Does Malnutrition Affect Fecundity? A Summary of Evidence

John Bongaarts

Summary. Moderate chronic malnutrition has only a minor effect on fecundity (reproductive capacity), and the resulting effect on fertility (actual reproduction) is very small. Among the fecundity components examined here in noncontracepting populations, age at menarche and the duration of lactational amenorrhea appear to be the ones most affected by malnutrition. But from neither of those effects can a difference in fertility of more than a few percent be expected between poorly and well-nourished women in developing countries.

That malnutrition has an effect on human reproductive capability is suggested by several research findings. For example, by the end of their reproductive years well-nourished Hutterite women have given birth, on the average, several more times than relatively poorly nourished women in developing countries or in historical populations. It is also well established that famines cause reductions in birthrates. These facts provide support for the current hypothesis that malnutrition impairs fecundity, the ability to reproduce (*1*, *2*). But the strength of this relationship and its demographic significance are controversial issues, which have recently become a focus of a number of investigations (*3*). A strong link between nutrition and fecundity would have important implications, especially for food aid programs for the developing world. If improving nutrition in those countries increased their birthrates, it would exacerbate an already serious population growth problem.

The object of this article is to review existing evidence for the effect of malnutrition on human reproductive capability and to make an assessment, even if a rather crude one, of its demographic impact. Because findings vary with the degree of malnutrition, a distinction will be made between the chronic moderate malnutrition that prevails in the poorer classes of many developing countries and the severe malnutrition resulting from famine and starvation. The discussion will focus on the effects in females; the literature contains relatively little information about the effects in males.

Evidence from Fertility Differentials

Inferences about the effect of chronic malnutrition on the ability to reproduce are often made from fertility differences among populations which differ with respect to nutrient intake. Such inferences, however, are not reliable, because the ability to reproduce (fecundity) is not necessarily, and in fact is only rarely, reflected in actual reproductive performance (fertility). If one were to collect fertility and nutrition data for a large number of populations from around the world, one would find a strong negative correlation between nutrition and fertility. Nutrient intake is highest in the Western industrialized nations with only about two children per woman at the end of the reproductive years, whereas malnutrition is most prevalent in the poorest underdeveloped countries, where completed fertility is typically six to seven births per woman. One can obviously not conclude from these data that good nutrition impairs fecundity, because the low fertility in the developed countries is the consequence of voluntary fertility control.

In general, the fertility of a population is determined both by its fecundity, which sets an upper limit to fertility, and by a number of fertility-inhibiting behavioral factors, which can reduce actual fertility to a fraction of this upper limit. The most important behavioral factors are (i) late marriage and marital disruption, (ii) deliberate birth control by contraception, abstention, or induced abortion, and (iii) breast-feeding, which can delay the return of ovulation after a birth (*4*). One or more of these factors reduces fertility to well below its potential level in all known populations. This fact greatly complicates the analysis of the effect of nutrition on fecundity.

In order to circumvent this problem, the analysis of fertility differentials is usually confined to populations in which contraception and induced abortion are virtually absent. However, neglect of the other behavioral factors can still make it difficult to draw conclusions. An example is provided by the often-cited comparison of Hutterite fertility with the fertility of poor populations in underdeveloped countries such as Bangladesh. Hutterite women who reached the end of the child-bearing years in 1950 had an average of 9.5 childbirths (*5*), women in Bangladesh in the early 1970's only about 7 (*6*). Since the use of contraception or induced abortion is negligible in both populations, it has been suggested that this fertility difference is due to the high prevalence of malnutrition in Bangladesh (*2*). While that would appear to be a plausible explanation, there are several counterexamples. In the rich and better-nourished populations of Kuwait and Saudi Arabia, where contraception is virtually absent and marriage is early and universal, fertility is approximately the same as in Bangladesh (*6*). Particularly instructive is the case of the Amish population, which is culturally very similar to the Hutterites. Both populations consist of descendants of Swiss settlers in the United States, and both enjoy a high standard of living and good nutrition. Most aspects of daily life are under strict social and religious control in both societies, and there is minimum contact with the world outside their agricultural communities. Fertility is natural, that is, without deliberate control, yet Amish fertility is only 6.3 births per woman (*7*).

The existence of extremely high marital fertility in a few historical populations is also difficult to reconcile with the nutrition hypothesis. There is good evidence of a low level of nutrition in the laboring classes in mid-19th-century England, but the mean birth interval—an inverse indicator of marital fertility—was short, 20 to 24 months, about the same as among the Hutterites (*2*, *8*, *9*). Similarly, age-specific marital fertility rates of the 18th-century Canadian population were very close to those of the Hutterites (*10*), despite a probably substantial difference in nutrition levels.

Clearly, these simple comparisons of

The author is an associate of the Center for Policy Studies, The Population Council, New York 10017.

Table 1. Measures of general fertility, marital fertility, and determinants of fertility for the Hutterites (marriages of 1921 to 1930), Bangladeshi (in 1970 to 1975), and three villages of Ile-de-France (18th century). The total general fertility rate is the average number of births per woman at the end of the reproductive years. The total marital fertility rate is the average number of births per woman married at age 20 and remaining married to the end of the reproductive years.

