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Socioeconomic inequalities in childhood mortality: the 1970s to the 1980s*

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Abstract

The last three decades have witnessed substantial reductions in childhood mortality in most developing nations. Despite this encouraging picture, analysis of WFS and DHS survey data shows that socioeconomic disparities in survival chances have not narrowed between the 1970s and 1980s, and in some cases, have widened. Changes in mother's education and father's occupation contributed only modestly to secular declines in mortality. In most countries studied, no more than 20 per cent of the national trend could be accounted for by compositional improvements. The median contributions of improvements in mother's education and father's occupation were ten and eight per cent, respectively.

The last three decades have witnessed substantial reductions in childhood mortality in most developing countries. The record of socioeconomic progress has been less even. Nevertheless today's mothers are more likely than the mothers of the previous generation to be educated, to be living in an urban setting and to be married to a man with a non-manual occupation. We also know, particularly from extensive analysis of WFS data, that the survival chances of children vary widely between socioeconomic strata, with the educational attainment of the mother being a particularly strong predictor. From this perspective, two important questions arise. Are socioeconomic disparities in child survival widening or narrowing? And to what extent can mortality decline be attributed to changes in the socioeconomic composition of populations?

Answers to these and other questions relating to changes in the age pattern of mortality are sought by a joint analysis of WFS and DHS survey data for 15 developing countries that have participated in both survey programs. We make no pretence that these countries are representative of the developing world or of particular regions, but their number and geographical spread are sufficient to permit tentative generalizations. Life-table measures of mortality for five-year calendar periods are produced at the national level and for socioeconomic subgroups. This approach permits an unusually long historical dimension to the study of trends. It also encounters severe problems of data consistency between the WFS and DHS which are assessed in the first substantive section of the paper. In a few countries, the two sets of estimates are incompatible. These cases are dropped from the analysis. In the majority of cases, however, they match well and may be regarded (with caution) as a single continuous historical record.

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Design of the analysis

The data collection procedures of WFS and DHS are well known and require no elaboration here (Singh 1984, IRD 1990). With regard to mortality measurement, both survey programs used methods that are identical in their essential features: an initial count of live births by sex and survival, followed by a birth history in which date of birth, survival status and age at death are recorded for each child.

From these data, period measures of mortality are obtained by standard life-table procedures. For each survey, three periods of interest were defined: the five years preceding the survey plus the year of fieldwork; and the two preceding quinquennia. Thus the entire analysis is restricted to events and exposure within the 15 to 16 years before each survey. In calendar terms the study period covers a little over two decades: from the mid-1960s (the most distant WFS period) to the mid-1980s (the most recent DHS period). To press the analysis beyond this span would have encountered increasing problems of data quality and truncation. Even within this shortened time span, mortality estimates suffer from the progressive loss of children born to older mothers, as attention shifts from most recent to less recent periods. However, exploratory analysis confirmed that the truncation bias was negligible within the 15-year period under study and could be ignored.

One of the potential advantages of birth histories over summary measures of mortality from censuses and household surveys is their potential for examination of the age-specific probabilities of death. While most of the results in this paper take the form of ${}_5q_0$ estimates (termed 'overall childhood mortality'), trends in infant mortality (${}_1q_0$) and child mortality (${}_4q_1$) are also presented. The placing of the main emphasis on values of ${}_5q_0$ is justified in terms of both their relatively low sampling error and their robustness to errors in reporting age at death.

As noted earlier, the substantive focus of the paper is on socioeconomic differentials in childhood mortality. The choice of characteristics for inclusion in the analysis was limited to those measured in a reasonably comparable manner in both survey programs, and by the additional consideration of sample distributions. There were only four serious contenders: maternal education, paternal education and occupation, and urban-rural residence. In view of its strong association with child survival, maternal education was an obvious choice. The existence of level of school reached and number of years completed on both WFS and DHS files enabled us to define three groups (no schooling, some primary schooling, some secondary or higher schooling) in a consistent manner for both WFS and DHS.

The handling of paternal characteristics was less straightforward. Ideally we required a measure of economic status but neither WFS nor DHS has devised a satisfactory way of measuring this multifaceted concept. Paternal education is no doubt a predictor of income and standard of living but it is highly correlated with maternal education and, for this reason, was unlikely to yield results that differed appreciably from the maternal education results. Father's occupation has the advantage of identifying position within the economic structure more adequately than education, but is typically not well measured. A recurrent problem with this variable is the existence of an often large but ambiguous sales and service category. On the reasonable assumption that a combination of paternal education and occupation might represent socioeconomic status better than either single characteristic, the following categories were defined:

(a) white collar: professional, managerial or clerical workers with at least five years of schooling, plus sales or service workers with secondary or higher schooling.

(b agrarian: self-employed farmers or agricultural employees.

(c) blue collar: all skilled and unskilled manual workers, plus professional, managerial or clerical workers with less than five years schooling, plus sales and services workers with less than secondary schooling, plus missing values and never married mothers.

The sensitivity of estimates to the decision to place all missing values and never-married mothers in the blue collar category was assessed by running new tabulations with these two responses placed in a separate category. The only case where these adjustments made a noticeable difference to the blue collar estimate was Kenya, but, as will be shown later, this country was excluded from the main study on other grounds.

The third and last characteristic selected for the analysis was urban-rural residence. Though it can be demonstrated that much of the urban-rural differential in childhood mortality merely reflects differences in educational composition, the inclusion of the variable is warranted because of its relevance to health policies. The demonstration that large differences in mortality exist between the two strata may lead, and occasionally have led, to shifts in health resources. All WFS and DHS files contain a coding of type of place of residence, often in the form of dichotomy but sometimes with a more detailed classification of urban areas. Large and smaller urban centres were always grouped together to form a single urban stratum. We were unable to verify that the distinction between urban and rural localities was always identical in both surveys for the same country. However, the policy of both survey programs is to follow the official or census definitions of each country. It is possible that official definitions were changed in the period between the two surveys, but it is most improbable that this occurred in sufficient instances to invalidate our analysis.

To summarize, period measures of morality were calculated at the national level and for subgroups defined in terms of maternal education, paternal occupation/education, and rural-urban residence. Trends were calculated in a straightforward manner from estimates for different periods; and changes in differential mortality were assessed in form of absolute and relative differences for subgroups at different points in time.

Consistency and reliability of WFS and DHS estimates

The value of this analysis rests largely on the degree of consistency between WFS and DHS estimates. This issue is addressed in Figure 1, which presents an overview of the two sets of estimates. For several countries the conjuncture of the trend lines is perfect. Mexico, Egypt, Tunisia, and Dominican Republic fall into this category. In a further group (Sudan, Senegal, Morocco, and Ecuador), the match is very close, almost certainly within the bounds of sampling error. The remaining seven countries are more problematic. In six instances, the DHS estimate for the period approximately 10 to 14 years before the survey is lower than the WFS estimates for the most recent quinquennium before the survey. These disparities suggest – though do not prove – that deaths occurring in the more distant past suffer greater omission than recent deaths. In the remaining case, Ghana, the reverse occurs: DHS estimates are higher than the corresponding WFS estimates.

The magnitudes of these discrepancies are shown in Table 1 in some form of estimates for the same five-year period, mostly taken from an earlier analysis of DHS data quality (Sullivan, Bicego and Rutstein 1990). Ghana provides by far the most serious instance of obvious error. For the period centred on 1977 the WFS estimate of childhood mortality is 38 per cent lower than the corresponding DHS figure. A WFS analysis by Adansi-Pipim (1985) showed that WFS also yielded lower mortality estimates than the 1971 Supplementary Enquiry; thus the probable cause of the WFS-DHS discrepancy is severe omission of deaths in the Ghana WFS, even for the most recent period.

Table 1
Comparison of WFS and DHS estimate of overall childhood mortality for the same five-year reference period

	Midpoint of	Level of $_5q_0$	Percentage

	reference period	WFS	DHS	difference
Senegal	1976.0	261	259	+1
Ghana	1977.2	120	166	-38
Sudan (North)	1976.5	149	141	+5
Peru	1975.5	144	132	+9
Egypt	1975.5	208	206	+1
Morocco	1977.7	144	155	-8
Indonesia (Java/Bali)	1973.8	158	140	+11
Kenya	1975.5	143	116	+19
Dominican Rep.	1972.9	127	117	+8
Ecuador	1977.3	116	110	+5
Tunisia	1976.1	103	101	+2
Mexico	1974.4	94	93	+1
Thailand	1972.9	88	74	+15
Colombia	1974.0	105	84	+19
Sri Lanka	1973.3	85	59	+31

Source: Sullivan et al. 1990

In Sri Lanka, Kenya and Colombia, DHS estimates are appreciably lower than WFS ones for the overlapping period. In Sri Lanka the root problem may be related to the conditions under which the DHS was conducted. Part of the country had to be excluded from the sample frame and no doubt it was more difficult to execute a high-quality national survey in the 1980s than in the 1970s, for obvious reasons. In Kenya, the defect also lies primarily with the DHS; comparison with other sources suggests omission of dead children particularly by older DHS respondents. Diagnosis of the problem in Colombia is more difficult. Indeed, the longer historical perspective provided in Figure 1 indicates that the discrepancy may be restricted to the overlapping period and is not as serious as the 19 per cent gap for this period might imply.

On the basis of these comparisons we decided to exclude Ghana, Sri Lanka and Kenya from the main analysis of socioeconomic differentials. In all three cases, there were solid grounds for doubting the validity of national estimates for one or other of the two surveys, even for the most recent period. If national estimates are suspect, then even more uncertainty must surround subnational figures. For the remaining twelve countries, the two sets appeared sufficiently consistent to justify more detailed examination.

Figure 1
Trends in overall childhood mortality, circa 1965 to circa 1985

Consistency between WFS and DHS, of course, does not guarantee accuracy. Indeed a comparison with the series compiled by Hill and Pebley (1989), who drew upon a variety of data sources and report only for countries having information judged to be of reasonable quality and consistency, indicates major differences for several countries. The most startling example is Tunisia, where WFS and DHS trends are totally consistent but are much lower than the Hill-Pebley estimates. For the period centred on 1972, Hill and Pebley give a ${}_5q_0$ value of 180, compared to values from WFS of 130 for 1970 and 106 and 104 for 1976 from WFS and DHS. It is inappropriate to attempt here a resolution of these and other differences in estimated childhood mortality levels and trends, because it would involve detailed country-specific evaluations. It must suffice to stress that mortality trends for many developing countries are not known with certainty and that the results presented below should be interpreted with caution. Interpretative emphasis should be given to general patterns rather than country-specific results.

National levels and trends

We start the presentation of results with an examination of national levels and trends in childhood mortality. By comparing the estimate obtained in the WFS for the period 10 to 14 years prior to the

survey (centred approximately on 1965) with the most recent DHS estimate (centred approximately on 1985), a 20-year perspective on change is obtained.

In eleven out of twelve countries, substantial declines in childhood mortality are recorded (Table 2). The maverick in this group is North Sudan, but a special degree of caution is required for this country because of the possibility that both WFS and DHS suffer from omission. Farah and Preston (1982), for instance, obtained much higher estimates of childhood mortality from the 1973 Census than those derived from WFS.

Table 2
National levels of overall childhood mortality: circa 1965 and 1985

	Level		Decline	
	c.1965	c.1985	Abs	Percentage
Senegal	295	191	104	35
Egypt	248	107	140	57
Indonesia (Java/Bali)	206	95	111	54
Peru	195	111	84	43
Morocco	187	102	85	45
Dominican Republic	159	88	70	44
Ecuador	156	82	74	47
Tunisia	151	65	86	57
Sudan (North)	148	127	21	14
Thailand	131	45	86	66
Mexico	128	61	67	52
Colombia	123	43	80	65
Median	158	92	85	50

Note: Countries are ordered by the level of $_5q_0$ in 1965

The median percentage decline over the 20-year period is 50, with eight countries falling in the range of 40 to 60 per cent. There is no obvious relationship between the initial value of $_5q_0$ in the 1960s and the speed of decline. Indeed those countries with initially low levels (Thailand, Mexico, Colombia) appear to have experienced particularly pronounced percentage declines, with the net result that there is greater relative variability in childhood mortality in 1985 than in 1965.

The analysis is extended in Table 3 with a comparison of trends for the decade 1965-1975 (based on the WFS) with those for the decade 1975-1985 (based on the DHS). The striking finding is that most of the twelve countries experienced a greater average annual decline in the latter than the former decade.

Table 3
Average annual percentage declines in overall childhood mortality: circa 1965 to 1975 and 1975 to 1985

	c. 1965–1975	c. 1975–1985
Senegal	1.2	4.0
Egypt	2.7	6.2
Indonesia (Java/Bali)	3.1	3.3
Peru	2.7	2.2
Morocco	2.4	4.7
Dominican Republic	2.9	2.8
Ecuador	3.2	4.9
Tunisia	3.4	4.5
Sudan (North)	0.0	1.0
Thailand	4.0	3.9
Mexico	3.0	3.9
Colombia	1.7	7.0
Median	2.8	4.0

It is unlikely that this general conclusion is an artefact of data errors. Furthermore, it is totally consistent with Hill and Pebley's results for a much larger group of countries. Shown below is the average annual decline in ${}_5q_0$, calculated from the median values for four main developing regions, taken from table A-1 of their paper.

	c. 1962–1972	c. 1972–1982
Americas	1.7	7.5
Asia	3.4	5.0
Middle East	0.8	2.4
Africa	0.6	1.1

The results in Table 3 strongly support the view that the rise in interest rates in the early 1980s, the international debt crisis and the ensuing retrenchment of government subsidies and social expenditures has not resulted in a slowing down of improvements in child survival. On the contrary, the evidence suggests that the decade from the mid-1970s to the mid-1980s saw an acceleration rather than a downturn in the rate of decline of childhood mortality, among the countries studied.

One advantage of the birth history approach to mortality estimation over the application of indirect methods to census-type data is the ability to describe the age pattern of mortality. It is not claimed that reporting of ages at death is highly accurate: for instance, ages are often severely heaped at twelve months, the critical boundary between infancy and childhood. But it is unlikely that the extent of heaping has changed and thus trends in infant and child mortality should not be badly distorted.

Table 4 summarizes trends in infant and child mortality between the mid-1960s and the mid-1980s. Without exception, declines in the probability of dying between the age of one and five years have been sharper than declines in infant death rates. The median value for the twelve countries shows an annual percentage decline in childhood that is twice as great as that for infancy.

Table 4
Average annual percentage declines in overall childhood mortality ($_5q_0$), child mortality ($_4q_1$) and infant mortality ($_1q_0$): circa 1965 to 1985

	Annual percentage decline			Ratio $_1q_0/_4q_1$	
	$_5q_0$	$_4q_1$	$_1q_0$	c.1965	c.1985
Senegal	2.4	3.0	1.8	0.60	0.75
Egypt	4.5	6.0	4.0	1.54	2.24
Indonesia (Java/Bali)	3.6	5.4	2.7	1.27	2.27
Peru	2.9	4.3	2.2	1.33	2.00
Morocco	3.3	5.7	2.1	1.23	2.35
Dominican Republic	2.8	4.3	2.2	1.84	2.91
Ecuador	3.6	4.8	3.1	1.68	2.32
Tunisia	4.1	7.2	2.8	1.28	3.13
Sudan (North)	0.7	1.1	0.4	1.03	1.18
Thailand	4.8	6.5	4.4	2.21	3.50
Mexico	3.5	5.3	3.0	2.05	3.36
Colombia	5.2	7.6	4.3	1.68	3.30
Median	3.6	5.4	2.8	1.44	2.34

Over the 20 years, the age pattern of early mortality has changed profoundly. In the mid-1980s the chance of dying in infancy was typically 2.3 times the chance of dying in childhood, compared to 1.4 times in the mid-1960s. While these findings are by no means unexpected, they do underscore the growing contribution of neonatal and early post-neo-mortality to overall childhood mortality and the corresponding need to address deficiencies in maternal health and in maternity services.

Socioeconomic composition and national trends

In the preceding section, the substantial declines in mortality over the period 1965 to 1985 were described. In this section we assess the extent to which these declines may be attributed to improvement in socioeconomic structure.

Table 5 and Figure 2 summarize changes between 1965 and 1985 in three key indicators: the schooling of the mother; the occupation of the father; and urban-rural residence of the family. The estimates apply not to mothers but to the composition of births which is affected not only by structural change in the general population, but also by the fertility among members of each socioeconomic stratum. These two factors tend to offset each other, to the extent that expanding, more advantaged groups have low birth rates. This tendency may account for the small size of the increase between 1965 and 1985 in the proportion of births classified as urban. The median values indicate a negligible increase from 41 per cent urban in 1965 to 45 per cent in 1985. There are even two countries (Peru and Morocco) where the proportion urban has declined. The only case of a very large increase is Indonesia, a country characterized by exceptionally modest urban-rural fertility differentials.

Table 5
Socioeconomic composition of births: circa 1965 and 1985

	Percentage having mothers with some schooling		Percentage non-agricultural		Percentage urban	
	c. 1965	c. 1985	c. 1965	c. 1985	c. 1965	c. 1985

Senegal	5	17	39	55	32	35
Egypt	34	46	57	NA	38	41
Indonesia (Java/Bali)	28	82	43	62	17	32
Peru	55	82	52	60	60	52
Morocco	7	14	59	53[a]	40	34
Dominican Republic	78	91	50	70	44	57
Ecuador	79	89	56	68	41	49
Tunisia	9	44	42	61	49	50
Sudan (North)	19	41	60	66	26	34
Thailand	72	90	31	41	12	17
Mexico	69	83	56	NA	54	61
Colombia	78	90	58	67	59	62
Median	45	82	50[b]	62[b]	41	45

[a]15% of cases were unclassified in DHS
[b]Excluding Egypt, Morocco and Mexico

The impact of expanding schooling for girls, however, overwhelms the generally negative association between education and fertility. For these twelve countries, the median percentage of children born to mothers with at least one complete year of schooling has increased from 45 per cent in 1965 to 82 per cent in 1985. Particularly outstanding changes may be noted for Indonesia (an increase from 28 to 82 per cent) and for Tunisia (nine to 44 per cent). By comparison, the shift in terms of father's occupation is modest. While the proportion of children born to fathers with non-agricultural occupations has risen in all countries for which reliable data are available, the changes are relatively small.

