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Osmani, S. R., & Sen, A. (2003). The Hidden Peanlties of Gender Inequality: Fetal Origins of Ill-Health. *Economics and Human Biology*, 1, 105-121.

[Link to publication record in Ulster University Research Portal](#)

Published in:
Economics and Human Biology

Publication Status:
Published (in print/issue): 18/02/2003

Document Version
Publisher's PDF, also known as Version of record

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ELSEVIER

Economics and Human Biology 1 (2003) 105–121

ECONOMICS
AND
HUMAN BIOLOGY

www.elsevier.com/locate/ehb

The hidden penalties of gender inequality: fetal origins of ill-health

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Abstract

This paper is concerned with the interconnections between gender inequality and maternal deprivation, on the one hand, and the health of children (of either sex) and of adults that the children grow into (again, of either sex). The basic message of the paper is that women's deprivation in terms of nutrition and healthcare rebounds on the society as a whole in the form of ill-health of their offspring—males and females alike—both as children and as adults. There are a variety of pathways through which women's deprivation can affect the health of the society as a whole. This paper focuses on the pathways that operate through undernourishment of the mother. Maternal deprivation adversely affects the health of the fetus, which in turn leads to long-term health risks that extend not just into childhood but into adulthood as well. There are, however, important differences in the way children and adults experience the consequences of maternal deprivation via fetal deprivation. In particular, the pathways that lead to their respective risk factors and the circumstances under which those risk factors actually translate into ill-health are very different. These differences are best understood through the concept of 'overlapping health transition' in which two different regimes of diseases coexist side by side. Gender inequality exacerbates the old regime of diseases among the less affluent through the pathway of childhood undernutrition. At the same time it also exacerbates the new regime of diseases among the relatively more affluent through a pathway that has come to be known as the 'Barker hypothesis'. Gender inequality thus leads to a double jeopardy—simultaneously aggravating both regimes of diseases and thus raising the economic cost of overlapping health transition.

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Keywords: Gender inequality; Fetal deprivation; Ill-health

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1. Introduction

In disparate ways, gender inequality survives in most parts of the world in different forms, from Japan to Zambia, from Ukraine to the USA. However, in some areas, such as North Africa and Asia (including China), inequality directly involves matters of life and death, and takes the brutal form of unusually high mortality rates of women, and a consequent preponderance of men in the total population. By drawing on the experience of South Asia, where gender inequality finds a particularly sharp expression, we argue that gender discrimination hurts not just women, but imposes a heavy economic cost on the society by harming the health of all, including that of men. South Asia is currently undergoing an ‘overlapping health transition’, in which a new regime of diseases is gaining prominence even as the old regime continues to wreck havoc. Gender discrimination aggravates both regimes at the same time—albeit through different pathways—and thereby raises the economic cost of managing the health transition.

In most of the Subcontinent, with only a few exceptions (such as Sri Lanka and the state of Kerala in India), female mortality rates are significantly higher than expected given the mortality of men (in the respective age groups). This type of gender inequality is not the result of conscious homicide.³ Rather, the mortality disadvantage of women comes about mainly through a widespread neglect of health, nutrition and other interests of women that influence survival.⁴

While these differences in sex-specific mortality rates may escape immediate notice (since the demography of comparative incidence of mortality is not altogether transparent), their quantitative significance and cumulative impact can be brought out with some immediacy by specially devised indicators, such as the incidence of “missing women”.⁵

The time has come pay more attention to those consequences of gender inequality that have not yet received much attention, and indeed, have not yet been at all adequately investigated. These include the *interconnections* between gender inequality and maternal deprivation, on the one hand, and the health of children (of both sexes) and of adults that they grow into, on the other. Our argument is that women’s deprivation in terms of nutrition and healthcare rebounds on the society in the form of ill-health of their offspring—males and females alike. Given the uniquely critical role of women in the reproductive process, it would be hard to imagine that the deprivation to which women are subjected would not have some adverse impact on the lives of all—children as well as adults—who are “born of a woman” (as the *Book of Job* describes every person).

A variety of pathways exist through which women’s deprivation affect the health of the society as a whole. This paper focuses on the pathways that operate through under-nourishment

³ It would be a mistake to try to explain this large phenomenon by invoking the occasional cases of female infanticide that are reported from China or India; these are truly dreadful events when they occur, but they would seem to be statistically extremely rare.

⁴ On this see Bardhan (1974, 1984), Chen et al. (1981), Kynch and Sen (1983), Sen and Sengupta (1983), Sen (1984, 1985), Harriss and Watson (1987), Drèze and Sen (1989, 1995), Harriss (1990), and Kanbur and Haddad (1990), among other contributions.

⁵ See Sen (1988, 1989, 1990, 1992b), Drèze and Sen (1989, 1995), Coale (1991), and Klasen (1994), among many other contributions. In this form, women’s survival disadvantage has received considerable attention in public discussions in recent years.

of the mother. Maternal deprivation adversely affects the health of the fetus, which, in turn, leads to long-term health risks that extend beyond childhood into adulthood as well. There are, however, important differences in the way children and adults experience the consequences of maternal deprivation via fetal deprivation. In particular, the pathways that lead to their respective risk factors and the circumstances under which those risk factors actually translate into ill-health are very different. One way of understanding these differences is to refer to the notion of ‘health transition’ that many developing countries are currently experiencing.

