Martin (1999), *Design for a Life: How Behaviour Develops* (London: Jonatahan Cape).
- Benton, D. (2010), "The Influence of Dietary Status on the Cognitive Performance of Children", *Molecular Nutrition & Food Research*, 54(4), 457-470.
- Bhargava, A. (1994), "Modelling the Health of Phillipino Children", *Journal of the Royal Statistical Society A*, 157, 417-32.
- Celedon, J.M. and I. de Andraca (1979), "Psychomotor Development during Treatment of Severely Marasmic Infants", *Early Human Development*, 3, 267-275.
- Chavez, A. and C. Martinez (1979), "Consequences of Insufficient Nutrition in Child Character and Behaviour", in D.A. Levitsky, ed., *Malnutrition, Environment and Behaviour* (Ithaca, NY: Cornell University Press).
- Chavez, A. and C. Martinez (1984), "Behavioural Measurements of Activity in Children and their Relation to Food Intake in a Poor Community", in E. Pollitt and P. Amante, eds., *Energy Intake and Activity* (New York: Alan R. Liss).
- Collins, K.J. and D.F. Roberts, eds. (1988), *Capacity for Work in the Tropics* (Cambridge University Press, Cambridge).

Colombo, M. and I. Lopez (1980), "Evolution of Psychomotor Development in Severely Undernourished Infants Submitted to an Integral Rehabilitation", *Pediatrics Research*, 14 (1), abstracts.

Cottrell, E.C. and J.R. Seckl (2009), "Prenatal Stress, Glucocorticoids and the Programming of Adult Disease", *Frontiers in Behavioral Neuroscience*, 3, Online-Open Access.

Creedy, J. and G. Kalb, eds. (2006), *Research on Economic Inequality, Vol. 13: Dynamics of Inequality and Poverty* (London: Emerald Publishing).

Cunha, F. and J. Heckman (2007), "The Technology of Skill Formation", *American Economic Review*, 97(Papers & Proceedings), 31-47.

Cunha, F., J. Heckman, and S. Schennach (2010), "Estimating the Technology of Cognitive and Noncognitive Skill Formation", *Econometrica*, 78(3), 883-931.

Dasgupta, P. (1993), *An Inquiry into Well-Being and Destitution* (Oxford: Clarendon Press (Oxford)).

Dasgupta, P. (2000) "Reproductive Externalities and Fertility Behaviour", *European Economic Review* (Papers & Proceedings), 44 (4-6), 619-644.

Dasgupta, P. (2003), "Population, Poverty, and the Natural Environment", in K.-G. Mäler and J. Vincent, eds, *Handbook of Environmental Economics, Vol. I* (Amsterdam: North Holland), pp. 191-247.

Dasgupta, P. (1997), "Nutritional Status, the Capacity for Work and Poverty Traps", *Journal of Econometrics*, 77(1), 5-38.

Dasgupta, P. (2009). "Poverty Traps: Exploring the Complexity of Causation", in J. von Braun, R. Vargas Hill, and R. Pandya-Lorch, eds., *The Poorest and the Hungry: Assessments, Analyses, and Actions* (Washington, DC: International Food Policy Research Institute), pp. 129-146.

Dasgupta, P. and D. Ray (1986), "Inequality as a Determinant of Malnutrition and Unemployment, 1: Theory", *Economic Journal*, 96(4), 1011-1034.

Deolalikar, A.B. (1988), "Nutrition and Labour Productivity in Agriculture: Estimates for Rural South India," *Review of Economics and Statistics*, 70, 406-13.

Ellison, P. (2010), "Fetal Programming and Fetal Psychology", *Infant and Child Development*, 19(1), 6-20.

Evans, G.W. and M.A. Schamberg (2009), "Childhood Poverty, Chronic Stress, and Adult Working Memory", *PNAS*, 106(16), 6545-6549.

Ferro-Luzzi, A. (1985), "Work Capacity and Productivity in Long-term Adaptation to Low Energy Intakes", in K. Blaxter and W.C. Waterlow, eds., *Nutritional Adaptation in Man* (John Libbey, London).

Fumagalli, F., R. Molteni, G. Racagni, and M.A. Riva (2007), "Stress During Development: Impact on Neuroplasticity and Relevance to Psychopathology," *Progress in Neurobiology*, 81, 197-217.

Georgieff, M.K. (2007), "Nutrition and the Developing Brain: Nutrient Priorities and Measurement", *American Journal of Clinical Nutrition*, 85(Supplement), 614S-620S.