Population	Total general fertility rate (births per woman)	Marital fertility		Determinants of fertility		
		Total marital fertility rate (births per woman)	Mean birth interval (months)	Median age at marriage	Contra-ceptive practice	Mean duration of lactational amenorrhea (months)
Hutterites	9.5	10.9	24.0	22.0	Absent	6.0
Bangladesh	7.2	6.8	37.4	17.0	Very low	17.9
Ile-de-France	6.1	10.1	26.0	25.5	Very low	8.6*

*Estimated by subtracting the mean duration of the interval between marriage and first birth from the mean duration of the interval between first and second births.

the fertility of different populations do not allow the conclusion that there is a substantial effect of nutrition on fecundity. What then explains the high fertility of the Hutterites? To demonstrate that the behavioral factors are responsible for the fertility differentials requires a detailed analysis of the fertility determinants, which is not possible in many populations because of lack of data. The needed data are available, however, for the well-nourished Hutterites and the poorly nourished population of Bangladesh.

Table 1 summarizes these data. The estimates of general fertility indicate that, on the average, Hutterite women have 2.3 more children in their reproductive lifetimes than women in Bangladesh (*5, 6*). This fertility difference increases if one controls for the Hutterites' later age at marriage. The second column in Table 1 presents fertility rates standardized for age at marriage. The total marital fertility rate given equals the average number of births women in these populations would have if they married at exactly age 20 and remained married throughout the reproductive period. This rate is 39 percent less in Bangladesh than among the Hutterites (*10, 11*). The mean birth intervals are correspondingly shorter in the Hutterites (*8, 9*) than in the Bangladeshi (*12*).

The problem is now reduced to finding the causes of the difference of 13.4 months between the mean birth intervals of the two populations. It is evident from the last column of Table 1 that the primary factor is the difference in the duration of lactational amenorrhea. The difference of 11.9 months (*8, 13*) accounts for 89 percent of the birth interval difference of 13.4 months. The remaining 11 percent, or 1.5 months, may be due to the seasonal absence of husbands (*14*), a slightly lower frequency of intercourse, and the use of contraception or abortion by a small number of couples in Bangladesh.

The difference in lactational amenorrhea is not surprising in view of the difference in the breast-feeding practices of the two populations. Among the Hutterites lactation is relatively short and supplemental feeding is introduced early in an infant's life, whereas in Bangladesh most women nurse their infants for a long time—often until the next pregnancy occurs (*15*). Since the duration and pattern of breast-feeding are the primary determinants of the duration of amenorrhea (as will be shown below), the Bangladeshi women would be expected to have longer amenorrhea and therefore lower marital fertility than the Hutterite women.

For comparison, Table 1 also gives estimates of fertility rates and their determinants for the 18th-century population of Ile-de-France (*16, 17*). The general fertility rate of this population is substantially below that of the Hutterites and even slightly lower than in Bangladesh, the apparent cause being the relatively late age of women at marriage (*17*). The marital fertility rate, as well as the mean birth interval and the duration of lactational amenorrhea, are almost the same as among the Hutterites. Thus the fertility differences between the Hutterites and the populations of Bangladesh and Ile-de-France can be attributed to the various behavioral factors that inhibit fertility. There is consequently no need to hypothesize a substantial difference in fecundity between these populations.

In the evidence reviewed up to this point no direct estimates of fecundity have been available. However, in one recent study fecundity in two groups of populations is estimated with a reproductive model that calculates a fecundity rate. This rate is defined as the average number of times women would give birth over their reproductive lifetimes in the absence of celibacy, contraception, induced abortion, and lactation. It comes to 15.4 in a group of developed countries and 15.2 in a group of poor, less-developed countries (*4*), a difference that is not statistically significant. Since there is a significant difference in nutritional intake, this study also suggests that moderate chronic malnutrition has little effect on fecundity in the underdeveloped countries.

A more detailed understanding of the link between malnutrition and fecundity can be gained from an examination of the various biological factors that determine fecundity. The following factors can be identified: (i) age at menarche, (ii) age at menopause, (iii) prevalence of permanent (primary) sterility, (iv) ovulation-inhibiting effect of breast-feeding, (v) regularity of ovulation (among menstruating women) and the quality and quantity of sperm, and (vi) probability of intrauterine death. A population or group of women can be considered to have relatively high fecundity if the values of factors (ii) and (v) are relatively high and the values of the other factors are relatively low. Evidence for an effect of chronic malnutrition on each of these factors will now be reviewed briefly and, where appropriate, the impact on fertility will be discussed.

Menarche

In the contemporary Western world the average age at menarche is about 13 years (*16, 18–21*). Averages in developing countries are typically higher and vary substantially—for example, 12.4 in Cuba (*22*), 13 to 14 in India (*20, 23*), 13.4 in Sri Lanka (*24*), 15.0 among the South African Bantus (*25*), 15.7 in Bangladesh (*26*), and 18.8 among the Bundi in New Guinea (*22*). Estimates for 19th-century European populations are also high—around 16 years (*21*). Much effort has been devoted to analyzing the determinants of menarche, but there is still substantial disagreement about the relative importance of various biological, environmental, and socioeconomic factors (*18, 19*).