As a population approaches the end of demographic transition, it is not only the level of mortality that changes; the causes of mortality also change. In particular, infectious communicable diseases give way to chronic and non-communicable diseases as the major source of morbidity and mortality, the problems of undernutrition are replaced by those of overnutrition, and diseases of the old rise in importance relative to those of the young. The term ‘epidemiological transition’ or ‘health transition’ refers to this process of change.⁶

The western developed world went through this transition over a long period, spanning more than a century. However, the present-day developing countries are experiencing the same transition at a much faster pace owing to a historically unprecedented rapid pace of fertility decline and income growth. One consequence of the transitions being telescoped into a short period is the emergence of what might be called the phenomenon of ‘overlapping transition’, in which the disease patterns associated with the early and the late stages of transition are becoming simultaneously prominent.⁷ For instance, in the next 20 years, more than half of the world’s cardiovascular morbidity and mortality will occur in the developing world, India will have the largest number of diabetic patients, and coronary heart disease will become the leading cause of mortality in India.⁸ Yet, the diseases of the old regime, caused primarily by the undernutrition-infection nexus, will remain a major cause of morbidity and mortality.⁹

While the diseases of the two regimes exist simultaneously during ‘overlapping transition’, the population groups affected by them tend to be somewhat different. At the risk of slight simplification, it is fair to suggest that diseases of the old regime take their toll primarily among children of the poorer segments of the population, while diseases of the new regime are more common among adults of the relatively better off segments of the populations of the developing world.¹⁰ The findings below suggest that even though the aetiology and the population at risk from these two types of diseases are quite different, they are both exacerbated by the common factor of maternal deprivation operating via fetal deprivation. Thus, the neglect of females exposes children to diseases of the old regime, and at the

⁶ Epidemiologists tend to make further distinctions between these two terms, but the differences are not very important for our purpose. For elaboration of the concept of health transition, see, among others, [Caldwell \(1993\)](#) and [Chen et al. \(1994\)](#).

⁷ The concept of overlapping health transition is further elaborated in [Osmani and Bhargava \(1998\)](#).

⁸ See [Yajnik \(2001\)](#).

⁹ For the changing nature of morbidity and mortality in the developing world, see [Murray and Lopez \(1996\)](#) and [ACC/SCN \(2000a\)](#).

¹⁰ This is in contrast to the current state of the developed world, where the poorer people suffer more than the rich from diseases of the new regime such as the coronary heart disease.

same time reduces adult health through diseases of the new regime, albeit through different pathways. These connections between gender inequality and general health have remained relatively understudied. Hence, we focus on these “hidden penalties” more explicitly below. Recent medical studies have made the biological and medical connections in this complex relationship much clearer. There is also a general need to connect the social and economic literature with the state of medical knowledge.¹¹ However, before we delve into the new issues, we discuss the older issues (the “unhidden” and, by now, well known penalties of gender inequality), since the former can be helpfully understood as an extension of the latter.

2. Mortality differentials and missing women

It is sometimes presumed that there are more women than men in the world, since that is the case in Europe and North America, which have a female to male ratio of about 1.05. Yet, women do not outnumber men in the world; indeed there are only about 98 women per 100 men on the globe.¹² This “shortfall” of women is most acute in Asia and North Africa: the number of females per 100 males in the population is 97 in Egypt and Iran, 95 in Turkey, 94 in China and Bangladesh, 93 in India, 92 in Pakistan.¹³

Given similar health care and nutrition, women typically tend to have lower age-specific mortality rates than men do. Even though this difference favouring women’s survival is sometimes enhanced by non-biological, social influences (for example, the higher propensity of men to die from violence, and very often the greater tendency of men to smoke compared with women), the greater survival potential of women—given similar care—appears to reflect primarily biological differences. Indeed, even female fetuses tend to have a lower probability of miscarriage than male fetuses have.¹⁴ Everywhere in the world, more male babies are born than female babies, but throughout their respective lives the proportion of males goes on falling as we move to higher and higher age groups, due typically to greater male mortality rates. The excess of females over males in the population of Europe and North America comes about as a result of this greater survival chance of females in different age groups.

However, in many parts of the world, females receive fewer resources, and less attention and health care than males do. As a result of this bias, the mortality rates of females often exceed those of males in many countries. The concept of “missing women” conveys some idea of the enormity of the phenomenon of women’s adversity in mortality and enables us to understand the quantitative difference between (1) the actual number of women in these countries, and (2) the number we expect to see if the gender pattern of mortality were similar in these countries as in other regions of the world that do not have a significant bias against women in terms of health care and other attentions relevant for survival.

¹¹ See also Dasgupta (1993) and Osmani (1992).

¹² On the issue of sex balance of world population, see Kynch (1985).

¹³ It is only 84 in Saudi Arabia, though this ratio is considerably reduced by the presence of male migrant workers.

¹⁴ See Waldron (1976) and the literature cited there.

For example, if we take the ratio of women to men in Sub-Saharan Africa as the standard (there is relatively little bias against women in terms of health care, social status and mortality rates in Sub-Saharan Africa), then its female–male ratio of 1.022 can be used to calculate the number of missing women elsewhere (Sen, 1988; Drèze and Sen, 1989). Other standards and different procedures can also be used, and more ambitiously, it is possible to make some guess of the likely decrease in age-specific mortality rates of women if they were to receive the same care as men do (Coale, 1991). Moreover, historical techniques have recently been used to calculate the “gap” of missing women in contemporary third world by comparing the actual number of women with the expected number estimated on the basis of European demographic history (Klasen, 1994).

With India’s female–male ratio of 0.93, there is a difference of 9% (of the male population) between that ratio and the Sub-Saharan African ratio of 1.02. This method of estimation in 1986 yielded a figure of 37 million missing women in India. Using the same Sub-Saharan standard, the number of “missing women” in the world appeared to be more than 100 million¹⁵ (Sen, 1989; Drèze and Sen, 1989). The precise figures are, in fact, not particularly significant, but it is quite important to seize the fact that the magnitudes involved are extraordinarily large. Gender bias does take an astonishingly heavy toll in matters of life and death.

3. Five stylised facts of relative deprivation

Moving away from these old problems of gender bias in life and death to the ‘hidden penalties’ of gender bias referred to above, we may begin by noting five substantial phenomena that are quite widely observed in South Asia.

3.1. Greater undernourishment of girls than boys

At the time of birth, girls are no more nutritionally deprived than boys are, but this situation changes as society’s unequal treatment takes over: girls fall nutritionally behind in most areas of South Asia according to aggregate statistical evidence.¹⁶ But there has been some anthropological scepticism of the appropriateness of using aggregate statistics with pooled data from different regions to interpret the behaviour of individual families.¹⁷ There has also been some understandable reluctance to accuse South Asian families of serious impropriety and deeply unjust behaviour without evidence on the micro level. This raises two distinct issues. First, are the aggregate findings vindicated by “micro” investigations that compare individual families within the same neighbourhood? Second, how precisely do we interpret the mechanism of family decisions that possibly generate the relative deprivation of girls?