The contribution of this compositional change to the overall national mortality decline is estimated by standardizing childhood mortality for the period centred on 1985 by the composition of births in the mid-1960s. Because urban-rural changes are small, this factor was omitted, but two separate standardizations were performed: one for education and the other for occupation. The education standard was defined in terms of the proportions of 1965 births to mothers with no schooling, primary schooling and with secondary or higher schooling. The occupation standard was defined similarly in terms of three categories: agricultural, blue collar and white collar. Standardized values of ${}_5q_0$ for the period centred on 1985 were calculated. They represent hypothetical levels of childhood mortality that would have prevailed in the absence of any change in the socioeconomic composition of births since 1965. The difference between the observed ${}_5q_0$ and standardized ${}_5q_0$, divided by the observed change in ${}_5q_0$ between c. 1965 and c.1985, gives the percentage of that change attributable to the shift in the composition of births over the period.

Figure 2
Socioeconomic composition of births, circa 1965 to circa 1985

The results are given in Table 6 in the form of the percentage of the 1965 to 1985 decline in childhood mortality that is attributable to improvements in socioeconomic composition. The absolute decline in childhood mortality level and that part of the change explained by changing educational and occupational composition is shown in Figure 3. In Indonesia, for instance, where there was a decline in ${}_5q_0$ over the 20-year period of 111 per 1000, it is estimated that 20 per cent (or 22 per 1000, in absolute terms) of the decline reflects changes in educational composition. The corresponding figure for occupational change is only six per cent. These two figures, of course, cannot be summed to give an overall compositional effect.

While educational and occupational change has made a positive contribution in all countries to the secular decline in childhood mortality, the contribution is surprisingly modest. The direct effect of rising education is ten per cent or less for half of the twelve countries. In these cases, it may be inferred that declines within educational strata have been of overwhelming importance in explaining the secular decline in mortality. There are a number of important exceptions, however. In Peru and Ecuador, over one-third (36 and 35 per cent, respectively) of the decline is directly attributable to improvements in

educational composition, while, in a further three countries (Dominican Republic, Indonesia and Mexico), the contribution is about one-fifth. The predominance of Latin American countries suggests that improvements in maternal education may have played a particularly important direct role in increased child survival in this region. Colombia is the only Latin American country in this analysis to show small effects of educational change.

Table 6
The contribution of changing maternal educational and paternal occupational structure to the decline in overall childhood mortality: circa 1965–1985

	Abs. change in $_5q_0$	Percentage of change attributable to: education	occupation
Indonesia (Java/Bali)	111	20	6
Tunisia	41	10	8
Peru	36	36	11
Sudan (North)	21	7	17
Thailand	42	9	2
Mexico	67	17	NA
Dominican Republic	70	21	2
Colombia	80	9	2
Egypt	140	6	NA
Senegal	104	10	14
Ecuador	74	35	23
Morocco	85	4	NA
Median	72	10	8

Note: Countries are ordered by absolute change in percentage of births to mothers with some schooling.

It should also be noted that the pace of educational progress does not account for the Latin American pattern, nor indeed does it explain the intercountry variability of results in Table 6. Neither Tunisia and Sudan, for instance, which both rank high in terms of the absolute increase in the percentage of births to mothers with some schooling, record small contributions of educational composition to the decline in childhood mortality.

The results of the second standardization suggest that changes in occupational structure have been less important that changes in female education. This is to be expected in view of the fact that paternal characteristics generally exert a less decisive influence on child survival than those of the mother (Cleland 1990). It should be remembered that paternal occupation is measured at the survey date and thus may not accurately reflect occupation during the period of mortality risk. If such misclassification is significant, it would tend to attenuate estimates of the occupation-mortality relationship.

Figure 3
The contribution of change in education and occupational structure to decline in overall childhood mortality (c.1965–c.1985)

Socioeconomic inequalities in childhood mortality: convergence or polarization

In this section we address the theme that provided the main motivation for this paper. In the 1970s, WFS documented very large socioeconomic differentials in childhood mortality for most of the 41 participating countries (e.g. Hobcraft, McDonald and Rutstein 1984). Since that time, mortality has continued to fall, probably at an accelerated pace. It is of considerable interest and policy relevance to establish whether the last decade had witnessed a convergence in the survival chances of children from different socioeconomic strata or whether the huge inequalities persist.

Analysis of mortality trends among sub-populations using WFS-DHS data encounters severe limitations of small sample sizes. WFS and DHS samples are moderate in size and fragmentation into subgroups cannot proceed far before interpretation is obscured by sampling imprecision. The difficulty is exacerbated by changes in the composition of populations over the last 20 years. In several countries, the numbers of children born to mothers with secondary schooling or into a white-collar family fall steeply for earlier periods, effectively reducing the historical depth of any analysis. Analysis of differential mortality also makes heavy demands on the quality of data, because different types of mother may vary in their ability to recall more distant children who have died. Our response to these problems is to rely only on the most recent estimates from the two surveys. In effect, the examination

of subgroup trends is based solely on a comparison of WFS data for the five years preceding the survey (approximately the mid-1970s) with DHS data for their corresponding period (approximately the mid-1980s). Estimates based on less than 300 births are also suppressed.

The first set of results, comparing the mortality of children with uneducated and primary school mothers, is shown in Table 7. Both absolute and relative differences in ${}_5q_0$ for the two periods are given. For the majority of countries, absolute differences in childhood mortality have declined since the mid-1970s. However there are four cases where the reverse has occurred: Sudan, Indonesia, Egypt and Ecuador. In Ecuador, the substantive importance of this trend should be put into context by pointing out that, by the mid-1980s, only a small minority of births (eleven per cent) fell into the uneducated category. But in the other countries where the absolute difference has widened, mothers with no schooling still account for a considerable proportion of all births.

In terms of relative risks, the overall result is adequately captured by the median values. In the mid-1970s the children of uneducated mothers were 1.46 times more likely to die before the age of five years than the offspring of primary school mothers. In the mid-1980s, this figure was almost identical (1.42). This overall conclusion of no change in relative risks – with its implication that the percentage declines in ${}_5q_0$ were similar for both educational groups – holds for half the countries in this analysis (Peru, Morocco, Tunisia, Thailand, Mexico and Colombia). Reductions are recorded in Senegal and Dominican Republic but these are more than offset by increases in Sudan, Egypt, Indonesia and Ecuador.

Table 7
Differences in overall childhood mortality between children of mothers with no and primary schooling: circa 1975 and 1985

	Absolute		Relative (primary = 1.0)	
	c.1975	c.1985	c. 1975	c. 1985
Senegal	153	73	2.22	1.56
Egypt	18	38	1.10	1.41
Indonesia (Java/Bali)	9	27	1.05	1.27
Peru	62	47	1.43	1.37
Morocco	79	53	2.04	1.96
Dominican Republic	61	34	1.54	1.37
Ecuador	19	52	1.16	1.59
Tunisia	42	23	1.59	1.43
Sudan (North)	17	46	1.12	1.45
Thailand	40	20	1.49	1.41
Mexico	40	39	1.59	1.61
Colombia	34	18	1.33	1.37
Median	41	39	1.46	1.42

The analysis is extended in Table 8 to a comparison of primary and secondary schooling for those six countries where minimum sample size requirements are met. Again there is no evidence of any general tendency towards convergence. Relative risks have widened in four cases and narrowed appreciably only in Peru. This is a remarkable finding in view of the fact that secondary school mothers tended to be a smaller and more heavily selected minority in 1975 than a decade later. The growth in size of the upper stratum has not had the expected effect of diluting disparities in child survival.

Table 8
Differences in overall childhood mortality between children of mothers with primary and secondary or higher schooling: circa 1975 and 1985

	Absolute		Relative (secondary = 1.0)	
	c.1975	c.1985	c. 1975	c. 1985
Egypt	70	49	1.64	2.09
Indonesia (Java/Bali	85	64	2.27	2.71
Peru	97	77	3.17	2.50
Ecuador	63	42	2.12	1.90
Mexico	27	46	1.47	3.57
Colombia	46	27	1.80	2.20

The parallel analysis for paternal occupation is restricted to seven of the twelve countries (Table 9). Two are omitted because the white-collar group comprised less than 300 cases in WFS (Senegal, Dominican Republic) and an additional three because of lack of comparable occupational coding in the two surveys (Egypt, Morocco, Mexico). The reduced sample of countries, together with the wide variability in the magnitude of country-specific differentials, makes it difficult to discern a general pattern.

Table 9
Differences in overall childhood mortality by paternal occupation: circa 1975 and 1985

	Absolute white collar versus:				Relative (white collar = 1.0)			
	Agrarian		Blue collar		Agrarian		Blue collar	
	c.1975	c.1985	c.1975	c.1985	c.1975	c.1985	c.1975	c.1985
Indonesia (Java/Bali)	42	65	63	33	1.37	2.16	1.57	1.59
Peru	124	104	74	39	2.93	2.89	2.28	1.72
Ecuador	85	79	71	41	2.92	3.18	2.60	2.12
Tunisia	84	44	64	34	3.39	2.32	2.81	2.00
Sudan (North)	23	37	36	34	1.19	1.38	1.30	1.35
Thailand	65	35	14	17	2.64	2.93	1.35	1.95
Colombia	87	20	53	20	3.15	1.76	2.31	1.75

Taking the agrarian-white collar comparison first, it appears that two countries (Indonesia and Sudan) have experienced a divergence in both absolute and relative terms. In Thailand and Ecuador, relative risks have increased but absolute differences have diminished. In the remaining three cases, relative risks have narrowed (Tunisia, Colombia) or remained the same (Peru). The white-collar versus blue-collar comparisons hint at a general conclusion of convergence. Six countries record a narrowing in absolute terms. Relative risks have narrowed in four countries, changed little in two cases and have increased only in Thailand.

Examination of rural-urban differentials does not raise problems of sample size, and results for all twelve countries are shown in Table 10. The median value suggests a greater tendency towards increased polarization of mortality risks than towards convergence. In the mid-1970s, rural children

were 1.4 times more likely to die before the age of five years than their urban counterparts. In the mid-1980s the relative rural disadvantage has increased to 1.6. This general finding conceals considerable variation in national results. There are four cases of convergence and two instances of little change. But in the remaining six countries, relative risks have increased, with absolute increases recorded in two cases (Egypt and Mexico). These results are remarkable in the context of the economic policies of the mid-1980s, which are usually assumed to impinge upon the urban more severely than the rural population.

Table 10
Differences in overall childhood mortality between rural and urban children: circa 1975 and 1985

	Absolute		Relative (urban = 1.0)	
	c.1975	c.1985	c. 1975	c. 1985
Senegal	165	75	2.10	1.53
Egypt	44	60	1.28	1.83
Indonesia (Java/Bali)	50	39	1.46	1.57
Peru	87	78	1.79	2.05
Morocco	59	59	1.56	1.93
Dominican Republic	24	-4	1.23	0.89
Ecuador	32	38	1.35	1.61
Tunisia	22	24	1.23	1.44
Sudan (North)	27	24	1.21	1.22
Thailand	54	23	2.34	1.88
Mexico	27	61	1.33	2.66
Colombia	45	8	1.55	1.20
Median	45	39	1.41	1.59

Summary and discussion

The main findings of this analysis may be summarized as follows. During the 20-year period from the mid-1960s to the mid-1980s, all but one of the twelve countries experienced substantial declines in overall childhood mortality ($_5q_0$). Typically the decline amounted to a 50 per cent reduction. The fall in mortality between ages one and five years was much steeper than the drop in infant mortality. The relative improvement in child survival chances over the 20-year period was unrelated to starting mortality level in the mid-1960s. Some of the countries with the lowest levels in 1965 (Thailand, Mexico, Colombia) recorded proportionate increases in survival as large as those found in high-mortality countries such as Egypt and Indonesia.

Despite the economic problems faced by many developing countries in the 1980s, the pace of decline appears to have accelerated since the mid-1970s, with an annual average percentage decline of 4.0 for 1975-1985 compared to 2.8 for the period 1965-1975.

Compositional change – in terms of educational advance, shifts in occupational structure or increased urbanization – has not been a major force underlying mortality decline in most of the twelve countries. Its contribution, though in all cases positive, is overwhelmed by mortality declines *within* all socioeconomic strata. Latin America may provide a partial exception to this generalization. In all but one of the study countries from the region, improvements in maternal education accounted for 20 to 35 per cent of the national decline.

Socioeconomic differentials in mortality for the mid-1970s were compared to those observed in the mid-1980s. In general terms, there was little evidence of convergence in relative mortality levels although absolute differences diminished. The urban-rural disparity shows a slight tendency to increase rather than decrease. The relative advantage enjoyed by the children of primary school mothers over those of uneducated mothers has remained stable, while the advantage associated with secondary over primary schooling may have widened. In terms of paternal socioeconomic status, there is a suggestion of a narrowing of the blue-collar/white-collar divide, but no such tendency was observed for the agrarian/white-collar differential. The conclusion, though based on a small number of countries, may be stated with some confidence: the huge inequalities recorded in the 1970s have persisted into the 1980s, despite the large overall decline in mortality in this decade.

How does this new evidence relate to the existing body of knowledge? Should we be surprised that the steep falls in childhood mortality have not been accompanied by greater equality? And what is the appropriate policy response? Hitherto, systematic study of sub-national trends in infant or childhood mortality has been hampered by lack of reliable data for many developing countries. There are, of course, many exceptions, usually in the form of single- country studies. Thus the narrowing of differentials by maternal education is well documented for China (Yang and Dowdle 1985) and for Costa Rica (Rosero-Bixby 1985), while the reverse has been observed for Malaysia (DaVanzo and Habicht 1986). Data are relatively abundant for Latin America and a number of analyses of differential mortality across time and space are available for this region (e.g. Palloni 1981, 1985; Guzman 1989). Guzman reports results similar to this study: little evidence of a decline in the relative risks associated with low social class or low educational status. On the basis of his analysis, however, Palloni claims that increases in overall adult literacy bring about a reduction in education-related differentials in childhood mortality. High levels of literacy are associated with social equity and a commitment to make health services widely available. In such societies, personal characteristics of families and mothers lose their primacy as determinants of health. This interpretation receives support from the multi-country analysis by Cochrane, O'Hara and Leslie (1980), who estimate that the effect on child survival of maternal education diminishes with rising national literacy levels and health expenditures. Yet Bicego and Boerma (1991), in a comparative analysis of 17 countries, show that education-related differentials in childhood mortality are not narrowed in urban settings (relative to rural areas), where literacy levels and public expenditure on health and social services have been and continue to be greater.

We see from this analysis that rising literacy levels and declining mortality at the national level do not necessarily bring out greater equality. However, this unsatisfactory outcome should not blind us to the fact that all major socioeconomic strata appear to have benefited equally from the improvements in child survival – and perhaps in child health – that were achieved in the late 1970s and early 1980s. Any disproportionate gain by more privileged groups would have resulted in increased relative risks, which is not the main pattern observed in this study.

We should also remind ourselves that appreciable socioeconomic differentials in mortality persist even in highly developed countries. Relative differences by social class have remained essentially unchanged for decades in the USA (Syme and Berkman 1976) and in the UK (Townsend and Davidson 1982). The educational differential in US adult mortality has actually widened (Feldman et al. 1989). Much of the evidence for Europe has been summarized by Valkonen (1987). In those countries of Europe noted for their egalitarian social structures, such as Denmark and Sweden, social class differences in neo-natal mortality have almost disappeared, while in other countries they remain. But even in Denmark and Sweden, as well as most other European countries for which data are available, there are still large differentials in adult male mortality.

The appropriate policy response to large socioeconomic differentials in mortality, both in developing and developed countries, should be guided by the understanding of their origins. The DHS is in a stronger position than WFS to illuminate this area, because of its more adequate measurement of the proximate determinants of health. A review of published DHS results indicates rather modest socioeconomic differentials in childhood morbidity (Boerma, Sommerfelt and Rutstein 1991). If these findings are genuine and not an artefact of differential underreporting, it suggests that response to illness (i.e., recognition and treatment) may be more important than prevention of illness in explaining the large differences in survival chances between socioeconomic subgroups. But much more analysis is needed and it would be naive to expect quick, clear-cut answers. In developed countries, attempts to explain differential mortality in terms of specific risk factors and specific diseases have failed (see, for example, Marmot, Shipley and Rose 1984). There is much discussion of general factors that discriminate by social class or educational status, such as stress and ability to cope with ill-health, but thus far little convincing evidence.

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Breastfeeding, lactational infecundity, contraception and the spacing of births: implications of the Bellagio Consensus Statement*

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Abstract

While the nutritional, immunological and anti-bacterial benefits of breast milk are incontestable, the contraceptive effect of breastfeeding is more apparent in the aggregate than at the level of the individual. Nevertheless, it has recently been recommended that lactating women not consider adopting contraception until the earliest of their first post-partum menstruation, the introduction of supplementary feeding or their child's reaching six months of age. This article employs microsimulation to quantify the implications of this recommendation for the spacing of births and, in particular, for the proportion of birth intervals that are unacceptably short. The findings are not encouraging. The implementation of this protocol would not produce better birth spacing than a simpler strategy of initiating contraception early in the post-partum period and, unless implemented perfectly, the outcomes would be considerably worse. Breastfeeding should be viewed not as a method of birth control but as the best form of infant nourishment. Efficient contraception is the best way to ensure that children in modernizing societies can reap the benefits of breastfeeding, without being endangered by being weaned too early because of a new pregnancy.

Breastfeeding confers substantial health benefits on both mother and child. For the infant, the principal advantages of breast milk are nutritional, immunological and anti-bacterial. Breast milk is an excellent sole source of nutrition for the first four to six months of a child's life, and can continue to be an important part of a child's diet for many months thereafter. Immunological protection is conferred not only through colostrum, which is produced during the first few days post partum, but through ordinary breast milk thereafter, even after supplementation. Moreover, breast milk is a sterile fluid and contains powerful anti-bacterial agents (Gray 1980, Short 1984).

For the mother, breastfeeding encourages the involution of the uterus and, thus, the rapid return of uterine tone. It promotes an affectionate bond between mother and child. It is economical, an important consideration in the Third World, and it is convenient. Finally, through the prolactin-elevating effect of nipple stimulation, breastfeeding delays the return of normal ovarian function and thereby lengthens the interval between births (McNeilly 1979, Hatcher et al. 1990:470).

This latter effect also advantages the child by lessening the likelihood of displacement from the breast by a new pregnancy. Weaning foods are grossly inadequate in many developing countries, and children weaned too soon are at risk of various protein-calorie deficiencies, such as kwashiorkor[1] and marasmus, causing general debilitation, arrested development, wasting and, possibly, death (Jelliffe and

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1 Indeed, kwashiorkor, which can be recognized by oedema, pigmentation changes, wasting, growth failure and general misery, means in the Ga language of Ghana a disease occurring in a young child deposed from his mother's breast (Jelliffe and Jelliffe 1989:276).

Jelliffe 1989:276-285). Where medical services are poor or largely absent, children who are breast-fed are more likely to survive than those who are not (McCann et al. 1981:531).