Most investigators believe, however, that nutrition has a substantial effect.

That conclusion is based on four types of direct and indirect evidence. First, a direct link between nutritional intake and age at menarche was found in a U.S. study in which well-nourished girls reached menarche 2 years earlier than undernourished girls (*27*). Similarly, an Indian study concluded that girls whose diets were higher in calories and proteins had earlier menarche (*28*). Second, nutritional status as measured by anthropometric indicators is correlated with age at menarche. A number of reports from different societies has established that the probability of reaching menarche by a given age is positively related to body size and weight (*21, 26, 27, 29–31*). On the basis of this evidence, Frisch has developed a procedure for estimating a minimum weight for height necessary for the onset of menarche (*30*), but this method has been questioned (*32, 33*). Third, in Western societies where there are relatively reliable historical data on menarche, a decline in age of menarche of about 3 years has taken place since the end of the last century (*19, 21*). This decline is associated with an increase in body size and an improved diet. Fourth, socioeconomic status and age at menarche are negatively related in a number of countries. Differences ranging from a few months to about 2 years have been found between urban and rural populations and between high and low income groups (*19, 24, 25, 34, 35*).

Age at menarche signals the beginning of potential childbearing, but actual reproduction starts at marriage. The mean age at marriage is almost always higher than the mean age at menarche, the difference ranging from about 2 years in some traditional societies to more than 10 years in a number of contemporary European populations. In populations where the mean age at marriage is near 20 or higher, one can hardly expect a fertility effect from nutritional variations in age at menarche, but this fertility effect is quite small even if marriage takes place shortly after menarche and ages at the two events are correlated.

A simple numerical exercise can demonstrate this. Let us assume that in a hypothetical, but not atypical, poorly nourished population the mean age at menarche is 15 years and that a large improvement in nutrition can lower it to 13 years. Let us further assume that marriage is very early and that there is a significant correlation between menarche and marriage so that the mean age at marriage will also decrease—say from 17 to 16 years. This 1-year reduction in age at marriage will lengthen the actual reproductive life-span by about the same amount, thus increasing completed fertility. With a marital fertility rate of 250 per 1000 for 16-year-old women, this additional reproductive time would add 0.25 births per woman on average. Since average completed fertility is typically six or seven births per woman, this implies a fertility increase of about 4 percent. A decrease in adolescent subfertility adds less than 1 percent to this estimate (*36*). Clearly, substantial changes in age at menarche following improvements in nutrition can be expected to raise fertility by at most a small percentage. The insensitivity of fertility to modification in the age at menarche has also been confirmed by computer simulations (*37*).

Menopause

In developed societies the mean age at menopause ranges from 47 to 50 years and the median is 1 or 2 years higher (*20, 38*). The few reported estimates from developing countries are much less consistent: median ages of 44 in India (*39*) and 43.7 in a malnourished population in New Guinea (*20*) are among the lowest observed anywhere. Mean ages of 47.4 in rural Ceylon (*24*) and 47.5 in poorly nourished mid-19th-century England (*2*) are nearly the same as in well-nourished populations of today. A relatively high mean of 50.7 years has been found among the Bantu in South Africa (*40*). The available evidence regarding the determinants of age at menopause is conflicting and inconclusive. A few studies report a time trend in age at menopause or an effect of nutrition or socioeconomic status (*20, 24, 38, 41*), but other investigations fail to find the same relationships (*38, 42, 43*). These confusing results are probably due in part to methodological problems, including inappropriate statistical analyses, recall errors in retrospective data, and misreporting of age (*20, 38, 43*).

No conclusive evidence regarding nutritional variations in age at menopause is provided by these studies. Even if there is such an effect, it would be of demographic interest only if the timing of a woman's final childbirth—and hence her fertility—were significantly affected. The small amount of available evidence suggests very little effect of nutrition on the mean age at last birth. This mean age is 40.9 among the well-nourished Hutterites (*9*) and—contrary to expectations—slightly higher (41.7 years) in a poorly nourished mid-19th-century English population (*2*). The use of contraception and induced abortion was virtually absent in both populations. The mean age at last birth is also close to 40 years in a number of other historical natural-fertility populations (*44*).

Prevalence of Permanent Sterility

Only 3 percent of the married Hutterite women had never borne a child by the time they reached age 50 (*5*). It has been suggested that that is an unusually low rate of permanent sterility and that higher sterility can be expected in poorly nourished populations (*2*). Recent demographic surveys in a number of poor developing countries do not support that view. For example, in Bangladesh 2.1 percent of married women aged 45 or over, and in Pakistan and Nepal 2 and 2.6 percent respectively, have never given birth (*45*).