¹⁵ Klasen’s (1994) estimate with European historical comparisons is a little lower, viz. about 80 million.

¹⁶ The empirical literature on this was critically assessed in the 1980s in Kynch and Sen (1983), Sen (1984, 1985), Harriss (1990) and Kanbur and Haddad (1990).

¹⁷ On this and related issues, see among other contributions, Harriss and Watson (1987), Basu (1989), and Basu and Jeffery (1996).

There have actually been some detailed micro studies on this issue, which do not contradict the results of aggregate statistics.¹⁸ One case study from India involved the weighing of every child in two large villages (Sen and Sengupta, 1983). The study used weight-for-age as an indicator of nutritional status of children under five, and showed clearly how an initial condition of broad nutritional symmetry turned gradually into a significant female disadvantage. Following the same methodology, a subsequent survey a decade later in the same villages (plus four additional ones) demonstrated the continued existence of female disadvantage despite an overall improvement in the nutritional status of both boys and girls (Sengupta and Gazdar, 1996). Hence, the microstudies tend to confirm the patterns that emerges from aggregate statistics.

Regarding the second issue, it is important to emphasise that the lower level of nourishment of girls may not relate directly to their being underfed vis-à-vis boys, and the differences may particularly arise from the neglect of health care of girls compared with that of boys. There is some direct information of comparative medical neglect of girls vis-à-vis boys in South Asia. Undernourishment may well result from more morbidity, which can both adversely affect the absorption of nutrients and the retention of bodily functions.¹⁹ This can either add to asymmetries of food consumption, or work on its own, without any food asymmetry. In either case, there is clear evidence of anti-girl bias in family behaviour no matter what the exact causal mechanisms underlying it are. One way or another, the social norms and individual behaviour in most of South Asia seem to permit considerable gender bias.

3.2. Higher maternal undernourishment

Women of reproductive age suffer more from undernourishment in South Asia than in other regions of the world as indicated by the Body Mass Index (BMI)²⁰ (Tables 1 and 2). Even with a number of different cut-off points used to indicate mild, moderate and severe undernutrition, it is remarkable that the prevalence of female undernutrition is consistently far higher in Bangladesh than in any other developing country for which comparable data are available. Over half the women of reproductive age in Bangladesh are undernourished, whereas the highest prevalence of undernutrition in the countries of Sub-Saharan African is around 20%.²¹

Another indicator of the especially severe health and nutritional disadvantage suffered by the South Asian women is the incidence of anaemia, which can reflect both dietary deficiency and the deleterious effect of infection on iron utilisation. The incidence of anaemia is much greater in the Subcontinent compared even with other poor countries in the world, including Sub-Saharan Africa (Table 3).

¹⁸ See, for example, Chen et al. (1981) and Kynch and Sen (1983), and the micro studies cited there.

¹⁹ On this see also Kynch and Sen (1983), Sen (1984, 1985), and Osmani (1997), and the cited empirical sources of the comparisons made in these studies.

²⁰ BMI is weight (in kilograms)/height squared (in meters) and is the most widely accepted measure of adult nutrition.

²¹ Comparable data on the female BMI are not available for other South Asian countries. But other evidence on maternal undernutrition, including the incidence of anaemia discussed below, would seem to suggest that the problem of low BMI might well be quite common for women in the whole region.

Table 1
Percentage of undernourishment of children (below 5) by sex in two rural areas of West Bengal, India

Group	Below I	Below II	Below III	Below IV	Undernourishment index
Shahajapur boys	94	71	39	6	53
Shahajapur girls	92	73	44	9	55
Kuchli boys	79	52	19	7	39
Kuchli girls	90	75	48	8	55

Notes: The categories of undernourishment are based on standard weight curves used in West Bengal and are defined as follows. Above I: well-nourished; between I and II: slightly undernourished; between II and III: moderately undernourished; between III and IV: severely undernourished; below IV: disastrously undernourished. The undernourishment index was calculated by first taking a weighted average of the four categories of undernourishment—the weights being 1 for slight, 2 for moderate, 3 for severe and 4 for disastrous. The weighted average was then normalised to run the score between 0 (in the case in which everyone is on or above line I) and 100 (in the case in which everyone is below line IV). Source: [Sen and Sengupta \(1983\)](#), Table 4.

3.3. Greater incidence of low birth weight

It is also found that South Asia has the highest incidence of low birth weight (LBW) babies,²² compared with all other regions in the world ([Table 4](#)). About 21% of the infants born in South Central Asia in the year 2000 had low birth weight, nearly twice the average for all developing countries, and considerably higher than the figures for any other developing region for which data are available.²³ In addition, country-specific data in the WHO global database indicate very high prevalence of low birth weight in South Asian countries separately. For instance, in Bangladesh the prevalence is as high as 40% ([de Onis et al., 1998](#)).

3.4. Larger incidence of child undernutrition

South Asia suffers from the worst incidence of child undernutrition among all regions of the developing world, including Sub-Saharan Africa. In 1995 nearly half of the under-five children of South Asia were underweight, as compared to less than a third in the developing world as a whole, including Sub-Saharan Africa ([Table 5](#)). Separate estimates of chronic undernutrition (measured by stunting or low height-for-age) and acute undernutrition (measured by wasting, or low weight-for-height) also indicate a particularly dismal situation in South Asia ([ACC/SCN, 2000a](#)).

The prevalence of undernutrition in South Asia has actually been declining faster in the recent decades compared to all other developing regions ([Table 5](#)). Yet, the initial level of undernutrition was so high that it still remains the highest. This phenomenon has perplexed many observers, because South Asia should not fare worse than Sub-Saharan Africa on the basis of conventional determinants of child undernutrition—such as income,

²² Low birth weight (LBW) is defined as a body weight at birth of less than 2500 g. There are two main causes of LBW: premature birth and intrauterine growth retardation (IUGR). LBW is often used as a proxy for IUGR in developing countries because valid assessment of gestational age is generally not available to isolate the effects of premature birth. Some estimates, however, suggest that at least two-thirds of the cases of LBW in developing countries are caused by IUGR. For more on this and related concepts, see [ACC/SCN \(2000b\)](#), Box 1.

²³ South Central Asia includes both South Asia and the former Soviet republics of Central Asia.