Nevertheless, breastfeeding makes considerable nutritional demands on the mother. A repeated cycle of pregnancy and lactation can become a cumulative nutritional drain and, among women who are already nutritionally vulnerable, can lead to 'maternal depletion' syndrome (Jelliffe and Jelliffe 1989:227-229); whether or not this poses a risk for her children (Haaga 1989:120), it undoubtedly represents a less than ideal situation for the mother.

The importance of maintaining the lactational period in order to protect the child at the breast has been recognized since antiquity. For centuries from the time of Galen and Soranus, the recommended length of the nursing period ranged from 18 months up to three years, and European physicians and clerics advised that nursing women should protect their milk by abstaining from sexual relations (van de Walle and van de Walle 1972, Fildes 1988:23). The major concern was the health of the child, not the mother's protection from pregnancy (McLaren 1978:67, Fildes 1986:99-100). From at least the seventeenth century, however, some physicians were aware of the relationship between breastfeeding and temporary infertility (van de Walle and van de Walle 1972), and there is considerable evidence that this knowledge was not confined to the medically-trained (Fildes 1986:108-109).

In the first half of the nineteenth century, many doctors ridiculed as an old wives' tale the notion that breastfeeding might have a contraceptive effect. Some doctors, however, were becoming more receptive to the idea of a link between lactation and fertility (McLaren 1984:67), and in France one even advocated prolonging the nursing period as a means of holding in check the fertility of the working classes (McLaren 1990:188). By the latter half of the century the medical profession generally remained opposed to artificial contraception, but in the main accepted that lactation reduces fertility. Many doctors, faced with demands from their patients for birth control, therefore advised the use of breastfeeding as one of three acceptable 'natural' methods (McLaren 1978:125)[2].

Nevertheless, many doctors were also opposed to lengthy breastfeeding (McLaren 1990:188), there being widespread apprehension concerning the dangers that prolonged breastfeeding posed to the health of both mothers and children. Annie Besant, a pioneer in the campaign for contraception, decried the 'foolish ... attempts made by ignorant people to limit the family [by] the prevalent habit of over-lactation, arising from the mistaken idea that conception is impossible during the nursing of a child...' (1887:24).

By the early decades of the present century, breastfeeding was advocated chiefly on nutritional grounds. The importance both of breastfeeding, and of a proper time to introduce supplementary feeding, was stressed. In England, for example, the National Birth-rate Commission considered six months to be the optimum minimum period of breastfeeding and nine months to be the upper limit for establishing weaning. In addition, the Commission declared that

> Women, who for inadequate reasons refuse to suckle their offspring, and those who prolong lactation to one or even two years in the hope of preventing conception, are alike culpable (National Birth-rate Commission 1920:xciii).

2 The other two natural methods were abstinence and rhythm. Unfortunately, (following Galen) the safe period was generally thought to occur in the middle of the menstrual cycle (McLaren 1978:125). Thus, Besant (1887:36) noted that 'Dr. Tyler Smith writes:"In the middle of the interval between the periods, there is little chance of impregnation taking place. The same kind of knowledge is of use by way of caution to women who menstruate during lactation, in whom there is a great aptitude to conceive; pregnancy, under such circumstances, would be injurious to the health of the foetus, the child at the breast, and the mother herself, and therefore should be avoided, if possible" '.

The practice of artificial feeding had existed for certain groups of infants for millennia but wet nursing, that is, the suckling of a child by a paid servant, is equally ancient. A fashion for hand-rearing infants started in England in the late seventeenth century among wealthy families that had previously employed wet nurses, and much later on the Continent where the practice of wet nursing was far more widespread (Fildes 1986:262-292, Fildes 1988:190-291). With the growing availability of pathogen-free milk (Dyhouse 1978) (which contributed to sharply declining infant mortality from the late nineteenth century), and with technological advances in methods of sterilization and the design of feeding vessels, the practice of bottle-feeding had become almost universal in the West by the 1930s. As breastfeeding disappeared in Western countries so did the recognition of its contraceptive effect, which remained to to be 're-discovered' several decades later by demographers trying to explain why the subsequent birth interval should be shorter in historical populations if a child died than if it survived (Henripin 1954, Henry 1961, Knodel 1968).

In the 1970s, as the evidence mounted for long periods of nursing and post-partum amenorrhoea in many developing countries, there was some interest in the possibility of developing a physiologically-based birth-control method. In light of the great variability in durations of amenorrhoea among lactating women and the extent to which knowledge of the relationship between breastfeeding and birth-spacing was based on 'tenuous assumptions, biased observations, and widely divergent methodologies' (Masnick 1979:110), lactational amenorrhoea was considered far too unreliable a birth-control method for the individual woman. Moreover, lactational amenorrhoea was less effective than the oral contraceptives and IUDs then being promoted, and average periods of lactational amenorrhoea were considerably shorter than the average durations of use of these modern contraceptive methods (van Ginneken 1974, 1977). In the words of one commentator, '[this confirms] my impression that breast milk is for feeding babies, not for preventing babies' (Thomson 1977:52).

Subsequently, the physiological basis of the contraceptive effect of breastfeeding has been firmly established by clinical studies employing more refined techniques of hormonal assay. In some quarters confidence has been renewed in the use of breastfeeding as a contraceptive.

The Bellagio Consensus Statement

In 1988 an interdisciplinary group of researchers convened at the Rockefeller Foundation's conference centre in Bellagio in order to reach an agreement on the conditions under which breastfeeding can be used as a safe and effective means of regulating fertility.

The group considered lactationally-induced infecundity to be an appropriate birth-control method for many women, and recommended that it be incorporated into family planning programs and presented to potential users as one element of informed choice. The consensus was that:

> the maximum birth spacing effect of breastfeeding is achieved when a mother 'fully' or nearly fully breastfeeds and remains amenorrhoeic (bleeding before the 56th postpartum day being ignored). When these two conditions are fulfilled, breastfeeding provides more than 98% protection from pregnancy in the first six months. At six months, or if menses return or if breastfeeding ceases to be full or nearly full before the sixth month, the risk of pregnancy increases. As soon as one of the events occurs, consideration must be given to adoption of other means of family planning if a high degree of protection is desired or needed (Family Health International 1988:1204).

Two strategies were proposed for exploiting the fertility-inhibiting effects of breastfeeding. For women or couples who do not wish to use other family-planning methods or to whom other methods are not readily available, breastfeeding could be used as a birth-spacing method in its own right. Alternatively, if there are additional problems with contraceptive continuation, especially during

lactation, the use of breastfeeding could serve as a means of postponing the introduction of contraception. The group felt that the latter strategy, rather than one in which contraception is initiated while most women are still protected by lactational infecundity, could help to secure an interval between successive births of at least two years.[3]

In a reiteration of the Bellagio statement, Kennedy, Rivera and McNeilly (1989) documented the scientific basis for the consensus and discussed key factors affecting the establishment and maintenance of breastfeeding and the duration of lactational infecundity. In addition, they suggested that in populations in which long durations of breastfeeding are common, it might be possible to extend the period during which women can rely on lactational infecundity beyond six months.

The Bellagio recommendations have not gone unchallenged. Trussell and Santow (1991) cautioned against presenting breastfeeding as an alternative to contraception. They noted that the Bellagio formula (considering contraception at the earliest of cessation of full breastfeeding, the return of the menses and the child's reaching six months of age) could prove unwieldy in the field. Moreover, of the three events signalling the need to initiate contraception, only the last (assuming, of course, that the child survives), is foreseeable with certainty. The problem with supplementation is that it may occur unexpectedly or may not be fully under the mother's control; the problem with the return of the menses is that it is sometimes preceded by ovulation. The adoption of contraception before the return of fecundity will result in some redundant protection and may even be counter-productive if contraceptive discontinuation rates are high. Nevertheless, they argued, these disadvantages must be weighed against the costs of pregnancies that occur while considering an alternative birth-control method. In their view, couples should be actively encouraged immediately after confinement to acquire contraceptives that are compatible with breastfeeding, and this should be combined with an educational message promoting their adoption sufficiently early to minimize the possibility of an unprotected period.

Responding to these criticisms, Kennedy et al. (1991) emphasized that the Bellagio guidelines should not be interpreted as promoting breastfeeding at the expense of other birth-control methods. Far from deflecting users from other family-planning methods, the informed use of lactational amenorrhoea merely adds one more item to the 'cafeteria' of birth-control methods already available. They acknowledged that identifying the beginning of supplementary feeding is the weakest aspect of the lactational amenorrhoea method (LAM), but suggested that lactational amenorrhoea provides such a high degree of protection that in some populations the full-breastfeeding requirement of the Bellagio recommendations can be relaxed. They disputed the inevitability of a delay in adopting contraception after its need is indicated. Finally, they observed that the women who are choosing LAM have hitherto been reluctant to use family planning services and that the introduction of LAM is thus increasing contraceptive coverage.

Labbok (1991) also viewed LAM as an addition to the range of birth-control methods available to users; she stressed that LAM 'may' be used in sequence with other methods since the protection afforded by breastfeeding alone is insufficient to achieve adequate birth spacing.

The issues that emerge in this debate raise a number of practical concerns.

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3 A large number of studies, albeit some with questionable methodologies, have revealed a consistent pattern of higher neonatal and infant mortality among children born within two years of the previous live birth (See Gray 1980, Winikoff 1983). Hobcraft, McDonald and Rutstein (1983) found that, in all but two of 26 countries participating in the World Fertility Survey, neonatal mortality rates were at least 50 per cent higher among children born within two years of a preceding birth; and that in most of the countries such disparities persisted at least into the second year of life. This study revealed also an independent risk to an existing child when a younger sibling is born within 18 months, with mortality at age one rising by at least 50 per cent in 16 of the countries examined.

1) What pattern of birth spacing is implied by adopting contraception at the point indicated by the Bellagio guidelines?
2) How does LAM compare with adopting contraception in the immediate post-partum period?
3) What would be the effects on birth spacing if the adoption of birth-control were delayed beyond the earliest of the return of menses and the child's reaching six months of age?
4) Does LAM offer better protection than a simpler post-partum strategy if rates of premature discontinuation of contraception are high?
5) What would be the effects on birth spacing if the Bellagio guidelines were extended beyond six months?

To address these questions, a microsimulation model was constructed to estimate the rates of conception and the intervals between successive live births that would result from various post-partum strategies of family planning. In contrast to earlier investigations that have estimated the effects of different contraceptive strategies on average conceptive delays (Potter, Masnick and Gendell 1973, Potter, Kobrin and Langsten 1979), our focus here is on distributional measures and, in particular, on the proportions of conceptions and births that occur at extremely short durations since the last birth. To frame the problem in its simplest terms, the models make no allowance for infant deaths, induced abortion or secondary sterility. In addition, they assume that couples resume sexual relations no later than the end of the sixth week post partum. A brief account of the simulation method and the input assumptions is contained in Appendix A.

One *caveat* is in order. Supplementary feeding cannot be incorporated explicitly into the simulation models owing to a lack of sufficiently detailed information on duration-specific conditional probabilities of resuming menstruation and ovulation according to supplementation status. In the absence of such information, we can yet study the implications of the Bellagio guidelines for birth-spacing in populations in which breastfeeding is unusually long and unusually intense.

The simulation models are based on results reported for the largest single prospective study of lactational amenorrhoea and anovulation yet conducted, a clinical investigation of members of the Nursing Mothers Association of Australia (Lewis et al. 1991). In this study, most infants were given no supplementary food before five months and the mean time to supplementation was as long as 5.3 months. Only one-quarter of the women breastfed exclusively for a full six months, however, and thus most could not benefit from the maximum term specified by the Bellagio guidelines (Short et al. 1991:715). The simulations nevertheless present a best-case scenario in terms of durations of lactation and lactational amenorrhoea since few women in developing countries have the leisure to breastfeed with the dedication exhibited by the participants in the Australian study. In rural Thailand, for example, three-quarters of mothers were estimated to breastfeed for at least one year, but their median duration of full breastfeeding was only slightly longer than four weeks (Knodel, Kamnuansilpa and Chamratrithirong 1985:304-305); in a prospective study in Gaza, 83 per cent of women supplemented within three months (Anderson et al. 1986:154). Similarly, median durations of breastfeeding were found to exceed nine months in 17 of 28 WFS surveys; but in those surveys which included questions about the nature of feeding, unsupplemented breastfeeding accounted for only a small proportion of the total duration. For example, in the Philippines and Lesotho the median durations of breastfeeding were, respectively, 12.3 and 21.2 months but the median duration of full breastfeeding was, in each case, only 2.9 months (Leridon and Ferry 1985).

Conceptive delays

Pregnancy is not inevitable even in the absence of both breastfeeding and contraception. A preliminary step, therefore, is to establish the maximum and minimum proportions of women who could be expected to conceive within various durations under different patterns of post-partum behaviour.

The uppermost curve in Figure 1 shows the cumulative proportions conceiving by the end of each 30-day month of a hypothetical cohort of women who neither breastfeed nor use contraception. The input for this simulation comes from a prospective study of the return of the menses and ovulation among non-lactating women. The median duration of post-partum anovulation was 56 days and the maximum duration was less than five months (Cronin 1968). In this population six per cent of women would conceive within three months of the previous confinement, 46 per cent within six months and 84 per cent within one year. The simulation also illustrates the effect of heterogeneous fecundability on the rate of conception: in each month the most fecund are the most likely to conceive, and as highly fecund women are progressively filtered from the initial population the composition of the population still at risk of conceiving shifts toward the less fecund. Thus, the longer the waiting time to conception, the smaller the conditional probability of conceiving. For example, average monthly fecundability is initially 0.25 but decreases to 0.20 at six months, to 0.17 at 12 months and to 0.15 at 24 months.

Figure 1
Cumulative proportions of women conceiving, according to months post partum and breastfeeding and contraceptive statuses

The lowest curve simulates the cumulative probability of conceiving for breastfeeding women were they to adopt perfect contraception as soon as their menses return. This simulation is based on the distribution of the time to first menstruation during lactation reported by Lewis et al. (1991) in their prospective study of members of the Nursing Mothers Association of Australia. The proportions pregnant exceed zero very early on, and rise during the second semester, because ovulation may precede the first menstruation and because the probability of such an occurrence increases with the duration since the last confinement. Nevertheless, fewer than 0.5 per cent conceive within the first three months and fewer than two per cent conceive within the first six, and the proportion conceiving while in lactational amenorrhoea reaches only seven per cent at the end of one year. These estimates accord well with the supporting scientific evidence for the Bellagio consensus statement, and with calculations presented by Short et al. (1991) to illustrate the efficacy of the lactational amenorrhoea method.[4]

4 The results presented by Short and his colleagues diverge, however, from the simulations shown here in other, critical ways. Their estimates of proportions pregnant among non-breastfeeding, non-contracepting women should be comparable to the uppermost curve of Figure 1. Unfortunately, by assuming that, in the absence of breastfeeding, normal ovulation and sexual relations are resumed immediately after confinement, and, to a lesser extent, by assuming homogeneous fecundability, they exaggerate the potential fertility of non-lactating women and, hence, the effect of adopting perfect contraception when the menses return. They estimate that 25 per cent of women conceive

These two curves represent extremes. The proportions pregnant at three months range from a high of six per cent (achieved in the absence of both breastfeeding and contraception) down to 0.5 per cent (achieved when women breastfeed and then adopt perfect contraception when the menses return). The proportions pregnant within six months range from 46 per cent down to two per cent, and within one year from 84 per cent down to seven per cent.

An intermediate strategy is to breastfeed but not to adopt contraception. The middle curve therefore shows the proportions conceiving among a cohort of non-contraceptors who breastfeed until they conceive. In this case, we can expect three per cent of women to conceive within the first three months post partum, 14 per cent within six months and 51 per cent within one year. Thereafter, the curve converges rapidly to its upper limit, reaching 79 per cent by the end of 18 months and 92 per cent by the end of two years. Thus, while breastfeeding on its own provides a measure of protection against the risk of pregnancy, its contraceptive benefit is most evident during the immediate post-partum period and dissipates rapidly with increasing time since the last birth. Overall, among breastfeeding women, the effect of adopting perfect contraception when the menses return is to reduce the proportions pregnant at three months from three to 0.5 per cent, at six months from 14 to two per cent and at one year from 51 to seven per cent.

Lactational amenorrhoea and time to next birth

Not every conception results in a live birth. To show the implications of different patterns of post-partum behaviour for intervals between successive live births, the simulation model was extended to incorporate a heterogeneous risk of spontaneous foetal loss and gestation intervals specific to pregnancy outcome. In addition, the model generously assumes the same distribution of durations of post-partum amenorrhoea after a miscarriage or stillbirth as experienced by non-lactating women after a live birth.

Figure 2 shows the simulated proportions of breastfeeding non-contraceptors who bear another child according to the number of months since the preceding birth and, for comparison, the birth function for women who neither breastfeed nor use contraception. The latter curve represents a theoretical maximum which has never been observed. The median duration between successive births is only 16.4 months; overall, 60 per cent of birth intervals are shorter than 18 months and 84 per cent are shorter than two years. The results for our breastfeeding women are considerably better. The median birth interval rises by more than one-third, to 22.2 months. Nevertheless, four women in five have a birth interval no longer than two-and-one-half years; three women in five could expect to bear another child within 24 months of the previous birth, and one woman in four could expect to bear another child within only 18 months of the previous birth. Thus, breastfeeding alone reduces, but does not eliminate, high-risk short birth intervals.

These intervals are short, but such patterns of birth spacing are by no means unprecedented. Among the Hutterites of North America, for example, even shorter inter-livebirth intervals have been observed: 50 per cent of women bore a second child within 17.5 months of their first, and at birth orders up to and including the ninth the median times to a subsequent live birth ranged only between 19.0 and 21.7 months (Sheps 1965:72). Among the Hutterites breastfeeding was universal. Indeed, it was not uncommon for mothers to nurse their babies for more than one year, with weaning occurring only after another pregnancy had supervened. Nursing episodes were regulated, however, according to the dictates of rigid community schedules, and supplementation occurred as early as six weeks post partum (Huntington and Hostetler 1966). The fertility achieved by the Hutterites underscores the need for

during the first month post partum (as against none in the current simulations), that 58 per cent conceive within three months (as against a more realistic six per cent) and that 82 per cent conceive within six months (as against our 46 per cent).

breastfeeding to be full and for suckling episodes to be both frequent and intense if lactation is to have a substantial effect on the length of the period of post-partum infecundity.