- Gluckman, P. and M. Hanson (2006), *Mismatch* (New York: Oxford University Press).
- Gluckman, P.D., M.A. Hanson, and H.G. Spenser (2005), "Predictive Adaptive Responses and Human Evolution", *Trends in Ecology and Evolution*, 20, 527-533.
- Grantham-McGregor, S.M. (1990), "The Relationship between Undernutrition, Activity Levels and Development in Young Children", in B. Schurch and N.S. Scrimshaw, eds., *Activity, Energy Expenditure and Energy Requirements in Young Children* (Lausanne: Nestle Foundation).
- Immink, M.D.C. *et al.* (1984), "Microeconomic Consequences in Energy Deficiency in Rural Populations in Developing Countries," in E. Pollitt and P. Amante, eds., *Energy Intake and Activity* (Alan R. Liss, New York).
- Kar, B.R., S.L. Rao, and B.A. Chandramouli (2008), "Cognitive Development in Children with Chronic Protein Energy Malnutrition", *Behavioral and Brain Functions*, 4, article 31, open access.
- Levitsky, D.A. and B.J. Strupp (1995), Malnutrition and the Brain: Changing Concepts, Changing Concerns," *Journal of Nutrition*, 125(8), S2212-S2220.
- Lupien, S.J., B.S. McEwen, M.R. Gunnar, and C. Heim (2009), "Effects of Stress Throughout the Lifespan on the Brain, Behaviour and Cognition", *Nature Reviews/Neuroscience*, 10(6), 434-445.
- M.E.A. - Millennium Ecosystem Assessment - (2003), *Ecosystems and Human Well-Being* (Washington DC: Island Press).
- Narayan, D., with R. Patel, K. Schafft, A. Rademacher, and S. Koch-Schulte (2000), *Voices of the Poor: Can Anyone Hear Us?* (Oxford: Oxford University Press).
- Pollitt, E. (1990), *Malnutrition and Infection in the Classroom* (UNESCO, Paris).
- Pollitt, E. and P. Amante (1984), *Energy Intake and Activity* (Alan R. Liss, New York).
- Rice, F., G.T. Harold, J. Boivin, M. van den Bree, D.F. Hay, and A. Thapar (2010), "The Links Between Prenatal Stress and Offspring Development and Psychopathology: Disentangling Environmental and Inherited Influences", *Psychological Medicine*, 40(2), 335-345.
- Rooij, S.R. de, H. Wouters, J.E. Yonker, R.C. Painter, and T.J. Roseboom (2010), "Prenatal Undernutrition and Cognitive Function in Late Adulthood", *PNAS*, 107(39), 16881-16886.
- Roseboom, T., S.R. de Rooij, and R. Painter (2006), "The Dutch Famine and Its Long-Term Consequences for Adult Health", *Early Human Development*, 82, 485-491.
- Satyanarayana, K., A.N. Naidu, B. Chatterjee, and N. Rao (1977), "Body Size and Work Output," *American Journal of Clinical Nutrition*, 30(3), 322-5.
- Schultz, T.P. (2006), "Fertility and Income", in A.V. Banerjee, R. Benabou, and D. Mookherjee, eds., *Understanding Poverty* (New York: Oxford University Press).
- Schulz, L.C. (2010), "The Dutch Hunger Winter and the Developmental Origins of Health and Disease", *PNAS*, 107(39), 16757-16758.
- Schurch, B. and N.S. Scrimshaw, eds. (1987), *Chronic Energy Deficiency: Consequences*

and Related Issues (Lausanne: Nestle Foundation).

Sen, A. (1999), *Development as Freedom* (Oxford: Oxford University Press).

Shonkoff, J.P., W.T. Boyce, and B.S. McEwen (2009), "Neuroscience, Molecular Biology, and Childhood Roots of Health Disparities: Building a New Framework for Health Promotion and Diseases Prevention", *JAMA (Journal of the American Medical Association)*, 301(21), 2252-2259.

Spurr, G.B. (1990), "The Impact of Chronic Undernutrition on Physical Work Capacity and Daily Energy Expenditure," in G.A. Harrison and J.C. Waterlow, eds., *Diet and Disease in Traditional and Developing Countries* (Cambridge University Press, Cambridge).

Strauss, J. (1986), "Does Better Nutrition Raise Farm Productivity?", *Journal of Political Economy*, 94, 297-320.

Strauss, J. and D. Thomas (1998), "Health, Nutrition and Economic Development," *Journal of Economic Literature*, 36(2), 766-817.

Thomas, D. and J. Strauss (1997), "Health and Wages: Evidence on Men and Women in Urban Brazil," *Journal of Econometrics*, 77(1), 159-185.