Table 2
Prevalence of underweight (low BMI) among 20–49 years old women

Region/country	Severe	Moderate	Mild
Africa			
Benin	1.2	2.1	10.8
Burkina Faso	0.6	2.3	10.6
Central African Republic	1.2	2.1	12.0
Chad	2.1	4.2	13.5
Comoros	0.4	1.2	7.0
Cote d'Ivoire	0.4	1.5	6.7
Egypt	0.1	0.4	1.3
Ghana	0.8	1.8	1.9
Kenya	0.7	1.4	8.6
Madagascar	1.7	3.1	15.1
Malawi	0.4	1.3	6.3
Namibia	1.1	1.8	9.9
Niger	0.9	2.7	15.8
Tanzania	0.8	1.5	6.6
Uganda	0.5	1.3	7.5
Zambia	0.5	0.9	7.1
Latin America			
Bolivia	0.0	0.2	0.5
Brazil	0.3	1.2	5.1
Colombia	0.1	0.5	2.9
Dominican R	0.5	0.8	5.1
Guatemala	0.3	0.5	2.5
Haiti	2.0	3.6	13.0
Hondurus	1.1	1.0	6.5
Mexico	1.0	1.5	5.1
Nicaragua	0.2	0.3	3.0
Peru	0.0	0.1	0.9
Asia			
Bangladesh	9.5	13.5	28.3
Nepal	2.9	5.4	19.1
Uzbekistan	1.4	1.2	8.5

Note: severe: BMI < 16.0 kg/m²; moderate: 16.00 ≤ BMI < 16.99 kg/m²; mild: 17.00 ≤ BMI < 18.50. Source: ACC/SCN (2000a), Table 1.8.

Table 3
Maternal health: prevalence of anaemia among pregnant women (%)

South Asia	78
East and South-east Asia	35
Sub-Saharan Africa	43
Middle East/North Africa	38
Latin America/Caribbean	37
Developing countries	52

Source: UNDP (1984).

Table 4
Incidence of low birth weight babies (%)

South Asia	20.9
South-east Asia	5.6
Western Asia	4.5
Middle Africa	14.9
West Africa	11.4
Latin America/Caribbean	6.5
Developing countries	11.0

Source: ACC/SCN (2000a), Table 1.1.

Table 5
Child undernutrition in developing countries by region: 1970 and 1995 (percentage of underweight children under 5 years of age)

Region	1970	1995	Change 1970–1995 (% points)
South Asia	72.3	49.3	–23.0
Sub-Saharan Africa	35.0	31.1	–3.9
East Asia	39.5	22.9	–16.6
Near East and North Africa	20.7	14.6	–6.1
Latin America/Caribbean	21.0	9.5	–11.5
All developing regions	46.5	31.0	–15.5

Source: Smith and Haddad (2000), Table 1.

food availability, provision of health care, and education. This paradoxical phenomenon has come to be known as the ‘Asian enigma’ (Ramalingaswami et al., 1996).

3.5. Larger incidence of adult ailments

The phenomenon of ‘health transition’ has been sweeping through South Asia along with other regions of the developing world, bringing with it the emergence of new diseases such as diabetes and cardiovascular diseases. The South Asians suffer more from these diseases than many others who have progressed further along the path of transition, as indicated by the extent of fertility decline, population ageing and income growth. A number of studies on the ethnic pattern of mortality in the United Kingdom have demonstrated that people born in South Asia have a much higher incidence of mortality from cardiovascular diseases than for any other ethnic group, and that this difference cannot be explained by the traditional risk factors associated with such diseases.²⁴ More recently, evidence collected for the “Study on the Global Burden of Disease” has indicated that this pattern holds true for the adults born and residing in South Asia as well²⁵ (Murray and Lopez, 1996). South Asia is the only developing region in the world that has a mortality rate from cardiovascular diseases that

²⁴ See, McKieue and Marmot (1988) and the references cited there.

²⁵ The underlying country data upon which the global burden of disease has been estimated are not always of the highest quality. In particular, the surveys are not always statistically representative, and there are many gaps in the data that were estimated by interpolation. Nevertheless, this appears to be the best data set in existence.

Table 6
Death from cardiovascular diseases (per 1000 population)

Country/region	Male	Female	All
India	7.1	7.1	7.1
China	5.2	5.3	5.3
Other Asia	5.0	4.8	4.9
Sub-Saharan Africa	4.4	5.6	5.0
Latin America/Caribbean	5.0	4.7	4.9
Middle Eastern Crescent	7.1	6.9	6.9
Transition economies	10.0	10.2	10.1
Developed economies	6.9	6.9	6.9
World	6.3	6.5	6.4

Note: Middle Eastern Crescent includes Pakistan, Afghanistan, Middle East, North Africa and the former socialist countries of Central Asia. Transition economies includes the formerly socialist countries of East and Central Europe. Source: calculated from annex Table 6.g of Murray and Lopez (1996).

exceeds the rate for the developed world (Table 6).²⁶ The Indian rate is higher than that of China, even though the traditional risk factors such as animal fat consumption, smoking, and obesity are much higher in China.

4. Interconnections and causal links

The five phenomena discussed above are all distinctive features of South Asia. Are they causally connected, and if so how? Given the preliminary stage of this investigation, the answers to these questions must be somewhat speculative, but that is where we have to begin to initiate further empirical scrutiny.

Our hypothesis is that the five phenomena are integrally connected through a causal chain that involves both social behaviour and biological processes. The starting point of this chain is gender bias, which leads to ill-health (Fig. 1). The links in this chain are as follows: (1) gender bias leads to high maternal undernutrition; (2) maternal undernourishment results in intra-uterine growth retardation for the fetus, and leads to high prevalence of low birth weight;²⁷ (3) low birth weight, in turn, leads both to a high rate of child undernutrition, as well as to (4) the higher-than-expected prevalence of adult ailments, both directly and indirectly through child undernutrition. Taken together, the four links suggest that excessive gender inequality in South Asia accounts for the excessive prevalence of ill-health in this region. These connections are based on the following empirical foundations.