Figure 2
Cumulative proportions of breastfeeding women bearing another child, according to months post partum and contraceptive status

It is difficult to imagine any circumstances in which a regimen producing birth intervals as short as those of our breastfeeding women would be advocated as a means of promoting the health of mother and child. Indeed, a principal aim of the Bellagio guidelines was to minimize the incidence of dangerously short birth intervals, particularly those shorter than two years. Yet, the spacing pattern shown in Figure 2 actually reflects the consequence of following the first of the two Bellagio recommendations, namely, that couples rely solely on breastfeeding to postpone the next birth if other birth-control methods are either not available or not desired.

The Lactational Amenorrhoea Method

The second, and major, Bellagio recommendation is that couples who need or want a high degree of protection should *consider* using contraception at the earliest of the return of the menses, the cessation of full breastfeeding and the child's reaching six months of age. As we have already seen in Figure 1, the proportion conceiving within the first six months post partum when perfect contraception is adopted at the onset of menstruation is consistent with the two per cent failure rate that the Bellagio consensus statement attributes to lactational amenorrhoea. This figure, impressive though it is, could be

misleading if interpreted as a likely outcome of applying the lactational amenorrhoea method.[5] Not all women, even if fully breastfeeding, will remain lactationally infecund during the entire six-month period[6]; and none will be privileged to adopt perfect contraception. In any group relying on the lactational amenorrhoea method, the proportion conceiving within six months post partum will reflect in part the fecundity-reducing effect of breastfeeding. Nevertheless, of equal if not greater importance for conceptive delays will be the proportion of women who are again fecund and at risk of conceiving at each duration post partum, their promptness in initiating contraception when its need is first indicated, and their contraceptive practice after fecundity has returned.

Figure 3 illustrates the effects of adopting contraception affording different levels of protection at a point indicated by the application of LAM. These simulations are based on the durations of lactational amenorrhoea reported by Lewis et al. (1991). Contraceptive use is incorporated in the models by reducing each woman's fecundability by a constant factor sufficient to induce a designated failure rate in a group of fecund, non-lactating contraceptors during twelve months of continuous use. The failure rates encompass a wide span of contraceptive efficiencies[7], having been chosen to reflect the range of outcomes to be expected from the use of different contraceptive methods. Thus, three per cent corresponds to the pill, eight per cent to condoms, 14 per cent to withdrawal, and 25 per cent to spermicides or the diaphragm (Trussell and Kost 1987, Trussell et al. 1990, Bracher and Santow 1992).[8]

Figure 3
Cumulative proportions conceiving among women using LAM, according to months post partum and contraceptive failure rate

5 Nor should it be confused with the conditional probability of conceiving for a lactationally amenorrhoeic woman; see Labbok (1991:113).

6 More than 75 per cent of the 'enthusiastic' breastfeeders (Lewis et al. 1991:534) on whom the simulations are based managed to maintain lactational amenorrhoea for at least six months. Their experience, however, may be atypical. In a prospective study of non-contraceptors who were relying on lactational amenorrhoea to postpone the next pregnancy, Diaz et al. (1988:60-64) found that exclusive breastfeeding on demand maintained amenorrhoea and suppressed ovulation for as long as six months in fewer than 50 per cent of women. Moreover, while fewer than one per cent of lactating women conceived during amenorrhoea within the first six months post partum, over ten per cent of all lactating women conceived within that duration (Diaz et al. 1991:339-340).

7 For example, a first-year failure rate of three per cent corresponds to a monthly contraceptive efficiency as a high as 0.99, while a first-year failure rate of 25 per cent corresponds to a monthly efficiency of only 0.90.

8 Given difficulties in user-application or in access to contraceptive supplies, the failure rate associated with any particular method could vary substantially from the levels assumed here. For example, Moreno and Goldman (1991) estimated for 15 countries in Latin America, North Africa and Asia that first-year failure probabilities for the pill ranged between five and twelve per cent.

There is little variation between the curves during the immediate post-partum period. At three months the cumulative proportions conceiving are in each case less than one per cent. Even at six months the proportions conceiving range only between two and four per cent. The reason for this is that at the shortest durations virtually all women, regardless of their breastfeeding status, will be amenorrhoeic; and even at six months most are still lactationally infecund. At longer durations, as the number of women protected by lactational infecundity falls and the number protected by contraception rises, the curves become increasingly differentiated according to contraceptive efficiency: fewer than three per cent of those using the most efficient method conceive within twelve months of the birth of the reference child, and fewer than five per cent conceive within two years; comparable proportions for those using the least efficient contraception are eleven and 32 per cent.

As a basis of comparison, Figure 4 presents cumulative probabilities of conceiving for lactating women who adopt contraception at six weeks post partum, that is, before they resume sexual relations. In general, the comparable simulated conception functions in Figures 3 and 4 differ by less than one percentage point. These small differences reflect conceptions that occur within the first six months and before the return of the menses. Thus, so long as women initiate contraception when its need is first indicated by the Bellagio guidelines, they fare almost as well, as a group, as those who adopt contraception several months earlier. Indeed, since most women are protected initially by lactational amenorrhoea and all couples adopt other birth-control measures within six months post partum, the

critical factor here is not the time at which contraception is introduced but the reliability of the method adopted and the application with which it is used.

Figure 4
Cumulative proportions conceiving among women adopting contraception at six weeks post partum, according to months post partum and contraceptive failure rate

Thus far the simulations have assumed that contraception is adopted as soon as its need is first indicated by the Bellagio guidelines. Nevertheless, although Kennedy et al. (1991) dismissed the possibility of delays in adopting contraception beyond this point, such delays could come about in any one of a number of ways. A set of rules as complex as LAM is open to misinterpretation. For example, a mother might believe that within the first six months post partum the continuation of amenorrhoea indicates the continuation of reduced fecundity even though she has introduced supplementary feeding. Secondly, a mother might forget the six-month rule and continue to rely on amenorrhoea alone after that milestone had been passed. Thirdly, adhering strictly to the protocol, a couple could wait until the first sign that fecundity is returning before *considering* the acceptable alternatives; even though they ultimately adopt contraception there may be some time during which they have placed themselves at risk of an unwanted pregnancy. Overshadowing these difficulties are the supply problems so common in developing countries: a couple may appreciate the need for the timely introduction of contraception and may have chosen their method early in the post-partum period, but their forethought will benefit them little if the method they have chosen is unavailable when it is actually needed.

To test the sensitivity of LAM to even quite conservative assumptions about delays in initiating contraception, a series of simulations was run in which one-third of women adopt contraception as soon as its need is indicated by LAM, one-third delay adopting contraception for one cycle and one-third

delay for two. The resulting estimates of cumulative proportions of women conceiving within durations post partum are presented in Figure 5.

The effect of introducing short delays in initiating contraception is remarkable, even though not all women will be fecund before the first, or even the second, menstruation. In contrast to the two preceding sets of simulations, the curves initially climb rapidly, reaching seven per cent at six months and as much as eleven per cent at eight months. The cumulative proportions conceiving within the first eight months are almost undifferentiated according to contraceptive efficiency because pregnancies occur during this period more as a result of women's being completely unprotected than as a result of contraceptive failure. Thereafter, as all women are using contraception, whether or not they are again fecund, the primacy of the effect of contraceptive efficiency asserts itself and the curves come to resemble those of Figures 3 and 4, albeit at much higher levels.

The serious implications of these gaps in contraceptive protection for the proportions of births occurring after extremely short intervals are apparent in Table 1. Among breastfeeding women who adopt contraception early in the post-partum period, only one per cent of those using the most efficient contraception bear another child within 24 months of their preceding birth, and only three per cent bear another child within 30 months. For those using the least efficient method, the proportions with birth intervals no longer than 24 and 30 months leap to 12 and 22 per cent respectively, but even with such inefficient contraception only four per cent of women bear another child within 18 months. The outcomes for lactating women who initiate protection when its need is first indicated by LAM are only slightly worse. In contrast, with even modest delays in adopting contraception, eight per cent of women using the most efficient contraception have a birth interval no longer than 18 months and nine per cent have a birth interval no longer than 24 months. Among women using the least efficient contraception the picture is even gloomier: ten per cent bear a second child within 18 months, 17 per cent within 24 months and 27 per cent within 30 months.

Figure 5
Cumulative proportions conceiving among women using LAM with delays, according to months post partum and contraceptive failure rate

Up to this point we have assumed, somewhat unrealistically, that contraceptive use, once established, continues until pregnancy intervenes. Nevertheless, an important practical concern is the premature discontinuation of contraception. Fears have often been expressed that in populations with high rates of contraceptive discontinuation and traditional practices that confer some post-partum protection, the early adoption of contraception, though not necessarily detrimental to infant and maternal health, may have little impact on birth spacing because substantial proportions of women may actually abandon contraception before or shortly after the return of fecundity (see, for example, Bhatia et al. 1982). Indeed, this may have been one of the concerns that motivated the Bellagio meeting in the first place.

Table 1
Simulated percentages of women with birth intervals no longer than 18, 24 and 30 months, according to time of adoption of contraception and 12-month contraceptive failure rate

	Time of adoption of contraception

	6 weeks post partum				LAM				LAM with delays[1]			
	Failure rate				Failure rate				Failure rate			
Duration	**3%**	**8%**	**14%**	**25%**	**3%**	**8%**	**14%**	**25%**	**3%**	**8%**	**14%**	**25%**
18	<1	1	2	4	2	3	4	6	8	9	9	10
24	1	4	7	12	3	5	8	14	9	11	13	17
30	3	8	12	22	4	8	13	23	10	14	18	27

[1]Equal probabilities of adopting contraception in the first, second and third cycles after its need is first indicated by LAM.

Table 2 shows the implications for birth spacing of low, medium and high levels of premature contraceptive discontinuation according to the timing of the adoption of contraception and the efficiency of the contraception adopted. In light of the paucity of information on rates of contraceptive discontinuation in developing countries, both levels and patterns of contraceptive discontinuation must be assumed. The lowest level of discontinuation incorporated in the simulations is roughly comparable to one that we might expect in a developed country (Bracher and Santow 1992). As for patterns, we might expect discontinuation rates to fall with increasing duration of use as the population of users comes increasingly to comprise the more conscientious and motivated. This selectivity is incorporated in the models by holding the monthly probability of premature discontinuation constant for several months and then allowing various patterns of linear decline thereafter (see notes to Table 2). For low, medium and high variants respectively, the implied proportions of women prematurely abandoning contraception are six, ten and 19 per cent after four months of use, 15, 23 and 39 per cent after twelve months, and 23, 33 and 51 per cent after 24 months of use.

The upper panel of the table shows the proportions of women with birth intervals no longer than 18, 24 and 30 months for the low-contraceptive discontinuation variant. For each contraceptive failure rate and at each post-partum duration the proportion of post-partum acceptors who bear a second child is virtually indistinguishable from the corresponding proportion of women who adopt contraception as soon as its need is indicated by LAM. If one-third of the women using LAM delay initiating protection for one cycle and one-third delay for two, however, the proportions of women with short birth intervals rise markedly, and particularly those critical proportions with intervals no longer than 18 months.

Table 2
Simulated percentages of women with birth intervals no longer than 18, 24 and 30 months, according to pattern of contraceptive discontinuation, time of adoption of contraception and 12-month contraceptive failure rate

	Time of adoption of contraception											
	6 weeks post partum				LAM				LAM with delays[1]			
	Failure rate				Failure rate				Failure rate			
Duration	3%	8%	14%	25%	3%	8%	14%	25%	3%	8%	14%	25%
Low discontinuation[2]												
18	2	3	4	6	3	4	5	7	9	9	9	11
24	9	11	13	18	8	11	13	18	13	15	16	22
30	16	20	23	31	16	19	22	31	19	22	25	34
Medium discontinuation[3]												
18	3	5	5	6	4	5	5	7	9	10	10	12
24	13	16	17	21	12	14	16	20	16	18	19	24
30	23	27	30	37	21	24	28	35	25	28	31	37
High discontinuation[4]												
18	6	7	8	9	6	7	7	8	10	10	11	12
24	23	22	25	29	18	21	22	26	21	23	24	28
30	37	38	42	47	33	36	38	44	36	38	40	46

[1]Equal probabilities of adopting contraception in the first, second and third cycles after its need is first indicated by LAM.

[2]Monthly probability of premature discontinuation equals 0.015 during the first three months of use, declines to 0.010 at 12 months and remains constant thereafter.

[3]Monthly probability of premature discontinuation equals 0.025 during the first four months, declines to 0.015 at 12 months and to 0.010 at 24 months.

[4]Monthly probability of premature discontinuation equals 0.050 during the first four months, declines to 0.025 at 12 months and to 0.010 at 24 months.

Similar patterns emerge in both the medium and high variants of contraceptive discontinuation. As one might have predicted, the superiority of initiating contraceptive protection in accordance with LAM over early post-partum adoption becomes evident only with high rather than medium or low discontinuation, with more rather than less efficient contraception and at longer durations. For example, with high contraceptive discontinuation and contraception with a three per cent failure rate, the proportion of birth intervals no longer than 24 months is 18 per cent with LAM but 23 per cent if contraception is adopted at six weeks post partum. Conversely, with medium discontinuation and contraception with a 25 per cent failure rate, the proportion of birth intervals no longer than 18 months is seven per cent with LAM and six per cent with post-partum adoption. As with the low discontinuation variant, however, even a short delay in introducing contraception after its need is first indicated by LAM increases the likelihood that a birth will occur within 18 months of the previous birth. Thus, in some circumstances, initiating contraception in accordance with the Bellagio guidelines may indeed be preferable to an early post-partum strategy. Nevertheless, the superiority of LAM

appears in many cases to be at best only marginal, to depend on the timely adoption of contraception and to be most evident only when contraceptive discontinuation rates are so high that, whatever contraceptive strategy is followed, the proportion of dangerously short birth intervals is unacceptably large.

Various modifications to the Bellagio guidelines have been suggested. The second publication of the Bellagio consensus statement held out the possibility of extending the six-month limit in populations with long breastfeeding (Kennedy et al. 1989). On the basis of new evidence about the fertility-inhibiting effects of lactational amenorrhoea, Kennedy et al. (1991: 109) then proposed moving 'beyond Bellagio' to a reliance on lactational infecundity alone, without considering supplementation, for at least six months. Short et al. (1991) have moved even further by proposing the abandonment of not only the full-breastfeeding requirement but the six-month limit as well.[9] They suggest that well-nourished women who 'merely' wish to space their births might prefer to rely exclusively on lactational infecundity until their menses return: in developing countries 'where double protection is ... particularly wasteful' (p.716), they advocate a policy of postponing contraception until the first post-partum menstruation.

The implications of such a 'post-amenorrhoeic strategy' (Potter et al. 1973) are demonstrated in Figure 6, which shows cumulative proportions conceiving according to both contraceptive efficiency and the promptness with which contraception is initiated.[10] None of the outcomes resulting from this strategy is ideal. Even with the universal adoption of highly efficient contraception at the first menses, eight and eleven per cent of women conceive again within twelve and 18 months respectively. If there are short delays in adopting efficient contraception, or if less efficient contraception is used, the situation rapidly deteriorates further. Indeed, during the first 18 months post partum even a short delay in initiating protection is sufficient to cancel the inherent benefit of using highly efficient contraception, and women would actually be better served by the timely adoption of a less efficient method.

The implications of these conceptive delays for birth spacing are shown in the upper panel of Table 3. If protection is initiated at the onset of menstruation the proportions bearing another child within 18 and 24 months are comparable with the timely adoption under LAM and low contraceptive discontinuation (shown in the central block of the upper panel of Table 2). With modest delays in adopting contraception beyond the return of the menses, however, the proportions of women bearing another child within 18 and 24 months are comparable with the delayed adoption under LAM and high contraceptive discontinuation (third block of the lowest panel of Table 2). With both delays and medium discontinuation the proportions of short intervals exceed any shown in Table 2. Even with the most efficient contraception one in four women bears a second child within two years of the first; with the least efficient contraception nearly one in three does so.

9 Any proposal to extend the Bellagio guidelines beyond the original six-month limit would in any event require relaxing or modifying the full breastfeeding requirement since, in most cases, after six months breastfeeding alone would be insufficient to satisfy a child's nutritional needs.

10 In simulations incorporating delays, equal proportions of women are assumed to adopt contraception at the first, second and third menses.

Figure 6
Cumulative proportions conceiving among women following a post-amenorrhoeic contraceptive strategy, according to months post partum and contraceptive failure rate

Finally, what happens if we revert to LAM, but extend the rule beyond six to, say, nine months? The implications are shown in the lower panel of Table 3. As one would expect, the outcome in each case is more favourable than relying on the first menses alone as a guide to when to adopt contraception. Nevertheless, comparisons with the second and third blocks of Table 1 and the third block of the central panel of Table 2 show clearly that the outcomes are markedly worse than when contraception is adopted according to the original six-month rule.

Table 3
Simulated percentages of women with birth intervals no longer than 18, 24 and 30 months, according to time of adoption of contraception and 12-month contraceptive failure rate

	Time of adoption of contraception											
	Immediately				Delays[1]				Delays + Medium discontinuation[2]			
	Failure rate				Failure rate				Failure rate			
Duration	3%	8%	14%	25%	3%	8%	14%	25%	3%	8%	14%	25%
First menses												
18	4	5	6	7	10	11	11	12	12	12	12	14
24	8	10	13	18	20	22	24	27	25	26	28	31
30	10	14	19	28	24	28	31	37	35	37	40	45
LAM at 9 months												
18	4	5	5	7	10	11	11	12	11	11	13	14
24	5	8	10	15	15	17	18	22	20	22	23	26
30	7	11	15	24	16	20	22	31	28	31	33	40

[1]Equal probabilities of adopting contraception in the first, second and third cycles after its need is first indicated by LAM.

[2]Monthly probability of premature discontinuation equals 0.025 during the first four months, declines to 0.015 at 12 months and to 0.010 at 24 months.

We are now in a position to answer our original questions.

1) If contraception is initiated in perfect accordance with the Bellagio guidelines, between two and six per cent of women (depending on the efficiency of the method used) bear another child within 18 months, and between three and 14 per cent bear another child within two years.

2) The timely adoption of contraception under LAM produces outcomes that compare favourably with the results of immediate post-partum adoption of contraception. Nevertheless, the latter strategy reduces the possibility that an early ovulation unheralded by menstruation will lead to conception, and thus does reduce the risk of very short birth intervals.

3) Even a modest delay in adopting contraception after its need is indicated by the LAM protocol has serious consequences for birth spacing; this is despite the fact that normal ovulation need not occur before the first or even the second menstruation. Between eight and ten per cent of women bear a subsequent child within 18 months.

4) With low to medium rates of premature discontinuation (15-23 per cent of users abandoning contraception within the first twelve months of use), LAM does no better than the post-partum adoption of contraception. This applies only if LAM is followed perfectly; once some women delay adopting contraception the proportions of short birth intervals rise. For high rates of premature discontinuation (39 per cent of users abandoning contraception within the first twelve months), LAM performs better than the post-partum strategy, but the incidence of short intervals reaches an unacceptable level.