Inhibition of Ovulation by Lactation

The term lactation amenorrhea is often used to refer to the temporary absence of menses postpartum, because it is now well established that breast-feeding is the principal determinant of the duration of that amenorrhea. In the absence of breast-feeding the menses return shortly after birth in well-nourished populations (*46–48*) as well as in poorly nourished ones (*14, 49–51*). As the duration of breast-feeding increases so does the amenorrheic interval—approximately one additional month of amenorrhea for each 2-month increment in breast-feeding (*16, 52*). With long lactation mean amenorrheic intervals of 1 to 2 years are observed, not only in the poorest developing countries (*14, 53–55*) but also in the United States, where a group of women who practiced "natural" (unrestricted) nursing experienced an average of 14.6 months of amenorrhea (*56*).

An extensive set of data from the WHO Collaborative Study on Breast-feeding (25 subpopulations in nine countries) showed that, at any given time, postpartum variation in proportions of breast-feeding explained, on the average, 85 percent of between-population variance in the proportions of menstruating women (*57*). Other studies have found high correlations between mean duration of breast-feeding and of amenorrhea when comparing populations (*52, 58*) or subpopulations within countries (*46–49, 55, 59*). On the individual level the correlation is somewhat lower. The most plausible explanation for this—aside from measurement error—is that women differ not only with respect to how long they breast-feed but also with respect to their patterns of breast-feeding (*60, 61*). It has been shown that women who fully breast-feed have a lower probability of resumption of menses than women whose infants receive supplemental food such as fluids by bottle or solids (*48, 49, 54, 62*). The ovulation- and menstruation-inhibiting effect of breast-feeding and also the differential effect of breast-feeding patterns are believed to be due to a neurally mediated hormonal reflex system initiated by the suckling stimulations of the breast nipple (*63, 64*).

The question of interest here is whether maternal malnutrition affects amenorrhea independent of breast-feeding practices. Recent studies in relatively poorly nourished rural populations in Bangladesh and Guatemala provide tentative estimates of the importance of moderate malnutrition. In one study in Bangladesh, women were divided into three nutritional status groups on the basis of weight: less than 38.5 kilograms, between 38.5 and 42.4 kg, and over 42.4 kg. The observed mean duration of amenorrhea in these groups was 17.9, 17.5, and 16.8 months, respectively (*13*). There is a decline with increasing weight, but the differences are not statistically significant. The relation between duration of amenorrhea and other anthropometric measures was also not statistically significant.

In another study in Bangladesh nutritional status was estimated from measures of weight for height. The median duration of amenorrhea in the low, medium, and high nutritional status groups was 21.2, 20.4, and 20.2 months, respectively (*54*). There was no evidence of a threshold of weight for height necessary for the resumption of menses postpartum (*65*). It should be noted that these analyses do not control for duration of lactation. However, it is unlikely that this substantially affects the results, because women in rural Bangladesh usually breast-feed their infants until the next pregnancy occurs, and a large majority are still lactating when their menses return.

These results are confirmed in a study in Guatemala in which women were again divided into three nutritional status groups on the basis of anthropometric measures (*66*). The low and high nutritional status groups had mean weights of 43.7 and 55.6 kg and mean amenorrhea duration of 14.8 and 13.2 months, respectively. The difference in amenorrhea was statistically significant and slightly larger than in Bangladesh, possibly because the Guatemalan population is more heterogeneous with respect to nutritional status. Data on total caloric intake were also available, and their relation to postpartum amenorrhea is summarized in Table 2. This table allows direct comparison of the relative importance of lactation and nutrition as determinants of the duration of amenorrhea. Lactation is clearly the primary determinant. There is a fairly consistent, but not statistically significant, difference of the order of 1 month between the amenorrheic periods of women with high and low caloric intake.

Table 2. Mean duration of postpartum amenorrhea (months) in groups differing in caloric intake and in duration of lactation (number in parentheses). Low caloric intake = < 1309 calories a day, middle = 1309 to 1630 calories, high = > 1630 calories. [Data from (*67*)]

Daily caloric intake	Duration of lactation (months)			
	7 to 12	13 to 18	19 to 24	25 or more
Low	6.54 (13)	12.03 (35)	16.44 (45)	19.93 (14)
Middle	6.31 (13)	11.64 (33)	15.47 (57)	18.90 (10)
High	5.47 (14)	11.13 (30)	14.60 (47)	19.64 (11)
High-low difference	1.07	0.90	1.84	0.29

These findings seem to be inconsistent with the results of a Mexican study (*68, 69*) of two groups of women, one with and one without dietary supplementation. The group with the supplement had much shorter postpartum amenorrhea than the control group. Wray (*70*) has proposed a plausible explanation for this finding. It appears that the infants of many of the supplemented mothers also received dietary supplements. As a result, these infants needed less milk from their mothers, and the decreased suckling shortened the period of amenorrhea independent of the mother's nutritional status. The validity of this explanation appears to be confirmed by new observations from the same population which show that a small group of supplemented women whose infants receive no supplements had a mean amenorrheic interval of virtually the same duration as the unsupplemented mothers (*71*).

The small differences between nutritional status groups in duration of amenorrhea are not necessarily caused by a direct physiological effect of malnutrition on the mother. It is possible that unmeasured differences in pattern of breast-feeding between the poorly and well-nourished women in these populations are responsible. For example, if malnourished mothers have less food available for supplementing infant diets, or are later in introducing supplementation, their infants will suckle more and thus prolong amenorrhea.