The link between maternal undernutrition and low birth weight babies is highlighted by the Fourth Report on the World Nutrition Situation: “In developing countries, the major determinants of growth retardation in utero are nutritional: inadequate maternal nutritional status before conception, short maternal stature (principally due to undernutrition and

²⁶ The region called the Middle Eastern Crescent has a rate that is very close to India's, but it should be noted that this region includes the formerly socialist countries of Central Asia, which probably share with their counterparts in Eastern Europe the highest rates of mortality from cardiovascular diseases in the world.

²⁷ This linkage is well recognised in the medical literature, Battaglia and Simmons (1979), Kramer (1987, 1998), Henriksen (1999) and Prada and Tsang (1998).

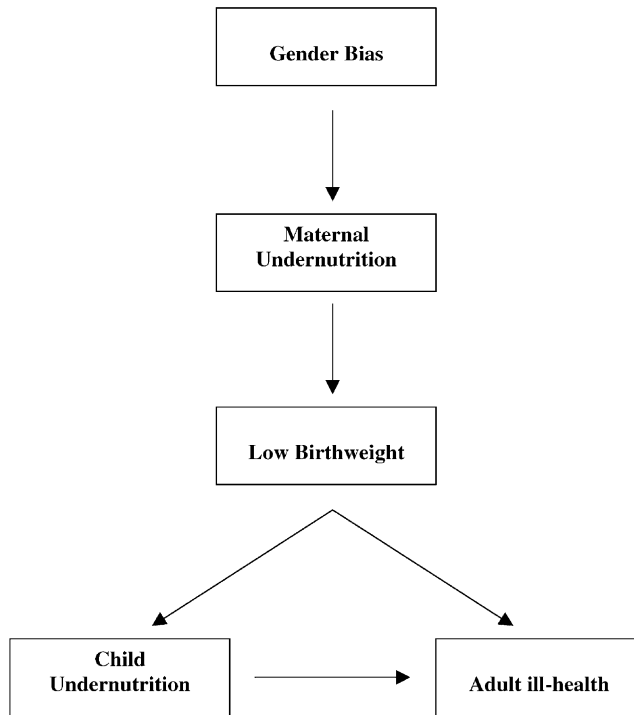


Fig. 1. The pathways from gender inequality to ill-health.

infection during childhood), and poor maternal nutrition during pregnancy (low gestational weight gain, primarily due to inadequate dietary intake). Maternal nutrition during pregnancy is especially important.” (ACC/SCN, 2000a, p. 4). Not surprisingly, the exceptionally high degree of maternal undernourishment in South Asia is reflected in an exceptionally high prevalence of low birth weight babies.

Statistical support for this inference, albeit of a somewhat indirect nature, is provided by an econometric exercise carried out by [Osmani and Bhargava \(1998\)](#), who examine inter-country variation in the prevalence of low birth weight. The explanatory variables includes (a) factors influencing the general level of nutrition and health in the country, such as per capita income, food availability, healthcare facilities, education and urbanisation, (b) factors related specifically to female agency such as female education and female share of the labour force, and (c) information on female age-at-first marriage, because very young mothers are more likely to deliver underweight babies than mature mothers. It is shown that even after allowing for the effects of all these variables, the prevalence of low birth weight in South Asia remains inexplicably high (as captured by a South Asian dummy). Ideally, the set of explanatory variables should have included also some measure of gender bias as reflected in female neglect in intra-household allocation of nutrients and healthcare. The limitations of data availability precluded this, but a consequence of this omission is that in any inter-country regression on low birth weight the effect of the exceptionally high degree

of female neglect in South Asia will show up as an inexplicably high prevalence of low birth weight in this region. This is what is actually found, as captured by the significantly positive coefficient of the dummy variable for South Asia.

The exceptionally high prevalence of low birth weight in South Asia goes a long way towards explaining the exceptionally high prevalence of child malnutrition in this region. There is, of course, no unique one-to-one relationship between the weight at birth and subsequent weight and height during childhood, because of the phenomenon of ‘catch up growth’ which may offset a weight deficit at birth by moving low weight babies along an accelerated growth path. Yet, an overwhelming body of epidemiological evidence suggests that, given the same environment, a baby born with low birth weight is much more likely to remain undernourished (in terms of height-for-age and weight-for-age) than one born with normal weight. The reason is not simply that the initial weight deficit is too big to be fully compensated by subsequent catch up growth, but more importantly, that the rate of catch up growth may itself be too little for them.

The reason lies in the higher susceptibility of low birth weight babies to the debilitating effects of the infection-undernutrition nexus. The processes that cause intra-uterine growth retardation also induce certain physiological changes in the fetus that impair its immune system. While growing up in an environment of poor health and sanitation, as is typical of the living conditions of the poor in developing countries, these babies with reduced immunological competence fall frequent prey to various kinds of infections. The infections, in turn, reduce their ability to absorb nutrients, resulting in slow physical growth, as manifested in low height-for-age and low weight-for-age. Thus, low birth weight babies are more likely to experience childhood malnutrition, even if they have the same food intake and similar environment of health and sanitation as babies with normal weight.²⁸

The biomedical linkage between low birth weight and childhood malnutrition is also confirmed by the cross-country regressions of [Osmani and Bhargava \(1998\)](#). Their results indicate that the ‘Asian enigma’—the unexplained excess of child malnutrition for South Asia, as captured by the dummy variable for this region—disappears if a birth weight variable is introduced into the regression as an additional explanatory variable. The ‘Asian enigma’ is, therefore, essentially a low birth weight enigma.

Two types of pathways link low birth weight to adult ill-health. The first pathway operates through child undernutrition, insofar as low birth weight (LBW) babies who suffer from growth retardation in their childhood generally grow up to be adults of short stature and low body mass (BMI). When the BMI falls below a critical value, a person is at a greater risk of suffering from various functional impairments such as high morbidity, and low work capacity.²⁹ Thus, low birth weight babies suffer more from the functional impairments associated with low BMI.

We have noted earlier that adult females in South Asia suffer from a higher degree of undernutrition, lower BMI, than their counterparts elsewhere in the developing world. Similar comparative figures do not exist separately for adult males, but some fragmentary evidence that exists for overall populations—comprising both males and females—does

²⁸ For references to the extensive medical literature on these linkages, see, among others, [Martorell et al. \(1998\)](#), and [ACC/SCN \(2000a,b\)](#).