5) Extending the six-month limit of LAM to nine months raises appreciably the proportion of short birth intervals. Delays in adopting contraception thereafter would negate any benefits that might be conferred by relaxing the six-month rule. Abandoning the upper limit altogether and moving to a post-amenorrhoeic strategy of adoption would have unfortunate, and potentially disastrous, effects on birth spacing.

To sum up, when the duration of lactational amenorrhoea is typically long, the birth spacing achieved by initiating contraception at the point at which the Bellagio guidelines say its adoption should be *considered* is similar to that achieved by post-partum adoption of contraception, although a slightly greater proportion of birth intervals will be no longer than 18 months unless discontinuation rates are exceedingly high. In fact, had it been possible to introduce the timing of supplementary feeding explicitly into the simulation model, the outcomes implied by the two strategies would have been even closer since adding an extra criterion can serve only to shorten the post-partum duration at which the adoption of contraception is indicated under LAM. To realize this near equivalence, protection must be initiated as soon as the need for contraception is indicated by the original Bellagio guidelines: the six-month rule can be neither abandoned nor extended with safety, nor is it safe to delay adopting contraception for even one or two months. Finally, as long as contraception is adopted at the earlier of the cessation of full breastfeeding and the return of menstruation, and in any event no later than six months post partum, the major determinant of patterns of early birth spacing is not the rule used to determine when contraception should be adopted, but the inherent efficiency of the method, and the conscientiousness with which it is used.

Discussion

The simulations demonstrate that, in ideal circumstances, initiating contraceptive protection according to the Bellagio guidelines produces outcomes only slightly worse than those resulting from a simpler strategy of adopting contraception in the immediate post-partum period. The simulations additionally demonstrate, however, that the Bellagio guidelines do not obviate the need for good contraceptive practice. Even short delays in initiating protection after its need is indicated by LAM produce much poorer outcomes than the simpler strategy. Moreover, any gain in overall contraceptive coverage achieved by postponing the introduction of contraception is generally insufficient to offset the negative effects of premature contraceptive discontinuation. In the worst-case scenario, where ‘there are no alternatives available or if a couple chooses not to use other family planning methods’ (Family Health International 1988:1204), the contraceptive benefit of the lactational amenorrhoea method would be the same as relying on breastfeeding alone. Since the contraceptive benefits of breastfeeding are available to all lactating women regardless of whether they employ the lactational amenorrhoea method, and since the implementation of the Bellagio guidelines would place additional burdens on family-planning users and workers alike, it is natural to question why LAM should be advocated at all.

One reason might be related to double protection. Virtually all women will be anovulatory at six weeks post partum; and many, if still breastfeeding, will still be anovulatory at six months. The early post-partum adoption of contraception means that some women will be ‘protected’ by contraception even before they are again fecund. For women who adopt contraception at six weeks post partum rather than according to the Bellagio guidelines, the additional period of redundant protection (that is, over any redundant protection associated with LAM itself) could be as long as four and a half months. On

average, however, the period will be considerably shorter, since some women will have started supplementing or have experienced the first menstruation before their babies reach six months of age.[11]

The use of mechanical or hormonal contraceptives during the period of double protection involves a financial cost that must be borne either by the individual couple or, if contraceptives are provided free of charge, by the State. This financial cost cannot be ignored even though the period during which it will be incurred is very short. On the other hand, since the early return of ovulation cannot be predicted for an individual woman, this double protection reduces the risk of conceiving very early in the post-partum period. Any accounting exercise would therefore also have to consider the financial, and emotional, costs of a mistimed pregnancy and the danger that it poses to the well-being of the mother and to the health, and even survival, of both the existing child and the new one.

A second cause for concern might be a fear that contraception may undermine the establishment and maintenance of breastfeeding. The most obvious target of concern is hormonal contraception.[12] It has often been recommended that oral contraceptives not be prescribed until six weeks post partum in order to allow breastfeeding to be successfully established beforehand (Hatcher et al. 1990:465-466); further, in light of the findings of studies conducted during the 1960s and 1970s that hormonal contraceptives can have deleterious effects on both the volume and composition of breast milk (GellŽn 1977, Hull 1981), it has sometimes been recommended that the use of hormonal contraceptives be delayed during lactational amenorrhoea until six months or more post partum.[13] The latter recommendation pertains to combined oral contraceptives and, particularly, to high-dose oestrogen formulations. A recent World Health Organization study of the effects of hormonal contraceptives confirmed the findings of earlier studies (McCann et al. 1981) that, in the absence of infant or maternal malnutrition, there is no evidence of important adverse effects from progestogen-only contraceptives[14]; and concluded that such contraceptives may actually prove to be beneficial to lactation (WHO Task Force on Oral Contraceptives 1988).

In some cases, adopting contraception that does not interfere with lactation during the period of double protection could actually be advantageous for contraceptive continuation, by giving women time to establish successful use of their chosen method before the need for it becomes acute. Mini-pills, for example, must be taken at the same time each day, and the user may require time to establish a pill-taking routine (Hatcher et al. 1990:319). The risk of discontinuation of the IUD because of side effects, the most common cause of premature discontinuation of that method, is highest during the first few months and then diminishes over time, even within the first year of use (Bracher and Santow 1992).

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11 For example, Kennedy et al. (1989:482) anticipated that supplementation will usually take place between the fourth and sixth months, as long as delaying supplementary feeding does not jeopardize infant health and development.

12 Non-appliance and barrier methods are clearly not a problem; neither is the IUD, medicated varieties of which release amounts of progestin too small to affect either the infant or the quantity of breastmilk (Hatcher et al. 1990:469).

13 See for example Gray and Huber (1983). In contrast, some authors have suggested that any potential hazard associated with hormonal contraceptives would be outweighed by their positive effect on birth spacing (McCann et al. 1981:555). Short (1984:29), in advocating the use of Depo-provera to protect the nursing period, pointed out that when a lactating woman becomes pregnant her milk also contains progestogen and oestrogen, a condition which the contraceptive was designed to prevent.

14 This is not to say that progestogen-only contraceptives pose a risk in malnourished populations; the WHO trials excluded from consideration mothers and babies with any sign of malnutrition, so there is simply no evidence either way.

Early adoption of these methods would allow women who are identified early on as unsuccessful users to switch to more compatible methods while the risk of conception is still low.

Many studies have observed an inverse relationship between breastfeeding and the use of contraception, and Millman (1985) has advanced several explanations for why this should be so. One is that the negative association reflects the adverse effects of contraceptives, and in particular high-dose pills, on lactation, but she does not put particular weight on this explanation since the negative association between breastfeeding and contraception has also been observed among women using non-hormonal methods.

Another explanation is that women substitute contraception for breastfeeding as a means of controlling their fertility. This substitution could be conscious, or unconscious. In the first case, Millman argues, women in the past may have been motivated to breastfeed, at least in part, by a desire to postpone conception.[15] Recognizing greater surety in modern contraception (and, possibly, an incompatibility between contraception and breastfeeding), they choose the more efficient method.[16] In the second case, women may simply take for granted the pattern of birth spacing associated with extended breastfeeding; yet breastfeeding durations have been shrinking, and with the menses returning more rapidly than previously, women may be prompted to adopt contraception in order to maintain the customary spacing between births.

A final explanation offered by Millman is that the relationship reflects the influence of a third factor that is associated negatively with breastfeeding and positively with contraceptive use. Attempts to identify a social or economic factor with these properties have been unrewarding. Yet this explanation holds considerable promise if one looks beyond socio-economic variables.

A promising candidate is sexual activity. Permeating the Bellagio recommendations, and many of the studies on which they draw, is the assumption that exposure to the risk of conception begins with the first normal ovulation. This ignores three critical, and interrelated facts. First, many women are not sexually active again by the time of first ovulation even if it occurs at six months post partum. Secondly, a very common precipitant of weaning in developing countries is a new pregnancy[17], and many women live in anxiety that such a pregnancy will occur too soon. Thirdly, in many societies, steps are taken to prevent this by protecting at least some, if not all, of the period of lactation by post-partum sexual abstinence (Caldwell and Caldwell 1977, Page and Lesthaeghe 1980, Bracher and Santow 1982, van de Walle and TraorŽ 1986).

Women may see breastfeeding as incompatible with contraception simply because breastfeeding was traditionally seen as being incompatible with sex. Many women who are breastfeeding may not use contraception because they feel they do not need to: either they are not sexually active, in which case their decision is entirely correct; or, with their attention being focused on the new baby, who

15 Beliefs in the contraceptive effect of breastfeeding are well documented for both historical populations (McLaren 1990) and in contemporary societies (van Ginneken 1974). Nevertheless, the widespread recognition that conception can occur in the absence of menstruation, and the commonness with which weaning takes place after a subsequent conception, provide clear evidence that even completely traditional women were aware that not just breastfeeding, but lactational amenorrhoea, offer imperfect protection.

16 See also Gomez de Leon and Potter (1989). There is indeed some evidence that women, if faced with a choice between breastfeeding and oral contraception, may choose contraception; and fears have been expressed about the consequences for infant health in areas where there is no adequate substitute for breast milk (McCann et al. 1981).

17 Areas where this has been documented include Bangladesh, South India, North India, Punjab, Java, the Philippines, Taiwan, Yemen, Iran, Lebanon, Nigeria, Zaire, Uganda, Botswana, Kenya, Burkina Faso, Guatemala and Mexico; and, in North America, in the Alaskan Eskimo and Hutterite communities (Whiting 1963, McCann et al. 1981, Santow 1987, Trussell et al. 1989).

occupies them greatly during the day and with whom they sleep at night, their sexual activity is greatly reduced. Other breastfeeding women may feel uneasy that they are sexually active, and certainly may be embarrassed to admit that they are sexually active by seeking contraceptive advice. These are the women who need assistance to adopt contraception. They need to be reassured that breastfeeding and contraception are entirely compatible and, indeed, that the proper use of contraception can, by protecting the period of lactation, be positively beneficial to their babies' health.

For women who still live and bear their children according to traditional proscriptions and prescriptions, a recommendation that they adopt contraception either according to the Bellagio guidelines, or in the immediate post-partum period, is irrelevant. It is not these women, however, who are the primary target of concern. Our concern must lie, rather, with the women who are already in the process of abandoning the traditional birth-spacing practices that bind them to traditional roles and patterns of behaviour (Mosley et al. 1977) but who are not yet confident, and competent, users of modern contraception.

It would be unfair to criticize the Bellagio signatories for failing to consider this problem since they met to reach a consensus about the conditions under which breastfeeding can be used as a safe and effective method of contraception (Kennedy et al. 1989:478), not to consider the use of contraception by breastfeeding women (Kennedy et al. 1991:108). Yet, in choosing to address neither the problem of the use of contraception to protect the period of lactation, nor the confounding factor of the presence or absence of sexual activity, the Bellagio signatories have ignored an enormous area of need, and one in which a significant contribution remains to be made.

A major criticism of family-planning services is that women are seen just once, close to the time of delivery, and that there is no adequate follow-up (Winikoff and Mensch 1991). In contrast, LAM would require clinic staff and out-workers to teach the method to their clients, to follow them up at frequent intervals, to question them about their breastfeeding and menstrual statuses and to counsel them about their options once the point identified by the Bellagio guidelines had been reached.

Yet, this would not come without a cost and there would no guarantee of increased contraceptive coverage; indeed, contraceptive coverage could be considerably worse. Few would dispute the advantages of including LAM in family-planning programs if, in addition to teaching the Bellagio guidelines, health workers were occupied in promoting breastfeeding for its nutritional, immunological and anti-bacterial benefits, in monitoring infant development and maternal nutrition and intervening where necessary, in assisting women to find the contraceptive best suited to them and in coaching them in its use, in distributing contraceptives and ensuring contraceptive supplies, in assisting women to overcome problems that could otherwise lead to their discontinuing contraception and, in programs providing abortion, in offering this back-up should it be needed. It is possible, however, that the advocacy of LAM could serve to confuse women about the separate and complementary advantages of breastfeeding and contraception, and divert scarce resources from already inadequate health services. In this event, the Bellagio signatories, by broadcasting the message that it is safe to delay initiating contraception, and by failing to broadcast the message that it is unsafe to use no contraception at all, will have done, however unintentionally, a grave disservice to both their clients and their clients' children.

Appendix

The computer microsimulation model on which the analyses are based takes the members of hypothetical cohorts of 10,000 parturient women from the birth of one child to the conception and birth of the next.

Each woman is initially amenorrhoeic after the birth of a child. The model first takes her through a period of post-partum infecundity to the return of menstruation and first normal ovulation. Her duration

in the amenorrhoeic state is simulated by comparing a random probability with an input distribution of durations of post-partum amenorrhoea, the menses being said to return in the first month in which the random probability exceeds the input cumulative proportion still amenorrhoeic. The actual day of first menstruation is estimated using linear interpolation. The times to first menstruation are those reported for 101 well-nourished members of the Nursing Mothers' Association of Australia (Lewis et al. 1991:532-533). In this group, the mean time of introduction of supplementary foods was 161 days, and most women breastfed exclusively at least into the fifth month. Fewer than 25 per cent of the study population menstruated during lactation within six months post partum and only 70 per cent did so within twelve months; and the duration of lactational amenorrhoea ranged from a low of 35 days to a high of almost two years.

The overall durations of post-partum amenorrhoea and post-partum anovulation tend to be very similar, but the resumption of ovulation does not coincide exactly with the return of the menses. It has long been established that both the timing and the sequence of the first post-partum menstruation and ovulation are closely linked to the intensity and length of lactation (Perez et al. 1971, 1972). More recent studies have determined that even if ovulation precedes the first menstruation, a sizeable proportion of first ovulations are followed by an inadequate luteal phase, and therefore cannot lead to a pregnancy (Eslami et al. 1990, Gray et al. 1990).

Based on the findings of Lewis et al. (1991:532), the simulations assume, first, that the proportion of first menstruations preceded by ovulation increases linearly from 0.3 in the first three months post partum to 0.9 at twelve months and then remains constant thereafter; and, secondly, that the proportion of ovulations followed by a normal luteal phase is constant at 0.6. The simulations allow some variability between women in the lengths of menstrual cycles and, for individual women, in the lengths of cycles until normal ovarian function is resumed, with the average length declining from 32 days for the first cycle, to 30 days for the second and to 28 days for the third and succeeding cycles. These distributions are based also on results reported by Lewis et al. (1991:534). With these parameters, and assuming further that ovulation occurs randomly between 11 and 15 days before menstruation (Santow 1978:69-71), the timing of the first normal ovulation relative to the first post-partum menses ranges between -15 days to more than three months. The simulations assume that a woman's fecundability returns to its fixed value at the first normal ovulation.[18]

In a variant of the model that did not include breastfeeding, the timing of ovulation was simulated by direct reference to the durations of post-partum anovulation reported by Cronin (1968) for non-lactating women. In that case, the mean and median times from a live birth to the first ovulation are 74 and 56 days respectively, with a maximum duration of just 140 days.

Sexual relations are assumed to begin at six weeks post partum and, therefore, once normal ovulation has resumed a woman is again at risk of conceiving. To determine the timing of conception, the simulations incorporate heterogeneous fecundability, whereby the innate risk of a *recognized* pregnancy occurring varies between women but remains constant for each woman from one cycle to the next. Fecundability is represented by a beta function with mean and variance of 0.250 and 0.010

18 Some demographic studies have found that waiting times from first menses to conception are extended by maternal breastfeeding, and have inferred that breastfeeding depresses fecundability even beyond the return of the menses 'presumably as a result of a higher proportion of anovulatory cycles or a decreased probability that a fertilized ovum will implant' (VanLandingham, Trussell and Grummer-Strawn 1991:152). This inference, however, is open to question since the same effect would be observed if sexual activity were reduced during breastfeeding, if sleeping arrangements of mother and child precluded frequent sexual contact, or if fathers practised techniques of incomplete intercourse, such as coitus interruptus, during this period (Santow 1987). Only a clinical study can resolve this issue; and the simulations take a conservative approach to the question.

respectively (Leridon 1975:105-107). Conceptive delays are simulated by generating a series of random probabilities, with conception being said to take place in the first cycle at which the random probability is smaller than or equal to a woman's characteristic fecundability. The time from a live birth to the next conception is thus a function of the time a woman spends in the post-partum infecundable state, the number of fecund cycles until she conceives and the lengths of her menstrual cycles.

Once a woman has conceived, it remains to estimate the time to the next live birth. Since conception does not lead invariably to a live birth, it is necessary to allow first for the possibility of foetal loss. The risk of foetal loss is represented by a beta function with a mean of 0.144 and a variance of 0.014 (Santow and Bracher 1989), with an associated distribution of gestation periods ending in spontaneous foetal death with a mean of 3.2 lunar months (French and Bierman 1962). After a foetal loss, the woman experiences an additional period of post-partum infecundity, the distribution of which is assumed to be the same as that reported by Cronin (1968) for non-lactating women after a live birth.

The final input to the model is the live-birth gestation interval. This distribution is also taken from French and Bierman (1962), and has a mean of 10.5 lunar months. The simulation terminates at the earlier of 30 months post partum and the occurrence of a live birth, and the process is then repeated for the next member of the cohort.

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Utilization of maternal health-care services in Peru: the role of women's education*

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Abstract

This article explores the hypothesis that formal education of women influences the use of maternal health-care services in Peru, net of the mother's childhood place of residence, household socioeconomic status and access to health-care services. The findings are consistent with the hypothesis; both cross-sectional and fixed-effects logit models yield quantitatively important and statistically reliable estimates of the positive effect of maternal schooling on the use of prenatal care and delivery assistance. In addition, large differentials were found in the utilization of maternal health-care services by place of residence, suggesting that much greater efforts on the part of the government are required if modern maternal health-care services are to reach women in rural areas.

Findings from numerous studies of infant and child mortality conducted in developing countries over the last decade show a nearly universal, positive association between maternal education and child survival, a relation which has persisted in many societies even when the household's socioeconomic status has been held constant (Cochrane, O'Hara and Lesley 1980, Rutstein 1984, United Nations 1985, Cleland and van Ginneken 1988, 1989). As a result, the study of the pathways through which female schooling exercises its positive leverage has become of increasing interest to researchers in recent years (Cleland and van Ginneken 1988, Cleland and van Ginneken 1989, Barrera 1990, Streatfield, Singarimbun and Diamond 1990). It has been suggested, for example, that educating women alters the traditional balance of power within the family, leading to changes in decision-making and allocation of resources within the household (Caldwell 1979, Caldwell, Reddy and Caldwell 1983); that education modifies women's beliefs about disease causation and cure and thus influences both domestic child-care practices and the use of modern health-care services (Caldwell 1979, Caldwell, Reddy and Caldwell 1983); that schooling enhances the woman's knowledge of modern health-care facilities, improves her ability to communicate with modern health-care providers and, by increasing the value she places on good health, results in heightened demand for modern health-care services (Caldwell 1979, Schultz 1984, Caldwell and Caldwell 1988); and that maternal schooling reflects a higher standard of living and access to financial and other resources, because better educated women are more likely to marry wealthier men or because of their own increased earnings (Schultz 1984, Ware 1984).