The effect on fertility of variations in amenorrhea duration are easily estimated from the resulting proportional changes in the mean birth interval. The estimates of the studies in Bangladesh and Guatemala are in fair agreement and suggest a difference of approximately 1 month in the amenorrheic intervals of the high and low nutritional status groups. With a typical mean birth interval of $2^1/_2$ to 3 years, a 1-month change would yield about a 3 percent difference in the marital fertility of the two extreme nutritional status groups.

Regularity of Ovulation and Quality and Quantity of Sperm

Since it is virtually impossible to estimate directly the regularity of ovulation and the quality and quantity of sperm on a population basis, it is necessary to rely on indirect measures such as the rate of conception among menstruating women. A conception rate is determined also by the frequency of intercourse, but any effect that malnutrition would have on any of these three determinants would be expected to be negative. One can therefore be reasonably sure, if no overall effect of malnutrition on conception rate is found, that it is not because of compensatory effects on any of them.

A convenient indicator of the rate of conception is the mean waiting time to conception. This is simply measured as the number of months between the occurrence of the first postpartum menses—a good indicator of the resumption of ovulation—and the next conception. The two prospective studies of poorly nourished women in Bangladesh and Guatemala discussed in the previous section were specifically designed to test the effect of nutrition status on the mean waiting time to conception (conceptions ending in live births in the Guatemalan case). In both populations contraception and induced abortion are very little used. In Bangladesh the mean waiting times in groups of women with low (< 38.5 kg), medium (38.5 to 42.4 kg), and high (> 42.5 kg) weights were 11.3, 10.7, and 10.0 months, respectively. The differences were not statistically significant after control for the confounding effect of age and absences of husband (*13*). There was no correlation between other anthropometric measures and mean waiting time. In Guatemala the low, medium, and high nutritional status groups had waiting times of 6.4, 6.1, and 6.3 months, respectively (*66*). Compared with that of other, mostly better-nourished, populations, the mean waiting time is above average but not unusually long in Bangladesh and relatively short in Guatemala (*12, 16*).

Intrauterine Mortality

Although maternal nutrition is one of the determinants of an infant's birth weight (*72, 73*), good evidence for a link between moderate malnutrition and fetal mortality does not exist. The study of this subject is hampered by severe methodological problems in measuring the prevalence of intrauterine mortality, especially during the early months of gestation (*16*). As a consequence, estimates of fetal mortality rates vary widely among populations, even if one restricts comparisons to studies that employ the life table method. Seven independent studies in the United States and Sweden yielded estimates ranging from 12.5 to 33.9 fetal deaths per 100 conceptions (*16, 74*). Similar investigations in underdeveloped countries report figures within this range: 14.9 percent in Matlab Thana, Bangladesh (*14*); 13.6 percent in the Punjab, India (*75*); 17.6 percent in Lahore, Pakistan (*76*); and 16 to 19 percent and 30 percent in two surveys in South India (*77*). A comparison of reported rates of stillbirths (fetal deaths after week 28 of gestation) among populations indicates a negative trend with modernization, from about 4 percent of conceptions in the least-developed countries to about 1 percent in the Western world (*73*). However, this trend has only a minor effect on the overall fetal mortality rate, because stillbirths constitute no more than a small fraction of all fetal deaths.

Genetic, health, nutritional, and environmental factors have all been suggested as possible causes of fetal mortality, but it has proved impossible to clearly determine the importance of each factor (*78–82*). The absence of a statistically significant difference in the fetal mortality rates of the developed and the developing countries suggests that nutrition does not have a large effect. This tentative conclusion is supported by the results of the Guatemalan investigation discussed earlier (*66*). In that population maternal nutritional status did not affect the mean duration of the interval between the end of postpartum amenorrhea and the next birth. Any increase in intrauterine mortality would have lengthened that interval; the absence of a nutritional differential implies, therefore, that the link between maternal nutrition and the incidence of fetal mortality is either absent or very weak.

Fecundity and Fertility in Famine

The discussion up to this point has focused on the effects of chronic malnutrition. We turn now to the impact of acute malnutrition in famines.

Famines are invariably associated with large but temporary reductions in fertility. During food crises in 17th- and 18-century Europe rises in food prices were followed by declines in birthrates (*84, 85*). Similarly, the rate of conception reached a minimum during famines in recent world wars (*86, 87*). Perhaps best documented is the Dutch famine which took place from October 1944 to May 1945 in an otherwise well-nourished population (*87*). The birthrate did not change during the actual famine period, but 9 months later fertility was reduced by more than 50 percent. A fertility decline of nearly the same magnitude was observed after the 1974–1975 famine in Bangladesh. The price of rice began to rise in 1974 and reached a peak in early 1975. Associated with this food shortage was a 50 percent increase in the death rate, predominantly among infants, and a decline in the birthrate from about 45 per 1000 population before the famine to 27.5 for the period from April 1975 to March 1976 (*88*).