²⁹ The evidence on the consequences of low BMI for adults is summarised in [Shetty and James \(1994\)](#).

seem to indicate that the South Asian adults are, on the whole, more undernourished than their counterparts elsewhere (FAO, 1996, Table 25). This is evidently a consequence of an exceptionally high prevalence of low birth weight, operating through the pathway of childhood undernutrition.

There is, however, a different pathway that links low birth weight directly to adult ailments, without being mediated by childhood undernutrition. This pathway has very recently been brought into light by the pioneering work of Barker and his colleagues. They have shown that the pernicious consequences of low birth weight actually extend far beyond the disabilities related to poor nutritional status in adult life. Their work has identified a link between low birth weight and a number of adult ailments that had not been traditionally linked to undernutrition as such, viz. hypertension, diabetes (type 2), cardiovascular diseases, obstructive lung disease, renal damage, and some forms of cancer.³⁰ This pathway actually leads to greater health risk for those low birth weight babies that avoid nutritional stress beyond the first year of life. Adults who were low birth weight babies, but are enjoying relative affluence in their adult life are more prone to these diseases than those who remain undernutritional stress even as adults. Moreover, if they also experience overnutrition in late childhood, then they are in even greater danger to contract these diseases in adult life than those who remained undernourished throughout their childhood (Eriksson et al., 2001). This is a quite distinct pathway which leads from fetal deprivation not to diseases of the old regime, but instead, to those of the new regime that are emerging in the process of health transition driven by fertility decline and economic affluence.

The epidemiological work demonstrating the association between low birth weight and chronic adult diseases was initially based on data from developed countries (Great Britain, Sweden and Finland), but more recent research has confirmed the existence of the link between low birth weight and cardiovascular diseases for developing countries as well, including India (Barker et al., 1995; Yajnik, 2001). The most widely held hypothesis explaining this link is due to Barker. Known as the ‘fetal origin of adult disease’ hypothesis, it postulates that the same nutritional deprivation that causes intrauterine growth retardation leading to low birth weight also sows the seeds of ailments that afflict adults. When faced with inadequate access to nutritional resources (owing to maternal undernutrition), the fetus undergoes an adaptive mechanism in its physiology as well as metabolic processes. This adaptation serves the fetus well by helping it cope with nutritional stress in utero, but the same adaptation also hampers its ability to cope with the conditions of relative plenty that might be encountered in later life. Hence, there is a greater risk of contracting chronic diseases for low birth weight babies who grow up to be relatively affluent adults.

There are, however, also contrary arguments to this use of Barker’s findings. Some have argued that the epidemiological studies demonstrating the association between low birth weight and adult diseases have not taken sufficient care to control for the effects of such confounding factors as genetic predisposition and social behaviour, that also bear on the risk to chronic diseases.³¹ One difficulty in this regard is that a proper testing of the Barker hypothesis requires longitudinal data, tracing the history of adults back to their fetal status.

³⁰ See, especially, Barker (1993, 1995, 1998) and Barker (1993). For other types of evidence on the association between early deprivation and adult health, see Costa (1993), Komlos (1998) and Cameron (2002).

³¹ Some of these issues are discussed in ACC/SCN (2000b).

Such data are not at all in plentiful supply, and the few data sets that do exist do not often contain adequate information on the confounding factors, primarily because these data sets were assembled in the first place for very different purposes. Many of the criticisms are being addressed, however, by the newer studies, which confirm that the Barker hypothesis survives even after allowing for the confounding factors. Much work remains to be done of course before the association is conclusively established, but the accumulated evidence is already large enough to suggest a genuine and strong association between fetal deprivation and chronic diseases.³²

Much scientific work also remains to be done to clarify the processes of biological adaptation that are supposed to be initiated by fetal deprivation and to explain quite how these processes make a person prone to various chronic diseases. Until the nature of these mechanisms is fully understood by scientists working in the laboratory, the sheer strength of epidemiological evidence may not suffice to convince the scientific community fully. Yet, the required scientific activities have already begun, and a number of plausible mechanisms have been suggested.³³ All this makes the Barker hypothesis a highly credible one.

By invoking this hypothesis, it is possible to argue that the phenomenon of exceptionally high levels of cardiovascular diseases in South Asia referred to earlier is a consequence of the exceptionally high prevalence of low birth weight in this region. While poverty remains widespread in South Asia, it is also true that significant proportions of the population are escaping the poverty trap, especially in the last three decades. Many of those who have managed to escape poverty within a single generation were born as low birth weight babies, because their mothers belonged to a generation that was steeped in poverty and mired in a culture of gender inequality. They are the ones who now find themselves highly susceptible to the diseases of the new regime. This problem will of course exist in all developing countries where some people are escaping poverty within a single generation, but it happens to be particularly acute in South Asia because gender inequality in nutrition has been especially severe in this region.³⁴

5. The neglect of women and its many penalties

The penalties of gender bias are indeed extensive. Not only is gender inequality itself—first and foremost—a major social failure and transgression, it also leads to many other adverse consequences, such as:³⁵

- increasing the *mortality* rates, of women in particular, but also of others (child mortality relates directly to women's education and literacy);

³² See, among others, Barker (1998), Eriksson et al. (2001) and Yajnik (2001).

³³ See, for example, Ozanne and Hales (2001), Harding et al. (2001), Seckl et al. (2001), and other studies presented at the First World Congress on Fetal Origins of Adult Disease held in Mumbai, India, during 2–4 February 2001.

³⁴ This causal inference must be taken as tentative at this stage, however, given the scientific queries that still surround the Barker hypothesis and the uncertain nature of the data on the prevalence of diseases. Much more scientific and quantitative empirical work will have to be done before it can be asserted with force. But, as we have tried to argue here, sufficient ground already exists to make this inference at least a plausible one.

³⁵ See, for example, Osmani (1992, 1997) and Sen (1983, 1984, 1985, 1992a,b, 1996, 1999, 2000).

- contributing to high *fertility* rates (indeed women's empowerment is one of the strongest influences in cutting birth rates);
- limiting the strength and coverage of *economic progress* (many of the successful experiences of rapid economic activities have crucially depended on women's initiative, particularly in East and South-east Asia, but increasingly also in other countries, including Bangladesh);
- impairing *political participation* and the practice of democracy (there is much evidence, not least from South Asia, that a greater role of women in grass-root politics can help to change the agenda of social discourse).