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Because one of the principal objectives of primary health-care programs in developing countries is to improve child survival through increased utilization of preventive maternal and child health-care services, the question of whether mothers' education affects health service use is of considerable interest to health-policy makers. An improved understanding of the role played by female education can assist in the design of health interventions and, at the same time, advance our knowledge of the association between maternal education and child mortality. Evidence from previous research suggests that maternal education has a positive effect on the use of health-care services in Africa (MbackŽ and van de Walle 1987), some Middle-Eastern countries (Tekce and Shorter 1984, Abbas and Walker 1986), Asia (Akin et al. 1986, Wong et al. 1987, Streatfield, Singarimbun and Diamond 1990) and Latin America (Fern‡ndez 1984, Monteith et al. 1987).

Furthermore, both Barrera (1990) and Caldwell (1979, 1990) have argued that educated mothers are more likely than uneducated women to take advantage of modern medicine and comply with recommended treatments because education changes the mother's knowledge and perception of the importance of modern medicine in the care of her children. In a study of child nutrition in the Philippines, Barrera (1990) found that access to health-care services benefited children of educated mothers more than children of mothers with less schooling, a finding which suggested to the author that educated mothers were more likely to take advantage of available public health-care services than uneducated women. In Nigeria, Caldwell (Orubuloye and Caldwell 1975, Caldwell 1979) also found that educated women benefited more from available public health-care services than did uneducated mothers.

In contrast, Rosenzweig and Schultz (1982) view female schooling and health-care services as partial substitutes for information regarding knowledge of diseases, treatment of illness and child-care practices, and hypothesize that the effect of education on child health becomes less important as access to public health-care services improves. Presumably, in areas where such services are readily accessible, they are used by both educated and uneducated women, and thus the advantage conferred by schooling on health outcomes is narrowed. Using data from Colombia, Rosenzweig and Schultz (1982) found partial support for this hypothesis. Their results are consistent with the findings of other studies that have shown that differentials in child mortality by maternal education are less pronounced in countries with strong public-health programs, such as Costa Rica and Cuba (Behm 1979, Palloni 1981, Schultz 1990).

It is unlikely that the observed effects of maternal education on child-health outcomes simply reflects health knowledge and habits acquired in school, although they may play some role (Lindenbaum, Chakraborty and Elias 1989). Since the positive influence of maternal education on child health has been documented in a wide range of developing countries and in rural and urban areas of the same societies, where quality of schooling and teaching about healthy behaviours are likely to differ widely, persistent educational effects, found even at very low levels of schooling, seem more likely to reflect the development of cognitive skills, exposure to new ideas and 'modern' institutions (Caldwell 1990). Education could thus influence a woman's beliefs about disease causation and cure and the value she places on modern medicine.

We should note, however, that some portion of the observed effects of education may be spurious. Behrman and Wolfe (1987a, 1987b) have proposed that the association between female schooling and health outcomes may reflect not the influence of education, but the woman's childhood background, for which education serves as a proxy. The authors suggest that, in analyses without controls for the woman's childhood environment, education may serve as a proxy for human capital more generally, including health-related skills and habits acquired during childhood, and thus standard estimates of

education effects may overstate their impact on health outcomes. The authors found support for this thesis from a study of health outcomes in Nicaragua.

In this article we investigate the hypothesis that female schooling influences the use of maternal health-care services in Peru. We should note at the start, however, that our purpose is not to measure the effects of education on changes in attitudes or to model health-service demand in low-income countries *per se*, but rather to investigate the broad nature of the association between female education and the behaviour of the mother with respect to utilization of maternal health-care services. We are especially interested in examining the extent to which the relation between maternal education and the use of health-care services is confounded by the woman's childhood background, the household's socioeconomic status and access to health-care services; and whether female schooling retains a net effect on maternal health-service use, independent of such other determinants. The overall objectives of this study are to provide information for health-policy makers charged with the implementation of maternal health-service programs, and to enhance our understanding of the potential role played by health-service utilization in the association between maternal education and child survival. To examine these issues, we use data from the Peruvian Demographic and Health Survey conducted in 1986.

The setting

As one of Latin America's poorest countries, Peru has one of the highest child-mortality rates in the region. In 1980–85, for example, the probability of dying between birth and age five was 0.112 in Peru compared with 0.028 in Chile, 0.042 in Colombia, 0.060 in Brazil and 0.090 in Ecuador (Hill and Pebley 1989). Sharp regional and residential differentials characterize patterns of infant and child mortality within the country, where the Sierra (the Andean Mountain region) has the highest infant-mortality rate (110 per 1000) followed by the eastern jungle region (85 per 1000) and by the Pacific coastal area (56 per 1000 outside Lima and 34 per 1000 in Lima); rural infants are almost twice as likely to die as urban ones (IMR of 101 per 1000 in rural but 54 per 1000 in urban areas) (Instituto Nacional de Estad'stica 1988). In addition to residential variations in mortality, sharp differentials are also found by the educational level of the mother. In 1981–86, the infant-mortality rate of children of mothers with fewer than three years of schooling was 124 per 1000 compared with 22 per 1000 among infants of mothers with at least six years of education (Instituto Nacional de Estad'stica 1988).

Urban-rural and regional differentials in infant and child mortality reflect variations in living conditions, patterns of economic development and the distribution of health-care services within the country. Of Peru's three distinct geographic and ecological regions, the Pacific coastal area has been historically the most urbanized and industrialized (Mart'nez 1986, Wicht 1986). Relative to coastal areas, the Sierra, which houses close to 40 per cent of the population, is agricultural, rural and poor. Findings from a recent study on welfare distribution show, for example, that the rural areas of the Sierra are home to Peru's poorest households while relatively well-off families are concentrated on the coast, particularly in the Lima metropolitan area (Glewwe 1987). Finally, the forested eastern slopes of the Andes and the Amazon river basin make up the Selva which, largely owing to its geography and climate, has remained the least developed and populated region in Peru (Mart'nez 1986, Wicht 1986).

Despite recent governmental efforts to redistribute health-care facilities and to improve availability of preventive health-care services, access to modern medicine varies widely among the three regions and urban and rural areas. The Ministry of Health (MOH) is the principal provider of health-care services for Peru's poor population, but MOH services reach fewer than half of the Ministry's target population (Zschock 1988a). Rural areas are particularly unevenly and poorly served by the MOH, the only provider of modern medicine in rural areas, which places rural residents at a disadvantage relative to urban residents (Carrillo 1988, Zschock 1988a). In contrast, urban dwellers, especially those living on the coast, have access to another set of government-funded health-care services through Peru's

Social Security System, although it has been estimated that fewer than 20 per cent of the population have access to services funded by Social Security (Mesa-Lago 1988). There are also private health-care providers in Peru, who own a small number of hospitals and health centres, although again they operate mainly in and around Lima (Carrillo 1988, Costa and Vera la Torre 1988).

Regional and residential as well as cohort variations are also evident in women's educational attainment. Since the 1950s, educational reform has been an important component of governments' economic development policies, and this emphasis on education has led to a rapid increase in the number of schools and rising enrolments (Paulston 1972, Hay 1976, Fern‡ndez 1986, King and Bellew 1989a). The growth in attendance is reflected in rising levels of educational attainment among both men and women, although gender differences have continued to persist. These advancements have not been uniform across the country, however, as gains in rural areas have lagged behind those made in urban areas, where many of the new schools were located (King and Bellew 1989b). Given the skewed distribution of health-care services and the concentration of better-educated women in urban areas, we expect to find that much of the effect of maternal education on health-service utilization is confounded by access to services.

Data and methods

Source of data

The data used in this study come from the Peruvian Demographic and Health Survey (DHS) of reproductive-aged women carried out in September–December 1986 with the standard DHS questionnaire.[1] In addition to a retrospective fertility history and information on contraceptive use, fertility preferences, marriages, and the respondent's and her current partner's general background and work experience, the questionnaire included a maternal and child-health component which obtained information on the use of health-care services by the mother during pregnancy and delivery, childhood immunizations, and prevalence and treatment of diarrhoea for children born since January 1, 1981. A two-stage, cluster sampling procedure, designed so that the sample was self-weighing within each of Peru's 17 geographic domains, was used to select women to be interviewed. In total, 4,999 women were surveyed with the standard DHS questionnaire during Autumn 1986 (Instituto Nacional de Estad'stica 1988, Goldman, Moreno and Westoff 1989).

Our sample is based on last births born to ever-married[2] women during the five years prior to the interview date.[3] One reason for restricting analysis to ever-married women, as defined, is that information on family income was not collected in the DHS: husbands' education and occupation are important indicators of the household's economic status, and these data are available for ever-married women only; and no comparable information on household economic status is available for never-married women. Only the first child from each multiple birth was included in the sample, because

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1 Two retrospective surveys of reproductive-aged women were carried out in Peru as a part of the Demographic and Health Survey (DHS) project in conjunction with Instituto Nacional de Estad'stica (INE) and Consejo de Poblaci—n. These two surveys included one that used the DHS standard questionnaire for high contraceptive prevalence countries (with minor modifications) and another that employed an experimental questionnaire designed to test a variety of methodological issues related to the measurement of levels and determinants of fertility, contraception, child health and infant and child mortality (see Goldman, Moreno and Westoff 1989).

2 'Ever-married' refers to all women who have been in either a consensual union or a legal marriage

3 Births included in this study come only from the five-year period prior to the survey, because the tape of the standard Peruvian DHS survey, used in these analyses, included health information only for children born during the 59 months immediately preceding the date of the interview.

information for all children of multiple births is identical with respect to maternal health-service use. The sample for this analysis includes 1,925 births.

Measurement of maternal health-care services

Two dependent variables were created from questions included in the maternal-health component of the DHS questionnaire on sources of prenatal care and delivery assistance for all live births that occurred within five years of the survey date.[4] Because of the central role played by modern health care in mortality reduction in developing countries (Caldwell 1990), the question of interest in this study is whether maternal education influences the use of modern health-care services. Thus, in both the analysis of prenatal care and assistance at delivery the dependent variable is coded 1 if the woman obtained services from a doctor or a trained nurse/midwife, and 0 otherwise.[5] Women had utilized the formal health-care sector more frequently for prenatal care than delivery assistance; mothers had received prenatal care for 60.1 per cent and delivery assistance for 54.9 per cent of last births born during the five years before the survey. This difference is largely due to patterns observed in rural areas, where women received prenatal care for a larger percentage of all pregnancies than deliveries. These proportions are consistent with the findings of the National Health and Nutrition Survey (ENSSA) conducted in Peru in 1984 (Instituto Nacional de Estad'stica 1986), and the results obtained from the DHS experimental survey carried out at the time of the DHS standard questionnaire (Goldman et al. 1989).

Analytic framework

Our model of maternal health-service use draws on the conceptual framework of health-seeking behaviour developed by Kroeger (1983). Based on an extensive review of the anthropological and sociomedical literature of health care, Kroeger (1983) proposed that determinants of utilization in developing countries could be grouped under three broad headings: (1) predisposing factors including age, sex, household composition and size, ethnic group affiliation and education; (2) characteristics of illness, expected benefits from treatment and beliefs about disease causation; and (3) characteristics of the health-care system, including cost and quality of care. According to this framework, education is only one of many factors influencing decisions concerning the utilization of health-care services.

In fact, maternal education is likely to be associated with many of the other determinants identified above. The educational level of the mother is, for example, likely to be related to access to health-care services and to financial resources available to obtain modern medicine, because educated women are more likely to live in urban areas and come from higher-income families. Furthermore, the educational level of a Peruvian mother depends on her birth cohort, childhood place of residence and ethnic group or native language, because of the rapid increase in educational opportunities during the last four decades, the faster expansion of schools in urban than rural areas and potential language barriers to

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4 The DHS data do not permit us to control for the nature of the woman's pregnancy or her health endowments. It is possible, for example, that a woman seeks the services of modern health-care professionals simply because she has encountered difficulties during her pregnancy. In this case the effects of the covariates hypothesized to determine health-service use could be biased (Rosenzweig and Schultz 1983; Grossman and Joyce 1990). For example, if uneducated and poor women are more likely to have worse health endowments and are more likely to encounter difficulties during pregnancy and are thus more likely to seek modern health-care services than educated and wealthy women, then the estimated effects of maternal education and household socioeconomic status could be underestimated.

5 Other possible responses included untrained nurse (*auxiliar*) or birth attendant (*partera*); other; or no one. In the case of delivery assistance, family members were also included as a separate category. In Peru, *auxiliar* and *partera* are considered to be a part of the informal health-care sector.

schooling. These factors may also influence health-care behaviour and are likely to confound the association between female schooling and maternal health-service use.

We have distinguished a number of explanatory variables in addition to maternal schooling that may influence health-care behaviour and be associated with maternal education. Therefore, the analytic strategy employed here is to estimate a sequential set of equations based on a schematic framework, that takes into account the association between maternal education and other determinants, and that reflects the fact that the values of these variables are acquired at different stages of the women's life.[6]

Measurement of maternal education

It is possible to determine the number of years a woman had attended school from two questions included in the DHS. We have chosen to represent maternal education in four categories based on years and levels of schooling attained, instead of using the total number of years as a continuous variable. The four categories of female schooling are: no education, 1–3, 4–5, and 6+ years of schooling (Table 1). The middle categories divide primary-school education into two groups and the highest educational level indicates at least some secondary schooling. Relatively few women had received post-secondary education, and because of considerations of sample size, it was not possible to subdivide the highest education group.

As in most demographic surveys, questions in the DHS about women's education pertained to the time of the interview. This is not considered a serious problem, because all births included in these investigations occurred during the five years prior to the survey. Furthermore, the vast majority of women probably had also completed their education prior to marriage and childbearing.

Table 1
Characteristics of the sample used in the analyses of prenatal care and assistance at delivery, last live births to ever-married women 1981–1986, Peru DHS standard survey

Variable	Percentage	Variable	Percentage
Mother's education		**Husband's employment**	
None[a]	15.9	Agriculture[a]	37.5
1–3 yrs.	23.8	Skilled and unsk. manual	31.0
4–5 yrs.	20.9	Sales and service	15.2
6+ yrs.	39.3	Prof. and clerical	16.3
Childhood place of residence		**Durable goods** [b]	
City[a]	33.8	None[a]	14.6
Town	22.9	Small items	33.1
Countryside	43.4	Large items	52.2
Age of mother		**Piped water**	
< 20 yrs.[a]	11.0	No[a]	58.2
20–29 yrs.	68.3	Yes	41.8
30+ yrs.	20.7		
Language of the questionnaire		**Birth order**	
Spanish[a]	94.7	First births	18.4
Quechua/Aymara	5.3	2–3	33.6
		4–6	28.1
		7+	19.9

6 A similar approach was employed by Farah and Preston (1982) in an analysis of child mortality in Sudan.

Residence		**Prenatal care**[c]	
Lima[a]	24.1	No	39.9
Urban coast	17.8	Yes	60.1
Urban Sierra	9.9		
Urban Selva	4.7	**Assistance at delivery**[c]	
Rural coast	7.5	No	45.1
Rural Sierra	27.3	Yes	54.9
Rural Selva	8.7		
Husband's education			
< 4 yrs[a]	23.7		
4–6 yrs.	26.1		
7–11 yrs.	15.2		
12+	35.0		N = 1,925

[a] used as a reference category.
[b] durable goods: small items are either a radio or a bicycle or both; large items are a refrigerator, a television, a motorcycle, a car, or all or some of these items.
[c] prenatal care and assistance at delivery coded yes if a woman received care or assistance from a trained health professional (a doctor or a trained nurse/midwife).

Measurement of other explanatory variables

To account for the influence of the woman's childhood environment, we have included the woman's reported childhood place of residence as a categorical explanatory variable (Table 1). As an indicator of the mother's ethnic group affiliation we have included a variable denoting the language in which the interview was conducted which is the only way to distinguish between Indian and non-Indian women in the DHS. Because many Indians are bilingual we can identify only a relatively small number of births in this way, but these births are likely to come from the most disadvantaged women in the country.

The third background characteristic included is the birth cohort of the mother (Table 1). Because the availability of modern health-care services has increased in recent years, more younger than older women had access to modern medicine at the time when they began childbearing. It is quite possible that such experience has an effect on behaviour; for example, older women may be less comfortable with modern medicine and more reluctant to take advantage of available services than younger women. On the other hand, experience and skills acquired by older women should have a positive influence on the use of health services.

To measure health-service availability, a categorical variable was created from region and urban/rural residence (Table 1).[7] The inclusion of the residential covariate also enables us to examine the effect of health-facility distribution on the utilization of maternal health-care services by place of residence. Because the above measure of access does not account for variation in service availability within the seven broad residential categories, and thus provides only a relatively crude indicator of access to services, we also present results from a fixed-effects model, which takes account of unobserved differences between sampling units, including, but not limited to, service availability. Thus,

[7] We first ran separate models for urban and rural areas and then estimated models for pooled data. The pooling of data was not a significant restriction and we therefore present models for pooled data because the model is more parsimonious and results in greater precision of estimated coefficients. In addition to the survey of women, a community-level inquiry was carried out in Peru, but in rural areas only, to collect information on health-service availability. Unfortunately, the quality of the community-level data is rather poor, with information on distance and time of travel to nearest health-care facilities missing for a substantial proportion of births (Elo 1990). Because of the poor quality of the community-level data, we have not used them in this paper.

in addition to capturing access to services, the fixed-effects model also accounts for unmeasured social and cultural factors at the local level, which may be important in determining health-service use, net of the other variables included in the analysis. Because the educational level of the mother is associated with current place of residence, access to health-care services is likely to be a key intervening variable between female education and utilization of maternal health-care services.