These correlations between the occurrence of extreme food deprivation and fertility provide little insight into the nature of the mechanisms involved. It is evident that fecundity is impaired in famine, because amenorrhea is often reported (*86, 87, 89*) and in severe starvation the gonads atrophy (*89, 90*). However, it is not clear whether this amenorrhea is entirely due to malnutrition or is in part caused by the fear of death and the anxiety that accompanies crisis conditions (*89–91*). Psychological stress alone can induce amenorrhea (*89, 92–94*). A number of other factors have been found to inhibit fertility during some famines: (i) decrease in libido (*88, 89*), (ii) separation of spouses by the search for food or work (*88, 89*), and (iii) voluntary fertility control by contraception, abstention, or induced abortion (*88*). A lack of sufficiently detailed data makes it impossible to estimate accurately the contributions to the fertility decline from each of these factors.

Further evidence regarding the effect of severe malnutrition on fecundity is available from studies of voluntary or self-inflicted starvation. In the classical experiment by Keys, male volunteers were subjected to a 50 percent reduction in caloric intake for 24 weeks; loss of libido was marked, and sperm motility and longevity were significantly reduced (*89*). In females large reductions in weight can produce amenorrhea, which is reversed after restoration of normal weight (*30, 95*), but much of this evidence comes from small samples of patients which may have been self-selected. In patients with anorexia nervosa the influence of psychological or psychotic factors is unclear. Also, amenorrhea frequently precedes major weight loss or persists for long periods after ideal weight has been reestablished (*89, 90, 95*), and significant numbers of severely emaciated women have been found to maintain regular menstruation (*89*). As a consequence, some investigators are reluctant to draw conclusions about the effect of weight on amenorrhea (*89, 96*).

Conclusion

Malnutrition can impair the function of the human reproductive process. This effect is strongest and most evident in famine and starvation, when both fecundity and fertility are reduced significantly. The precise causes of this reduction remain to be determined. The change in fecundity is most likely the direct result of severe malnutrition, although it could also be caused by psychological stress and anxiety. The decline in fertility is in part due to the decreased fecundity, but several behavioral changes probably contribute significantly.

Moderate chronic malnutrition has only a minor effect on fecundity, and the resulting decrease in fertility is very small. Among the fecundity components examined here, age at menarche and the duration of postpartum amenorrhea appear to be most affected, but in each case the effect could make a difference of only a few percent between the fertility levels of poorly and well-nourished women with caloric intake differences of the order of several hundred calories a day. Breast-feeding is the principal determinant of postpartum amenorrhea, and unrestricted breast-feeding is associated with lower fertility.

Although the evidence regarding the other fecundity components—age at menopause, prevalence of permanent sterility, regularity of ovulation and quality and quantity of sperm, and probability of intrauterine death—is insufficient to allow close quantitative assessment, it appears that these factors are even less influenced by chronic malnutrition than are age at menarche and amenorrhea, and some of them may not be influenced at all. It may therefore be concluded that a large improvement in the nutrition of mothers in the underdeveloped countries will at most result in a slight increase in fertility. Concern about the effect on fertility of food aid to poor nations (provided this aid does not include large quantities of infant formula) appears to be unwarranted.