To these penalties of gender inequality, we have to add the others on which this paper has focused, including the health of mothers and babies, the nourishment of children and their development, and the morbidity and survival of adults—an influence that seems to continue many decades later. In other words, what begins as a neglect of the interests of women ends up causing adversities in the health and survival of all in the developing world. Perhaps men can manage to get out of some of these “troubles” in the near future and even “continueth” somewhat longer by being a little more just to women.³⁶ The extensive penalties of neglecting women's interests rebounds, it appears, on men with vengeance.

Important as it is, this is not only a matter of justice, however. The analysis presented above has serious implications for hard-nosed economic calculus. We have noted earlier how the developing world has to deal simultaneously with two very different regimes of diseases existing side by side with almost equal ferocity. So long as the overlapping transition remains, the developing countries will be faced with a serious resource constraint in dealing with their health problems. Our analysis suggests that the problem will be the more severe for those among the developing countries where gender inequality is more prevalent. We have shown that gender inequality exacerbates the old regime of diseases among the less affluent through the pathway of childhood undernutrition. Simultaneously it also aggravates the new regime of diseases among the relatively more affluent (but whose mothers belonged to a less affluent generation) through the pathway of the ‘Barker hypothesis’. Gender inequality thus leads to a double jeopardy—simultaneously aggravating both regimes of diseases and thus raising the economic cost of overlapping health transition. Undertaking social reforms aimed at eliminating gender inequality may turn out to be the most cost-effective method of preventing this double jeopardy.

Acknowledgements

The authors are grateful for helpful comments on an earlier version of this paper by the participants of the First World Congress on the Fetal Origins of Adult Disease, held in Mumbai, India, during 2–4 February 2001, a workshop held at the Trinity College, Cambridge, on 4 July 2001 and a conference on Economics and Human Biology held at the University of Tübingen, Germany, during 10–13 July 2002. The usual disclaimer, however, applies.

³⁶ A fuller quote from the Book of Job referred to above states: “Man that is born of a woman is of few days, and full of trouble. He comes forth like a flower, and is cut down: he fleeth also as a shadow, and continueth not”.

References

- ACC/SCN, 2000a. Fourth Report on the World Nutrition Situation. United Nations Administrative Committee on Coordination, Sub-Committee on Nutrition. World Health Organisation, Geneva.
- ACC/SCN, 2000b. Low Birth weight. Nutrition Policy Paper No. 18. United Nations Administrative Committee on Coordination, Sub-Committee on Nutrition. World Health Organisation, Geneva.
- Bardhan, P., 1974. On life and death questions. *Economic and Political Weekly* 9, Special Number.
- Bardhan, P., 1984. Land, Labor and Rural Poverty. Columbia University Press, New York.
- Barker, D.J.P., 1993. Intrauterine growth retardation and adult disease. *Current Obstetrics Gynaecology* 3, 200–206.
- Barker, D.J.P., 1995. Fetal origins of coronary heart disease. *British Medical Journal* 311, 171–174.
- Barker, D.J.P., 1998. Mothers, Babies and Diseases in Later Life. Churchill Livingstone, London.
- Barker, D.J.P., Gluckman, P.D., Godfrey, K.M., Harding, J.E., Owens, J.A., Robinson, J.S., 1995. Fetal nutrition and cardiovascular disease in adult life. *Lancet* 341, 938–941.
- Basu, A., 1989. Is sex discrimination really necessary for explaining sex differentials in childhood mortality. *Population Studies* 43(2).
- Basu, A., Jeffery, R., 1996. Girls' Schooling, Women's Autonomy and Fertility Change in South Asia. Sage, London.
- Battaglia, F.C., Simmons, M.A., 1979. The low-birth-weight infant. In: Falkner, F., Tanner, J.M. (1979), *Human Growth*, vol. 1. Plenum Press, New York.
- Caldwell, J.C., 1993. Health transition: the cultural, social and behavioural determinants of health in the third world. *Social Science and Medicine* 36(2).
- Cameron, N., 2002. Physical Growth in a Transitional Economy: The Aftermath of South African Apartheid. Paper Presented in the Conference on Economics and Human Biology held at the University of Tubingen, Germany, 10–13 July 2002.
- Chen, L.C., Huq, E., D'Souza, S., 1981. Sex Bias in the family allocation of food and health care in rural Bangladesh. *Population and Development Review* 7.
- Chen, L.C., Kleinman, A., Ware, N. (Eds.), 1994. *Health and Social Change in International Perspective*. Cambridge University Press, Cambridge.
- Coale, A.J., 1991. Excess female mortality and the balance of the sexes: an estimate of the number of missing females. *Population and Development Review* 17.
- Costa, D., 1993. Height, weight, wartime stress, and older age mortality: evidence from the union army records. *Explorations in Economic History* 30 (4), 424–449.
- Dasgupta, P., 1993. *An Inquiry into Well-being and Destitution*. Clarendon Press, Oxford.
- de Onis, M., Blossner, M., Villar, J., 1998. Levels and patterns of intrauterine growth retardation in developing countries. *European Journal of Clinical Nutrition* 52 (Suppl).
- Drèze, J., Sen, A., 1989. *Hunger and Public Action*. Clarendon Press, Oxford.
- Drèze, J., Sen, A., 1995. *India: Economic Development and Social Opportunity*. Oxford University Press, Delhi.
- Eriksson, J.G., Forsen, T., Tuomilehto, J., Osmond, C., Barker, D.J.P., 2001. Early growth and coronary heart disease in later life: longitudinal study. *British Medical Journal* 322 (April).
- FAO, 1996. *The Sixth World Food Survey*. Food and Agricultural Organisation, Rome.
- Harding, R., Cock, M.L., Maritz, G.S., Louey, S., Joyce, B., Wignarjah, D., 2001. Lung Function and Structure Following Intra-Uterine Growth Restriction. Paper Presented at the First Congress on Fetal Origins of Adult Disease held in Mumbai, India, 2–4 February 2001.
- Harriss, B., 1990. The Intrafamily Distribution of Hunger in South Asia. In: Dreze, J., Sen, A. (Eds.), *The Political Economy of Hunger*, vol. I. Clarendon Press, Oxford.
- Harriss, B., Watson, E., 1987. The Sex Ratio in South Asia. In: Momsen, J.H., Townsend, J. (Eds.), *Geography of Gender in the Third World*. Butler and Tanner, London.
- Henriksen, T., 1999. Foetal nutrition, fetal growth restriction and health later in life. *Acta Paediatrica* 429S, 4–8.
- Kanbur, R., Haddad, L., 1990. How serious is the neglect of intrahousehold inequality? *Economic Journal* 100.
- Komlos, J., 1998. Shrinking in a growing economy? The mystery of physical stature during the industrial revolution. *Journal of Economic History* 58 (3), 779–802.
- Klasen, S., 1994. Missing women reconsidered. *World Development* 22.
- Kramer, M., 1987. Determinants of low birth weight: methodological assessment and meta-analysis. *Bulletin of the World Health Organisation* 65, 663–737.