Four proxy measures are used to control for family income and wealth. The first proxy is the woman's current husband's or partner's educational attainment, because education is closely associated with earnings of male workers in Peru (Glewwe 1987, Stelcner, Arriagada and Moock 1987). In addition to serving as a proxy for household income, husband's education also reflects tastes and preferences. The husband's attitudes towards modern medicine could, for example, influence the wife's decision of whether or not to seek modern health-care services. Caldwell has suggested that men with higher educational attainment may play a more important role in child-care decisions than men with less schooling (Caldwell 1990). The second proxy for the family's economic welfare is an index of husband's occupation; we use a standard classification of occupations employed by the World Fertility Survey (Table 1). The third measure is an index of durable goods with a categorization that reflects observed ownership patterns over different levels of household income in Peru (Glewwe 1987), and the fourth is a measure of housing quality (Table 1). Finally, maternal education itself is a determinant of the economic welfare of the household, particularly if the mother works. Preliminary analyses showed, however, that the mother's employment status was not a significant predictor of the utilization of maternal health-care services, and thus this variable is excluded from the models presented here.

Finally, birth order of the index child was included to capture both the woman's previous experience with pregnancy and birth, and family-size effects associated with health-service use, such as inconvenience of seeking health-care services when the mother has concurrent child-care responsibilities (Institute of Medicine 1988).

Equations and estimation

To assess the relative effects of our covariates of interest on maternal health-service use, we estimate a logistic regression. The model may be expressed as:

$$\ln[\pi/1-\pi] = \alpha + \beta X \qquad (1)$$

where π is the probability that the event occurs, α is the intercept and β is the vector of coefficients of the vector of covariates, X. In addition to estimating this model, we also estimate a fixed-effects logit model to eliminate the influence of unobserved differences between sampling units or clusters. The fixed-effects model takes account of differences in distance to health-care facilities among sampling units as well as other unobserved determinants of health-service use, which are homogeneous within clusters and which are not captured by the other covariates included in the analysis. The fixed-effects model has frequently been applied to the analysis of siblings (Griliches 1979, Behrman and Wolfe 1989, Geronimus and Korenman 1991, Hoffman, Foster and Furstenberg 1991), but to our knowledge has not been applied to control for community-level factors in the analysis of health-service utilization.

Let π_{ij} be the probability that an event occurs for a woman i (i=1,2) from a cluster j and that this probability is determined by a set of measured woman-specific characteristics, which may (Z_{ij}) or may not vary (X_j) among women in the cluster, and an unobserved cluster-specific effect (α_j):

$$\ln[\pi_{ij}/1-\pi_{ij}] = \alpha_j + \Gamma X_j + \beta Z_{ij} \qquad (2)$$

where Γ and β are the corresponding parameters of the vectors of covariates, X and Z. When the model is estimated without α_j, as in 'ordinary' logistic regression of equation (1), estimates of β may be

biased.[8] Comparisons of results obtained from 'ordinary' logistic regression with controls for our seven-category residential variable, and the fixed-effects model, allow us to test for the adequacy of controlling service availability by this residential covariate. The cluster-specific fixed-effect (α_j), is eliminated by first-differencing all variables between pairs of women in the cluster. In the subsequent analysis, the difference in the dependent variable is regressed on the differences in the explanatory variables. Note that by differencing all variables between pairs of women in the same cluster the effect of any variable that does not vary between women in the cluster (for example, α_j or region and urban/rural residence) cannot be estimated in this model.

The use of a fixed-effects model requires that we must first pair women within clusters. To do so we randomly selected pairs of women from each sampling unit.[9] Secondly, since it is impossible to distinguish the effects of characteristics unique to the cluster from the effects of the other covariates when women do not differ in the outcome of interest, only pairs of women who differ with respect to the outcome measure of interest can be included in the analysis (Chamberlain 1982). All pairs in which both women had an identical value for the dependent variable, are therefore excluded from the estimation of the fixed-effects logit model. Such exclusion leads to a reduction in the sample size, which can be quite substantial and may affect the precision of the estimated effects of the covariates. For the fixed-effects logit model our sample sizes are reduced to 214 pairs of women for the analyses of prenatal care and 169 pairs of women for analyses of delivery assistance.

The interpretation of the coefficients obtained from the fixed-effects logit model are the same as in the case of 'ordinary' logistic regression. We used the statistical package STATA to estimate all models. Three types of statistical tests are carried out: t-tests for testing the significance of individual coefficients (that is, for the net effects between each category and the reference category in the case of categorical covariates); global tests of significance, comparing fits of the sequential (nested) models; and the Hausman specification-test to examine significant differences between coefficients from the 'ordinary' logistic-regression model and the fixed-effects model (Hausman 1978).

Results

Maternal education

The bivariate effects of female schooling show a strong positive association between education and the use of maternal health-care services with the effects being somewhat stronger for delivery assistance than prenatal care (Model 1 of Table 2). Women with no education had received prenatal care for only 22.1 per cent of last births within five years of the survey while women with at least some secondary education had received care for 87.1 per cent, a relative odds of 23.57 (exp [3.16]). The comparable percentages for assistance at delivery were 13.4 per cent and 88 per cent, a relative odds of 47.47 (exp [3.86]) (Model 1 of Table 2).[10] There is no doubt, however, that these results are confounded by the woman's childhood background, access to services and the socioeconomic status of the household, and

8 The fixed-effects model captures a linear effect of unobserved community-level factors.

9 Note that if only one woman in a cluster had a birth within five years of the survey she would be excluded from subsequent analyses; 39 births were thus omitted. On the other hand, more than one pair of women from a cluster can be included

10 The unadjusted percentages reflect the percentage of women who used prenatal care or assistance at delivery without taking into account the effects of the other variables. The odds ratios are calculated by exponentiating the respective coefficient in Table 2. When a predictor is a dummy variable that takes on the values of 0 or 1, then exponentiating its coefficient yields the odds-ratio, the ratio of the odds of being in that category relative to the odds for being in the base (or omitted) category for that factor.

will be substantially attenuated when other determinants are incorporated in successively more complex models.

Model 2 of Table 2 estimates maternal health-service use as a function of the woman's background characteristics (childhood place of residence, birth cohort of the mother and ethnic background) in addition to her level of schooling. The introduction of the woman's background characteristics rather substantially attenuates the effects of maternal education at the highest level of schooling relative to the no education category, but there are smaller reductions at lower levels of education. Once the woman's background characteristics are held constant, the relative odds of receiving prenatal care for a woman with at least some secondary schooling relative to a woman with no education are reduced by 42.3 per cent from 23.57 to 13.60 (exp [2.61]), while the corresponding reduction for delivery assistance is 58.5 per cent, from relative odds of 47.47 to 19.69 (exp [2.98]). Thus, a portion of the unadjusted effect of maternal education clearly reflects the influence of the woman's childhood background, and in this sense is spurious. These results are consistent with the thesis proposed by Behrman and Wolfe.

Table 2
Logistic regression estimates (and z-statistics[a]): effects of maternal education, childhood residence, ethnicity and maternal age on prenatal care and delivery assistance

Covariate	Prenatal care		Assistance at delivery	
	Model 1	Model 2	Model 1	Model 2
Mother's education				
None[b]				
1–3 yrs.	0.86 (5.17)	0.81 (4.62)	1.02 (5.17)	0.89 (4.17)
4–5 yrs.	1.70 (9.94)	1.55 (8.24)	1.99 (10.19)	1.71 (7.80)
6+ yrs.	3.16 (18.08)	2.61 (12.70)	3.86 (19.15)	2.98 (12.76)
Childhood place of residence				
City[b]				
Town		-0.43 (2.61)		-1.04 (5.97)
Countryside		-1.17 (7.62)		-2.08 (12.55)
Age of the mother				
< 20 yrs.[b]				
20–29 yrs.		0.33 (1.85)		0.63 (3.23)
30+ yrs.		0.57 (2.68)		0.90 (3.84)
Language of questionnaire				
Spanish[b]				
Quechua/Aymara		-0.32 (1.24)		-1.72 (3.29)
Constant	-1.26 (9.15)	-0.69 (2.61)	-1.87 (11.15)	-0.80 (2.72)
	χ^2= 524.5 df=3*	χ^2= 606.3 df=8*	χ^2= 737.1 df=3*	χ^2= 968.7 df=8*

[a] z-statistic = absolute value of estimate/standard error. 1.96 and 1.64 are the critical values for significance at the 95% and 90% levels with a two-tailed test.
[b] reference category (captured by the constant term).
* χ^2 obtained by subtracting the deviance of the current model from the null model.

We hypothesized that access to services is a key confounding factor in the relation between female schooling and utilization of maternal health-care services. In Table 3, we present results from two

models which add controls for service availability to Model 2.. Model 3 controls for access to services by our seven-category residential variable, while the fixed-effects model takes account of unobserved differences in service availability among sampling units. The results from Model 3 (Table 3) show a further attenuation in the effects of maternal education on health-service use relative to the prior Model 2 (Table 2). For example, the relative odds that a woman with at least six years of schooling had received prenatal care compared to a woman with no education are now reduced to 8.33 (exp [2.12]), while the odds for delivery assistance are now 10.70 (exp [2.37]) (Model 3 of Table 3), compared to 13.60 and 19.69 obtained from Model 2, respectively. Moreover, the differences between women with no schooling and those who had received 1-3 and 4-5 years of education have also shrunk.

Table 3
Logistic regression estimates (and z-statistics[a]): effects of maternal education, childhood residence, ethnicity, maternal age and current residence on prenatal care and delivery assistance

Covariate	Prenatal care		Assistance at delivery	
	Model 3	Fixed effect	Model 3	Fixed effect
Mother's education				
None[b]				
1–3 yrs.	0.69 (3.72)	1.01 (2.23)	0.71 (2.92)	0.38 (0.79)
4–5 yrs.	1.30 (6.55)	2.00 (4.12)	1.41 (5.62)	0.90 (1.73)
6+ yrs.	2.12 (9.69)	2.48 (4.74)	2.37 (8.93)	1.61 (3.08)
Childhood place of residence				
City[b]				
Town	-0.03 (0.17)	0.33 (0.94)	-0.51 (2.61)	-0.07 (0.19)
Countryside	-0.33 (1.80)	-0.21 (0.59)	-1.01 (5.02)	-0.38 (1.10)
Age of the mother				
< 20 yrs.[b]				
20–29 yrs.	0.11 (0.57)	-0.35 (0.92)	0.31 (1.41)	0.16 (0.48)
30+ yrs.	0.33 (1.49)	-0.57 (1.20)	0.54 (2.03)	0.09 (0.20)
Language of questionnaire				
Spanish[b]				
Quechua/Aymara	0.13 (0.49)	-1.06 (1.52)	-0.90 (1.65)	-0.68 (0.77)
Place of residence				
Lima[b]				
Urban coast	-0.59 (2.85)		-1.69 (6.46)	
Urban Sierra	-0.67 (2.80)		-1.54 (5.25)	
Urban Selva	-0.83 (2.78)		-1.94 (5.68)	
Rural coast	-1.20 (4.78)		-2.24 (7.49)	
Rural Sierra	-2.16 (10.32)		-3.87 (13.94)	
Rural Selva	-1.97 (7.95)		-3.38 (10.75)	
Constant	0.39 (1.25)		1.33 (3.41)	
	χ^2= 751.4 df=14*		χ^2= 1286.9 df=14*	

[a] z-statistic = absolute value of estimate/standard error. 1.96 and 1.64 are the critical values for significance at the 95% and 90% levels with a two-tailed test.
[b] reference category (captured by the constant term).
* χ^2 obtained by subtracting the deviance of the current model from the null model.

The results from the fixed-effects model (Table 3), which captures unobserved differences between sampling units, further confirm the importance of maternal education on the utilization of maternal health-care services. In the case of prenatal care, maternal schooling had a somewhat more pronounced influence in the fixed-effects model than in Model 3, although only the difference for 4–5 years of education approaches significance (at the 10 per cent level) between Model 3 and the fixed-effects model. The results from the fixed-effects model for delivery assistance, on the other hand, show an attenuation in the effects of maternal schooling compared to the results from Model 3, although the two sets of coefficients do not differ from each other in a statistically significant way, except in the case of the highest level of schooling (at the 10 per cent level). The last modifier is new.

In Table 4 we present findings from our final model which takes into account the influence of the household's socioeconomic status and the woman's previous reproductive experience in addition to the covariates included in the previous models presented in Table 3. Maternal education retains a significant influence on the use of both prenatal care and delivery assistance in both Model 4, which includes our seven-category residential covariate, and in the fixed-effects model, although the effect is weakened by the inclusion of socioeconomic factors and the woman's previous reproductive experience. For example, the odds that a woman with at least six years of schooling received delivery assistance are reduced from 10.70 (Model 3) to 3.67 (exp [1.30]) (Model 4) compared to a woman with no schooling. The effects of the other education categories relative to the omitted category of no education were also reduced. Reductions in the effects of maternal education on delivery assistance are also found in the fixed-effects model when the household's socioeconomic status and birth order are added to the model (a comparison of results obtained from fixed-effects models presented in Tables 3 and 4). Although the education coefficients are no longer significant for the two lowest schooling categories in the fixed-effects model of delivery assistance (Table 4), we should note that the cross-sectional coefficients obtained from Model 4 and those obtained from the fixed-effects model do not differ in a statistically significant way.

In contrast to delivery assistance, the effects of maternal education (4–5 years) on prenatal care obtained from the fixed-effects model and from Model 4 are significantly different. The fixed-effects model shows a stronger influence of maternal schooling on prenatal care than Model 4, suggesting that our residential covariate provides an inadequate control of service availability in the case of prenatal care. Both variation in service availability among sampling units, and unmeasured social and cultural factors at the community level, which are captured by the fixed-effect model, may have confounded the relation between female schooling and the use of prenatal care in Model 4.

These results are consistent with previous analyses that have demonstrated the importance of maternal education in determining the use of health-care services in developing countries. Both cross-sectional and fixed-effects models yield quantitatively important and statistically reliable estimates of the positive impact of maternal schooling on the use of prenatal care and delivery assistance. Thus, there seems little question that the level of maternal education is an important indicator of the woman's propensity to seek the services of modern health-care professionals. However, if a woman's childhood place of residence fails to fully account for the influence of the woman's childhood background, in the absence of controls for the mother's parental home environment, the effects of education may reflect in part unobserved family characteristics.

Table 4

Logistic regression estimates (and z-statistics[a]): effects of maternal education, childhood residence, ethnicity, maternal age, current residence, socioeconomic status and birth order on prenatal care and delivery assistance

	Prenatal care		Assistance at delivery	
Covariate	**Model 4**	**Fixed effect**	**Model 4**	**Fixed effect**
Mother's education				
None[b]				
1–3 yrs.	0.49 (2.51)	1.06 (2.17)	0.44 (1.70)	0.25 (0.49)
4–5 yrs.	0.84 (3.80)	2.05 (3.91)	0.85 (3.03)	0.78 (1.34)
6+ yrs.	1.17 (4.66)	1.96 (3.40)	1.30 (4.22)	1.18 (1.96)
Childhood place of residence				
City[b]				
Town	0.12 (0.66)	0.48 (1.23)	-0.39 (1.94)	-0.11 (0.28)
Countryside	-0.04 (0.22)	0.04 (0.09)	-0.78 (3.74)	-0.31 (0.83)
Age of the mother				
< 20 yrs.[b]				
20–29 yrs.	0.22 (1.04)	-0.10 (0.22)	0.51 (2.05)	0.57 (1.33)
30+ yrs.	0.50 (1.97)	-0.15 (0.25)	0.91 (2.67)	1.09 (1.72)
Language of questionnaire				
Spanish[b]				
Quechua/Aymara	0.10 (0.36)	-1.04 (1.51)	-0.85 (1.56)	-0.18 (0.18)
Place of residence				
Lima[b]				
Urban coast	-0.49 (2.27)		-1.60 (5.85)	
Urban Sierra	-0.73 (2.94)		-1.57 (5.16)	
Urban Selva	-0.75 (2.40)		-1.82 (5.05)	
Rural coast	-0.52 (1.87)		-1.49 (4.51)	
Rural Sierra	-1.39 (5.73)		-3.12 (10.18)	
Rural Selva	-1.11 (3.97)		-2.47 (7.08)	
Husband's education				
< 4 yrs.[c]				
4–6 yrs.	-0.02 (0.12)	-0.32 (0.91)	0.08 (0.37)	0.28 (0.71)
7–11 yrs.	0.52 (2.50)	0.53 (1.15)	0.39 (1.59)	0.56 (1.29)
12+ yrs.	0.78 (3.46)	0.85 (1.82)	0.79 (3.05)	0.84 (1.80)
Husband's occupation				
Agriculture[c]				
Sk & unsk. manual	0.37 (2.23)	0.11 (0.26)	0.55 (2.88)	0.75 (1.83)
Sales and service	0.56 (2.67)	0.67 (1.52)	0.54 (2.26)	0.74 (1.56)
Prof., cler.	0.67 (2.70)	0.93 (1.73)	0.54 (1.98)	0.19 (0.35)
Index of durable goods				
None[c]				
Small items	0.27 (1.50)	-0.25 (0.63)	0.44 (1.91)	0.37 (0.84)
Large items	0.86 (4.25)	0.10 (0.23)	1.07 (4.45)	0.19 (0.42)
Piped water				
No[c]				
Yes	0.39 (2.43)	0.20 (0.42)	0.40 (2.24)	-0.09 (0.17)
Birth order[c]				
First births[c]				
2–3	-0.41 (2.13)	-0.72 (1.77)	-0.58 (2.45)	-0.60 (1.42)
4–6			-0.64 (2.49)	-0.79 (1.67)
7+			-0.81 (2.52)	-1.37 (2.31)
Constant	-0.69 (1.81)		0.20 (0.42)	
	χ^2= 846.0 df=24*		χ^2= 1370.1 df=26*	

[a] z-statistic = absolute value of estimate/standard error. 1.96 and 1.64 are the critical values for significance at the 95% and 90% levels with a two-tailed test.

[b] reference category (captured by the constant term).
[c] in the case of prenatal care birth order is coded as first births and second and higher order births.
* χ^2 obtained by subtracting the deviance of the current model from the null model.

Other determinants

The principal focus of this analysis is the impact of female schooling on the utilization of maternal health-care services. We can, however, obtain some perspective on the estimated education effects by examining the influence of the other determinants. Results from Model 2 (Table 2) imply that women who grew up in towns or the countryside are less likely to seek modern health-care services during pregnancy and delivery than women who grew up in cities. The findings from subsequent analysis, however, demonstrate that these effects are confounded by current place of residence and unobserved differences between sampling units. Once we take account of current residence the woman's childhood place of residence no longer retains an independent influence on prenatal care (Table 3). The effects of childhood place of residence are also substantially attenuated for delivery assistance, although they retain an independent influence in Model 4 (Table 4). However, these estimates are further attenuated and are no longer statistically significant in the fixed-effects model (Table 4), although the two sets of coefficients do not differ from each other in a statistically significant way. Of our other proxies for the woman's background characteristics, the effects of her ethnic group affiliation, captured by the language of the questionnaire, imply that non-Spanish speakers are less likely to seek maternal health-care services than Spanish-speaking women, although these effects do not retain statistical significance (Table 4).