References and Notes

1. R. E. Frisch, *Soc. Biol.* **22**, 17 (1975). A clear distinction should be made between the terms fecundity, fertility, and fecundability. Fecundity is the ability to reproduce. Fertility refers to actual reproductive performance (a fecund woman can be infertile). Fecundability is defined as the monthly probability of conception.
2. _____, *Science* **199**, 22 (1978); in (*3*), p. 91.
3. W. H. Mosley, Ed., *Nutrition and Human Reproduction* (Plenum, New York, 1978).
4. J. Bongaarts, *Popul. Dev. Rev.* **4**, 105 (1978). Frequency of intercourse is another behavioral factor in fertility, but it is a less important determinant of fertility differentials and trends than nonmarriage, deliberate birth control, or breast-feeding.
5. J. W. Eaton and A. J. Mayer, *Hum. Biol.* **25**, 206 (1953).
6. *Selected World Demographic Indicators by Country, 1950–2000* (ESA/P/WP.55, Department of Economic and Social Affairs, United Nations, New York, 1975).
7. H. E. Cross and V. A. McKusick, *Soc. Biol.* **17**, 83 (1970).
8. M. C. Sheps, *Popul. Stud. (London)* **19**, 65 (1965).
9. C. Tietze, *Fertil. Steril.* **8**, 1 (1957).
10. L. Henry, *Eugen. Q.* **8**, 81 (1961).
11. L. T. Rusticka, *Cholera Res. Lab. (Dacca) Sci. Rep. No. 11* (1978). The marital fertility rate is calculated from the total fertility rate for Bangladesh on the assumption that the ratio of marital to general fertility equals the one given by Rusticka.
12. J. A. Menken, thesis, Princeton University (1975).
13. A. K. M. Chowdhury, in (*3*), p. 401.
14. L. C. Chen, S. Ahmed, M. Gesche, W. H. Mosley, *Popul. Stud. (London)* **28**, 277 (1974).
15. S. L. Huffman, A. K. M. A. Chowdhury, W. H. Mosley, *Science* **203**, 922 (1979).
16. H. Leridon, *Human Fertility: The Basic Components* (Univ. of Chicago Press, Chicago, 1977).
17. J. Ganiage, *Trois Villages D'Ile-de-France au XVIII Siecle (Travaux et Documents)* (Institut National d'Etudes Demographiques, Paris, 1963), vol. 40.
18. F. E. Johnston, *Hum. Biol.* **46**, 159 (1974).
19. L. W. Zacharias and R. J. Wurtman, *N. Engl. J. Med.* **280**, 868 (1969).
20. R. H. Gray, in *Patterns and Determinants of Natural Fertility*, J. A. Menken and H. Leridon, Eds. (Ordina, Liege, 1979).
21. J. M. Tanner, *Sci. Am.* **218** (No. 1), 1 (1968).
22. J. Hiernaux, *Eugen. Q.* **15**, 12 (1968).
23. M. Kamat, in *Proceedings of the 6th International Conference, Delhi, India* (Planned Parenthood, New York, 1959), p. 114.
24. S. Chinnatamby, *J. Reprod. Fertil.* **3**, 342 (1962).
25. R. J. Burrell and J. M. Tanner, *Hum. Biol.* **33**, 250 (1961).
26. A. K. M. Chowdhury, S. L. Huffman, G. K. Curlin, *Soc. Biol.* **24**, 316 (1977).
27. R. E. Frisch, *Pediatrics* **50**, 3 (1972).
28. M. Bhalla and J. R. Shrivastava, *Indian Pediatr.* **11**, 487 (1976).
29. R. W. Hilman, P. Slater, M. J. Nelson, *Hum. Biol.* **42**, 570 (1971).
30. R. E. Frisch and J. W. McArthur, *Science* **185**, 949 (1974).
31. L. W. Zacharias, W. M. Rand, R. J. Wurtman, *Obstetr. Gynecol. Surv.* **31**, 4 (1976).
32. W. Z. Billewitz, H. M. Fellowes, C. A. Hytten, *Ann. Hum. Biol.* **3**, 51 (1976).
33. J. Trussel, *Science* **200**, 1506 (1978); J. Reeves, *ibid.* **204**, 881 (1979).
34. T. Laska Murzejewska, *Hum. Biol.* **42**, 2 (1970).
35. S. Madhavan, *Indian J. Med. Res.* **53**, 7 (1965); G. Gopalan and A. N. Naidu, *Lancet*, **1972-II**, 1077 (1972).
36. After menarche there is a period of up to several years during which women experience a relatively high incidence of irregular and anovular cycles. Although clear quantitative evidence is not available, it may be assumed that with the 2-year decline in age of menarche the fecundability of married adolescents rises somewhat. The following crude calculation provides an estimate of the resulting change in fertility: If fecundability increases by 20 percent, fertility will rise by only 6 percent, because the sensitivity of fertility to changes in fecundability is approximately 0.3 [J. Bongaarts, *Popul. Stud.* **30**, 227 (1976)]. Since married adolescents contribute only about 15 percent of completed fertility, the rise in fecundability will add less than 1 percent to completed fertility.
37. J. A. Menken and J. Bongaarts, in (*3*).
38. B. McMahon and J. Worcester, *Vital and Health Statistics* (National Center for Health Statistics, Washington, D.C., 1966), series 11, p. 19.
39. J. B. Wyon, S. L. Finner, J. E. Gordon, *Popul. Index* **32**, 328 (1966).
40. G. Frere, *S. Afr. J. Med. Sci.* **36**, 21 (1971).
41. N. Wolansky, *Curr. Anthropol.* **13**, 2 (1972).
42. L. Jaszmann, N. D. Van Lith, J. C. A. Zaat, *Int. J. Fertil.* **14**, 2 (1969).
43. S. McKinlay, M. Jefferys, B. Thompson, *J. Biosoc. Sci.* **4**, 2 (1972).