Turner, B.L. and A.M.S. Ali (1996), "Induced Intensification: Agricultural Change in Bangladesh with Implications for Malthus and Boserup," *Proceedings of the National Academy of Sciences*, 93, 14984-14991.

Viteri, F.E. (1971), "Considerations on the Effects of Nutrition on the Body Composition and Physical Work Capacity of Young Guatemalan Adults," in N.S. Scrimshaw and A.M. Altshull, eds., *Amino Acid Fortification of Protein Foods* (MIT Press, Cambridge, MA).

Walker, C.-D. (2005), "Nutritional Aspects Modulating Brain Development and the Responses to Stress in Early Neonatal Life", *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 29, 1249-1263.

Waterlow, J.C. - with contributions by A.M. Tomkins and S.M. Grantham-McGregor - (1992), *Protein Energy Malnutrition* (Sevenoaks: Edward Arnold).

WHO (1985), *Energy and Protein Requirements* (Geneva: World Health Organization, Technical Report Series 724).

World Bank (2010), *World Development Indicators* (Washington DC: World Bank).

Table 1
Clean Water and Sanitation, 2006
access

	clean water (%)	sanitation (%)
Low income countries	67	39
South Asia	87	33
(India)	89	28
S-S Africa	58	31
China	88	65
High income countries	99	99
World	86	60

Source: *World Development Indicators* (2010), Table 2.18

Table 2
Prevalence of Child Undernutrition, 2000-2008

	wasted	stunted	low birthweight (≤ 2.5 kg)
	percentage of children under 5		percentage
Low income countries	28	44	15
South Asia	41	47	27
(India)	44	48	28
s-S Africa	25	43	14
China	7	22	2
United States	1.3	4	8
World	23	35	15

Source: *World Development Indicators* (2010), Table 2.20

Table 3
Maternal Burden 2008

	TFR	Maternal mortality (per 100,000)
Low income countries	4.0	790
S-S Africa	5.1	900
South Asia	2.9	500
China	1.8	45
High income countries	1.8	10

Source: *World Development Indicators* (2010), Table 2.19

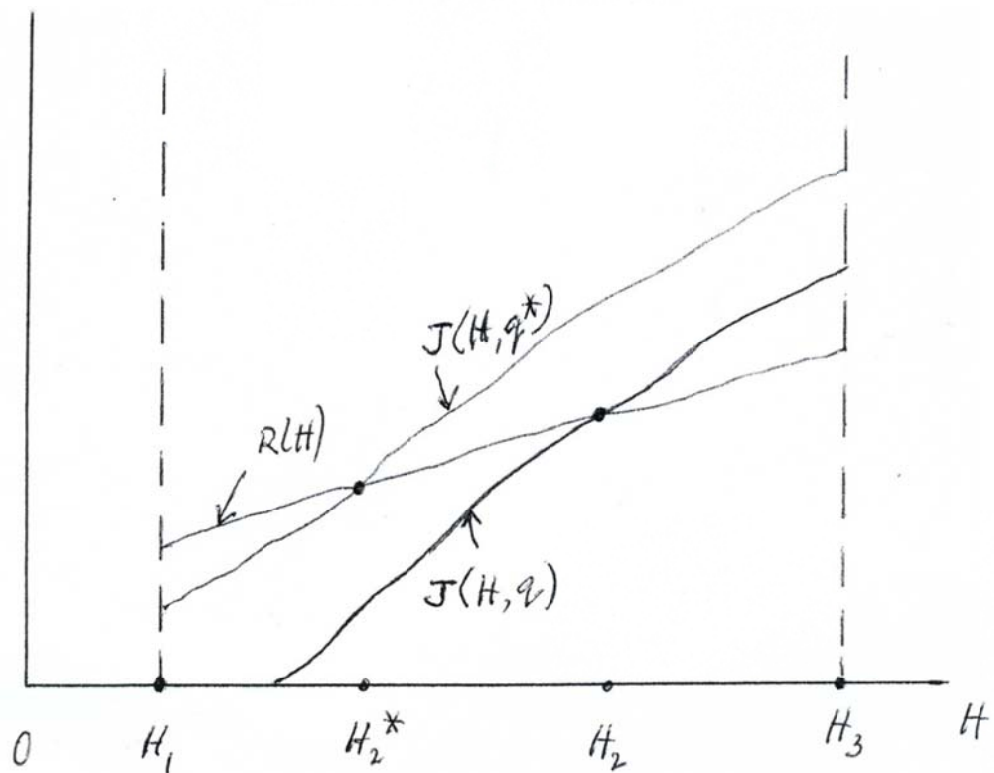


Figure 1