- Kramer, M., 1998. Socio-economic determinants of intrauterine growth retardation. *European Journal of Clinical Nutrition* 52 (S1), S29–S33.
- Kynch, J., 1985. *How Many Women are Enough? In Third World Affairs 1985*. Third World Foundation, London.
- Kynch, J., Sen, A., 1983. Indian women: well-being and survival. *Cambridge Journal of Economics* 7.
- McKiegie, P.M., Marmot, M.G., 1988. Mortality from coronary heart diseases in Asian communities in London. *British Medical Journal* 297 (October).
- Martorell, R., Ramakrishnan, U., Schroeder, D.G., Melgar, P., Neufeld, L., 1998. Intrauterine growth retardation, body size, body composition and physical performance in adolescence. *European Journal of Clinical Nutrition* 51 (S1), S43–S53.
- Murray, C.J.L., Lopez, A.D. (Eds.), 2002. *The Global Burden of Disease*. Harvard School of Public Health/World Health Organisation/World Bank, New York/Geneva/Washington, DC.
- Osmani, S.R., 1997. Poverty and Nutrition in South Asia, in ACC/SCN, *Nutrition and Poverty*. Nutrition Policy Paper No. 16. WHO, Geneva, pp. 23–51 (also published as the First Abraham Horwitz Lecture by the United Nations ACC/SCN. WHO, Geneva, 1997).
- Osmani, S.R. (Ed.), 1992. *Nutrition and Poverty*. WIDER Studies in Development Economics. Clarendon Press, Oxford.
- Osmani, S.R., Bhargava, A., 1998. Health and nutrition in emerging Asia. *Asian Development Review* 16(1).
- Ozanne, S.E., Hales, C.N., 2001. The Fetal Origins of Type 2 Diabetes. Paper Presented at the First Congress on Fetal Origins of Adult Disease held in Mumbai, India, 2–4 February 2001.
- Prada, J., Tsang, R., 1998. Biological mechanisms of environmentally induced causes in IUGR. *European Journal of Clinical Nutrition* 52 (S1), S21–S28.
- Ramalingaswami, V., Jonsson, U., Rohde, J., 1996. “The Asian Engima” in UNICEF. *The Progress of Nations 1996*, New York.
- Seckl, J.R., Welberg, L., Holmes, M., 2001. Glucocorticoids and Programming of the Brain. Paper Presented at the First Congress on Fetal Origins of Adult Disease held in Mumbai, India, 2–4 February 2001.
- Sen, A., 1983. Economics and the family. *Asian Development Review* 1.
- Sen, A., 1984. *Resources, Values and Development*. Harvard University Press/Blackwell Scientific Publications, Cambridge, MA/Oxford.
- Sen, A., 1985. *Commodities and Capabilities*. North-Holland/Oxford University Press, Amsterdam/Delhi, 2000.
- Sen, A., 1988. Africa and India: What Do We Have to Learn from Each Other? In: Arrow, K.J. (Ed.), *The Balance between Industry and Agriculture in Economic Development: Basic Issues*, vol. I. Macmillan, London.
- Sen, A., 1989. Women’s Survival as a Development Problem, *Bulletin of the American Academy of Arts and Sciences*, 43 shortened version published as *More than a Hundred Million Women Are Missing*. *New York Review of Books*, Christmas Number, 1993.
- Sen, A., 1990. Gender and Cooperative Conflict. In Tinker, I. (Ed.), *Persistent Inequalities*. Oxford University Press, New York.
- Sen, A., 1992a. Inequality Reexamined. Harvard University Press/Clarendon Press, Cambridge, MA/Oxford.
- Sen, A., 1992b. Missing women. *British Medical Journal* 304 (March).
- Sen, A., 1996. Fertility and coercion. *University of Chicago Law Review* 63.
- Sen, A., 1999. *Development as Freedom*. Knopf/Oxford University Press, New York/Oxford.
- Sen, A., 2000. The population problem and gender inequality. *The Nation* 24–31 (July).
- Sen, A., Sengupta, S., 1983. Malnutrition of rural children and the sex bias. *Economic and Political Weekly* 19 (Annual Number).
- Sengupta, S., Gazdar, H., 1996. Agrarian Politics and Rural Development in West Bengal. In: Drèze, J., Sen, A. (Eds.), *Indian Development: Selected Regional Perspectives*. Oxford University Press, Delhi.
- Shetty, P.S., James, W.P., 1994. Body Mass Index: A Measure of Chronic Energy Deficiency in Adults. *Food and Nutrition Paper No. 56*. Food and Agricultural Organisation, Rome.
- Smith, L., Haddad, L., 2000. Explaining Child Nutrition in Developing Countries: A Cross-Country Analysis. *Research Report No. 111*. International Food Policy Research Institute, Washington, DC.
- UNDP, 1984. *Human Development Report 1994*. United Nations Development Programme, New York.
- Waldron, I., 1976. Why do women live longer than men? *Social Science and Medicine* 10, 349–362.
- Yajnik, C., 2001. Fetal Origins of Cardiovascular Risk in Developing Countries. Paper Presented at the First Congress on Fetal Origins of Adult Disease held in Mumbai, India, 2–4 February 2001.