44. H. Charbonneaux, in *Patterns and Determinants of Natural Fertility*, J. A. Menken and H. Leridon, Eds. (Ordina, Liege, 1979).
45. World Fertility Survey, First Reports: *Bangladesh Fertility Survey 1975–1978* (Ministry of Health and Population, Bangladesh, 1978); *Nepal Fertility Survey 1976* (Ministry of Health, Nepal, 1977); *Pakistan Fertility Survey* (Population Planning Council of Pakistan, Pakistan, 1976).
46. E. Salber, M. Feinleib, B. McMahon, *Am. J. Epidemiol.* **82**, 347 (1966).
47. J. Pascal, thesis, University of Nancy, Nancy, France (1969).
48. A. Perez, P. Vela, R. G. Potter, G. Masnick, *Popul. Stud. (London)* **25**, 491 (1971).
49. P. K. Malkani and J. J. Mirchandani, *J. Obstet. Gynecol. India* **11**, 11 (1960).
50. M. Bonte *et al.*, *Int. J. Fertil.* **19**, 97 (1974).
51. R. G. Potter, M. L. New, J. B. Wyon, J. E. Gordon, *J. Chron. Dis.* **18**, 1125 (1965).
52. L. Corsini, in *Patterns and Determinants of Natural Fertility*, J. A. Menken and H. Leridon, Eds. (Ordina, Liege, 1979).
53. M. Singarimbun and C. Manning, *Stud. Fam. Plann.* **7**, 175 (1976).
54. S. L. Huffman, A. M. K. Chowdhury, J. Chakborty, W. H. Mosley, *Popul. Stud. (London)* **32**, 251 (1978).
55. P. Cantrelle and B. Ferry, in (*3*), p. 353.
56. S. K. Kippley and G. F. Kippley, *J. Obstet. Gynecol. Neonatal Nurs.* **1**, 15 (1972).
57. W. Z. Billewitz, *J. Biosoc. Sci.* **11**, 141 (1979).
58. R. Lesthaeghe and H. Page, *Popul. Stud. (London)*, in press.
59. A. K. Jain, T. C. Hsu, R. Freedman, M. C. Chang, *Demography* **7**, 255 (1970).
60. N. Solien de Gonzalez, *Am. Anthropol.* **66**, 873 (1964).
61. B. Winikoff, *Science* **200**, 895 (1978).
62. T. McKeown and J. R. Gibson, *J. Obstet. Gynaecol. Br. Emp.* **61**, 824 (1954).
63. J. E. Tyson and A. Perez, in (*3*), p. 11; J. Tyson, *J. Biosoc. Sci. Suppl.*, **4**, 23 (1977).
64. P. Delvoye, J. Delogne-Desnock, C. Robyn, *Lancet* **1976-II**, 269 (1976).
65. S. L. Huffman, A. K. M. A. Chowdhury, W. H. Mosley, *Science* **200**, 1155 (1978).
66. J. Bongaarts and H. Delgado, in *Patterns and Determinants of Natural Fertility*, J. A. Menken and H. Leridon, Eds. (Ordina, Liege, 1979).
67. H. Delgado *et al.*, in (*3*), p. 385.
68. A. Chavez and C. Martinez, *Nutr. Rep. Int.* **7**, 1 (1973).
69. A. Chavez *et al.*, in *Proceedings of the 9th International Conference on Nutrition, Mexico, 1972* (Karger, Basel, 1975).
70. J. D. Wray, in (*3*), p. 197.
71. A. Chavez, data presented at Title XII Workshop on Nutrition and Reproductive Competency, La Jolla, Calif., 1978.
72. A. Lechtig *et al.*, *Pediatrics* **56**, 508 (1975).
73. *World Health Organization, Technical Report Series, No. 457* (World Health Organization, Geneva, 1970).
74. These estimates of fetal mortality include all intrauterine deaths after the first missed menses (4 weeks from the onset of the last menstrual period).
75. R. G. Potter, G. B. Wyon, M. New, G. E. Gordon, *Hum. Biol.* **37**, 262 (1965).
76. A. K. Awan, *Am. J. Obstet. Gynecol.* **119**, 4 (1975).
77. Unpublished studies cited by C. Gopalan and A. N. Naidu. See also (*36*).
78. *World Health Organization, Technical Report Series, No. 461* (World Health Organization, Geneva, 1970).
79. *Maternal Nutrition and the Course of Pregnancy* (National Academy of Sciences, Washington, D.C., 1970).
80. *World Health Organization, Technical Report Series, No. 302* (World Health Organization, Geneva, 1965).
81. J. Yerushalmy *et al.*, *Am. J. Obstet. Gynecol.* **71**, 1 (1956).
82. D. Baird, in *Public Health and Population Change*, M. Sheps and J. Ridley, Eds. (Univ. of Pittsburgh Press, Pittsburgh, 1965).
83. J. Moustgaard, *J. Reprod. Med.* **8**, 1 (1972).
84. J. Meuvret, *Population* **1**, 643 (1946).
85. E. L. R. Ladurie, *Annales* **24**, 7 (1969).
86. A. Antonov, *J. Pediatr.* **30**, 250 (1947).
87. Z. Stein, M. Susser, G. Saenger, F. Marolla, *Famine and Human Development* (Oxford Univ. Press, London, 1975).
88. W. H. Mosley, in *Patterns and Determinants of Natural Fertility*, J. A. Menken and H. Leridon, Eds. (Ordina, Liege, 1979).
89. A. Keys, J. Brožek, A. Henschel, O. Michelson, H. L. Taylor, *The Biology of Human Starvation* (Univ. of Minnesota Press, Minneapolis, 1950).
90. J. A. Brazel, in (*3*), p. 29.
91. J. M. Moudot-Bernard, *Relationships Between Fertility, Child Mortality and Nutrition in Africa* (OECD Technical Papers, Organization for Economic Cooperation and Development, Paris, 1977).
92. A. Sydenham, *Br. Med. J.* **2**, 159 (1946).
93. P. G. McDonough, *Fertil. Steril.* **30**, 1 (1978).
94. S. L. Israel, *Diagnosis and Treatment of Menstrual Disorders* (Hoeber, New York, 1959).
95. A. H. Crisp and E. Stonehill, *Br. Med. J.* **3**, 149 (1971).
96. A. Levran, *Fertil. Steril.* **25**, 5 (1974).