Once we have controlled for the woman's other background characteristics and her education, results from Model 2 (Table 2) suggest that older women seem more likely to seek maternal health-care services than younger women. Such maternal age effects lose their significance for prenatal care, except for the highest age category in Model 4, although maternal age, at particular educational levels and birth order, has a stronger effect on delivery assistance (Table 4).[11] These results are consistent with a learning or experience hypothesis: it is possible, for example, that maternal age serves as a proxy for the woman's accumulated knowledge of health-care services and the value she places on modern medicine.

Women are more likely to seek maternal health-care services for first than higher-order births, controlling for maternal age and our other covariates (Table 4). Fern‡ndez (1984) interpreted similar findings to mean that women who have had more children tend to attach less importance to pregnancy and delivery than other women, particularly if they have not experienced difficulties with previous pregnancies. Birth-order effects, however, may also reflect the fact that having other children in the household is a factor in determining whether or not women seek maternal health-care services (Institute of Medicine 1988). Wong et al. (1987) also found that an increase in the number of children of preschool age in the family had a negative effect on prenatal-care utilization in urban areas of the Philippines. Such sibling effects could be caused by differences in attitudes of women with larger families, resource constraints, or by some other characteristics of larger households not measured in this analysis or in the Philippine study (Wong et al. 1987).

11 We tested whether the effects of maternal age varied by birth order by including an interaction term between maternal age and birth order in Model 4. The interaction was insignificant for delivery assistance, suggesting that birth-order effects are the same within each maternal age category. In the case of prenatal care, the interaction between maternal age and birth order showed a small overall net effect ($\chi^2 = 6.2$, df = 2). The positive effect of maternal age in the highest age group is somewhat stronger for first than higher-order births (results not shown).

From Models 3 and 4 we can also examine the effects of current residence on maternal health-service use. Despite the government's efforts to expand access to health-care services, our findings suggest that such efforts have been largely unsuccessful in reaching women outside the Lima metropolitan area, the coastal region more generally, and other urban areas. Although the effects of the residential covariate are somewhat attenuated when the socioeconomic variables are included in the model, residence retains a significant effect on maternal health-service use. The importance of residence is most clearly illustrated by the extreme differences in use between women living in Lima and the rural Sierra: the relative odds that a woman living in the rural Sierra received prenatal care or delivery assistance compared to a woman living in Lima are 0.25 (exp [-1.39]) and 0.04 (exp [-3.12]), respectively (Table 4).

Husband's education has a net effect similar to but weaker than mother's education (Model 4). The estimated effects of husband's schooling obtained from the fixed-effects model, although no longer statistically significant in most instances, also do not differ from the coefficients obtained from Model 4 in a statistically significant way. Husband's education is hypothesized to operate primarily as a proxy for economic well-being of the household, but at the same time, its effects are also likely to reflect attitudes toward modern medicine. The effects of other proxies for family income are also consistent with the theory that increased income has a positive effect on the utilization of modern health-care services. For example, women whose husbands work in agriculture appear the least likely users of modern health-care services (Model 4 of Table 4). Although the coefficients obtained from Model 4 and the fixed-effects model for husband's employment (Table 4) are not identical, and their significance levels have been substantially reduced in the fixed- effects model, none of the differences between the two sets of coefficients is statistically significant for either prenatal care or delivery assistance. Our other proxies for the socioeconomic status of the household (index of durable goods and piped water) lose their statistical significance and predictive power in the fixed-effects models of both prenatal care and delivery assistance (Table 4). The observed reductions most probably reflect the fact that the fixed-effects model takes account of unobserved differences between sampling units such as housing characteristics (water and sewerage facilities) as well as all other neighbourhood attributes (for example, overall levels of income).

Discussion

In this paper we have examined the hypothesis that female education influences maternal health-service use, net of the woman's childhood background, household's socioeconomic status and service availability. By investigating a sequential set of equations we also have explored the degree to which maternal schooling effects are attenuated by the other determinants also believed to influence the use of health-care services in developing countries. The focus on mothers' education was warranted on several grounds. Previous analyses of socioeconomic determinants of child health have found mothers' education to have a strong positive influence on child survival in most developing countries and, although father's education has also been identified as an important determinant in such analyses, maternal schooling has generally been more important. In 15 countries in Africa, Asia and Latin America, a 1985 United Nations study, for example, found the effects of maternal schooling on child survival to be on average twice as large as those of paternal schooling. We have documented stronger effects of mother's than husband's schooling on maternal health-service use. These results, undoubtedly, stem at least partly from the fact that women are the primary care-takers of children and therefore, mothers' attitudes and skills are especially important to the health of youngsters (Browner 1989, Schultz 1990).

The results from both the cross-sectional and fixed-effects model, controlling for service availability and the socioeconomic status of the household, confirmed the importance of maternal

education on the utilization of both prenatal care and delivery assistance. Although the differences in use among the four educational categories are narrowed substantially when the effects of all covariates are accounted for, important differences by the level of maternal schooling remain. At the same time, we also found that a portion of the unadjusted effect of maternal schooling reflected the woman's background characteristics and in this sense was spurious. These findings provided support for the thesis, proposed by Behrman and Wolfe, that education serves as a proxy for the woman's early environment. However, better measures of the woman's childhood background than those collected by the DHS are required to explore the extent of such confounding. Thus, it is possible that some portion of the education effect found here also reflects the woman's unobserved family background.

Our results with respect to the residential covariate are particularly instructive from the viewpoint of health-policy makers. Our findings confirm results of prior studies (see for example, Carrillo 1988, Zschock 1988a, 1988b) that have suggested that the very skewed distribution of health-care facilities in Peru is a major deterrent against the use of modern medicine, and that recent efforts to expand the availability of preventive health-care services have not been successful in reaching large segments of the rural population. We found large differences in the utilization of both prenatal care and delivery assistance between the Lima metropolitan area and other regions of the country, with women living in the rural areas of the Sierra and the Selva being particularly disadvantaged. Using coefficients obtained from Model 4 (Table 4) we have predicted the use of prenatal care and delivery assistance, according to maternal education in the Lima metropolitan area and in the rural Sierra (see below).[12]

These results clearly illustrate the importance of region of residence in determining maternal health-service use. For example, our estimates show that a higher percentage of uneducated women in Lima used both prenatal care and delivery assistance (62.0 per cent and 72.0 per cent) than women with the highest level of schooling in the rural Sierra (57.7 per cent and 41.1 per cent). In fact, simply equalizing educational levels in the two regions would do little to eliminate the estimated differences in use. For example, if educational attainment of women living in the rural Sierra were to increase to the levels observed in the Lima metropolitan area, holding all other factors constant, we would observe about a ten percentage-point increase in the usage of both prenatal care and delivery assistance in the region; from 43.3 per cent to 54.3 per cent in the case of prenatal care and from 27.2 per cent to 37.5 per cent in the case of delivery assistance,[13] levels far below those observed in Lima. Although our regional covariate is a rather crude proxy for service availability, the skewed distribution of health-care resources is likely to be the primary factor behind the above results, though unobserved factors, not measured by our other covariates, may also play a role. Thus, our results, taken together with findings from other studies, suggest that much greater efforts to redistribute health-care resources on the part of the government are required if modern maternal health-care services are to reach women in rural areas.

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12 The adjusted percentages control for the effects of all determinants of health-service use included in Model 4 and are calculated as follows. First, a predicted probability for each birth in the sample is calculated based on the logistic-regression coefficients obtained from Model 4 in Table 4, and the assumption that all births belong to each education/residence category in turn, but at the same time retain their actual values with respect of all other explanatory factors. The adjusted percentage is then taken as the mean of the predicted probabilities of all births in the sample for each education/residence category. The overall percentage of use within each region is calculated as a weighted average of the predicted percentages with the proportion of women in each educational category as weights.

13 The predicted level of usage in the rural Sierra is calculated as the weighted average of the adjusted percentages by maternal-education category presented above for the rural Sierra with the distribution of women by educational level in the Lima metropolitan area as weights.

Table 5
Predicted percentages of women who sought prenatal care or delivery assistance

Mother's education	Lima		Rural Sierra	
	Prenatal care	Delivery assistance	Prenatal care	Delivery assistance
None	62.0	72.0	34.4	20.6
1–3 years	71.1	78.2	44.0	26.9
4–5 years	76.8	83.3	51.0	33.4
6+ years	81.6	87.9	57.7	41.1
Total	79.0	85.5	43.3	27.2

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Comment

From a high mortality regime to a high morbidity regime: is culture everything in sickness?

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In the inaugural number of *Health Transition Review*, S. Ryan Johansson notes that the health transition has been marked by declining mortality but rising morbidity and suggests that this can be explained as the result chiefly or exclusively of a cultural inflation of sickness (Johansson 1991). So much common sense lies behind the idea that cultural forces have played a role that, until Johansson's essay, no one had taken the trouble to specify which forces should be included under the rubric 'cultural'. Johansson provides a long and inclusive list of items that she believes capture changes in behaviour and belief, rather than in experience. She makes a conventional argument about them. Cultural forces, she claims, account for the inflation of morbidity, and the effect is sufficiently self-evident that no demonstration of it is needed.

To the contrary. While it is almost certainly true that changes in behaviour and belief account in some part for rising sickness, it is also true that non-cultural forces account for some part. Most recent research has been directed toward identifying non-cultural forces and assessing their effect. That research incorporates arguments that are more rigorous in their demands for evidence and logic than the arguments brought to bear by Johansson, who takes little note of the effects of non-cultural forces and who, in assessing the role of cultural forces, sets the task of merely showing what may be plausible. The argument for cultural forces needs not only to be specified in the quite useful way that Johansson has done by identifying certain forces. It also needs to be tested in the light of what is known about changes in beliefs and behaviours across periods of declining mortality and rising morbidity and across periods of rising mortality and declining morbidity. How much and in what precise manner are changes in behaviour and belief meant to have contributed to rising sickness? Furthermore, much of Johansson's argument relies on defining cultural forces so broadly that they include not only behaviours and beliefs but also a wide range of biological, medical, economic, social and even demographic forces. Cultural factors will count for everything if they are defined broadly enough. But broad definitions are misleading and vague and they impede the effort to address high morbidity as a human problem that may be alleviated, in a way analogous to the manner in which once-high mortality has been reduced.

Johansson's case for a chiefly cultural inflation of sickness is built on a series of assumptions and analogies, of which some are valid and helpful and others are invalid and misleading. In the nature of scholarly debate it is the invalid and misleading assumptions and analogies that deserve discussion in this note. But it is worth remarking that I have selected for discussion weak parts of an essay that has many strengths.

Inverse health transitions

Material conditions improved in the developed countries during their mortality decline, which promoted higher expectations about health. People suffering sickness learned to perceive it better and became more willing to seek professional help. Medical practitioners learned to recognize a growing number of diseases that rarely or never cause death. Summarizing her essay in this way, Johansson sets forth a seemingly familiar group of changes in behaviour and belief that account for some part of changes in the incidence and duration of diseases and injuries. The problems, however, are these: are these forces correctly specified as cultural? Are they accurately stated? What part can they be shown to have played in the inverse health transition? On one hand, people now die later in life. On the other hand, holding age constant, people also more often regard themselves as sick, they take more time off from work, they spend not only more of their income on health services but also a rising proportion of their income (Riley 1990a). In these and other ways, too, people act as though their health deteriorated rather than improved during the mortality decline.

One of the signal contributions of Johansson's essay lies in its acknowledgement that sickness rates have increased. In countries that achieved low death rates early, such as most of western Europe; in those that began later but made more rapid progress toward low death rates, such as Japan; and in regions that have only recently achieved low death rates, such as the Indian state of Kerala, sickness prevalence has increased. If people now live longer, it is tempting to suppose that in the process they escape health hazards that their predecessors, who lived shorter lives, did not. Hence their additional sickness may be illusory. In terms of the threat to survival that each sickness poses, the additional sickness is illusory. But the tendency people have shown more often to act as though they are sick, even to the point of sacrificing income and enlarging their health spending, argues that the illusion lies not in behaviour but in the way sickness is graded. A gauge often used to distinguish serious sicknesses from other sicknesses is the threat a given malady poses to survival. In the traditional scheme of things diseases with high case-fatality rates were rated as important. Beginning in western Europe in the eighteenth century public authorities devoted a rising share of resources to sanitary and public-health measures. To a substantial degree those investments paid off, reducing mortality and lowering the average lethality of episodes of infectious disease. But people did not respond to the mortality decline by being well most of the time. Instead they responded to it by believing and acting most of the time as though they were sick. The central problem is to discover why this happened.

Behaviours and beliefs

Using lethality as a gauge served public authorities and individuals well in an era in which the leading health losses consisted of deaths so premature that they made life expectancy at birth little more than 30 years. But already at the beginning of the health transition, which in western Europe was the eighteenth century, individuals were not acting as though lethality was the leading motive behind the decisions they made about health status and care. Already the ratio of medical practitioners to people – combining the licensed practitioners consulted by wealthier people together with the unlicensed practitioners and apothecaries that ordinary people consulted – was close to or above the modern ratio. Already people offering remedies of various kinds advertised their wares aggressively, using claims and appeals quite similar to those employed by their modern counterparts (Ramsey 1988). There was plenty of literature giving people advice about health maintenance and a range of home encyclopaedias describing commonplace diseases and remedies for them. What is more, people expected clergymen and socially prestigious fellow citizens to be able to provide them with useful health advice and care, supplementing that received from apothecaries, folk healers, physicians, surgeons and others.

Before the health transition began, individuals took a lively interest in their health, and strove to maintain it when well and to regain it when sick. They fretted about infections that, in their experience, might result in death. They fretted also, and attempted to treat, many maladies that could not be expected to result in death (Porter and Porter 1988). Ralph Josselin, a seventeenth-century clergyman, who kept a diary in which he commented on the health experiences of himself, his wife and children and a wide range of other matters, recorded colds as readily as he did smallpox and even took the trouble to notice occasions when his eyes gave off a watery discharge (Macfarlane 1976). The same story can be retold many times for many different people who provide us with autobiographical accounts of their health (Porter and Porter 1988). It was in the eighteenth century that hypochondria was transformed from a disease of the abdomen into an imagined ailment with no anatomical location. In short, in that high-mortality regime ordinary people were not guided in their thinking about sickness and wellness by the idea that potentially fatal diseases bulked much larger in importance than other ailments. Their diaries, correspondence and letters show that they wished to avoid discomfort and pain. The same sources reveal how difficult it was to avoid discomfort and pain because, when compared to modern remedies, they show how little the available advice and therapy did to restore people to health.

Nevertheless the important point, from the perspective of the cultural inflation of sickness, is this: before the health transition started, west Europeans fretted about their health, tested themselves for signs of sickness, bought medications and medical services and in other ways acted the part of people seriously concerned about their own health. Their expectations about health were already high and carefully formulated. It is almost certainly true that those expectations could increase and that they have increased since the eighteenth century. It is an error to suppose that health expectations began to increase from circumstances in which people expected to be sick and, when sick, disregarded their ailments. In short, the boundaries for the merely cultural inflation of sickness are in many ways rather narrow. If the point is to compare the mortality and morbidity experience of people in countries now developed before and after the health transition, it is essential to explore the culture of sickness and wellness in both periods.

It is seriously misleading to make inferences about health experience in eighteenth-century Europe from what is surmised about health experience in developing regions of the globe in the mid-twentieth century, as Johansson does, referring, for example, to a practice attributed to 'various parts of Africa' in the 1960s according to which people sick for more than three days were denied aid and comfort. Whether or not this is true about some parts of Africa in the 1960s, it is untrue about Europe in the eighteenth century or at any point since. Moreover, the profile of diseases that people suffer, the ways disease is treated and attitudes toward recovery are profoundly different between high-mortality regions of the present-day world and Europe before its mortality decline began. Long before the eighteenth century European folk medicine had incorporated Hippocratic and Galenic advice about what people should do when they are sick, and the remedies that people used were often efficacious in the terms of those regimes. That is, they had the desired and expected result: they made the bowels move and they caused people to sweat, bleed and vomit. Long before the mortality decline began people attempted to treat their maladies and reacted to ailments in ways that can readily be accommodated within the modern range of reactions: self medication, consulting friends and associates, taking time off from work, seeking professional help and seeking institutional care.

Cultural forces and policy choices

In the developed countries of the world the health transition has progressed to the point at which it is now feasible to discuss redirecting attention from a primary focus on efforts to reduce mortality, salvaging for survival more of the potential life expectancy of humans and toward a primary focus on reducing morbidity, salvaging a healthier rather than a longer life expectancy. For this reason

especially the health experience of the countries that are now developed, and which had made some substantial progress toward economic development before their health transition began, has been an important source of insight about how mortality might be reduced in countries that entered the health transition later. If Western European experience furnishes information about means and measures of mortality control, it is also an important source of information about why morbidity has increased and therefore about what may be done to influence that trend. The point, of course, is not to find the most efficacious path to high morbidity, but to find in the experience of developed countries paths that short-circuit the inverse trends of mortality and morbidity.

By describing the problem as one that is chiefly or exclusively cultural, Johansson implies that the strategies for addressing the problem of too much sickness should be chiefly or exclusively cultural. The problems she addresses certainly exist. They help account for additions to the sum of sickness that are both real and perceived, that are serious and frivolous. As Johansson recognizes, most of the cultural factors she cites as having contributed to rising sickness rates can be regarded as achievements. It is, for example, a good thing that more people are able to recognize diseases that, in earlier days, they might not have been able to identify. But identification should not be confused with existence. Judging from the assurance with which their clergymen diagnosed smallpox as a cause of death in eighteenth-century Sweden, where most localities lacked trained medical practitioners, it appears that lay people readily recognized that disease. They did not recognize tuberculosis so readily and they had a difficult time identifying many diseases whose external signs are weak or which create no signs on the outside of the body, such as heart disease and many neoplasms. Nevertheless, they suffered tuberculosis and it is plausible to suppose that they also suffered heart disease and cancer. It is true that cultural changes concurrent with the health transition have added to the list of diseases that people report and to the security of the identifications now made. But it does not follow that people did not suffer from a disease before they or their physicians could name it.

Inflation and deflation

The list of diseases that physicians and people identify has grown longer. Some diseases that seem to have been long familiar have been identified; many more have been subdivided because enough was learned about their characteristics to recognize separate strains, such as in influenza; and a few new diseases have appeared to afflict humankind. Some of these newly identified diseases are associated chiefly with biological characteristics, such as micro-organisms, and others with emotions.

It is also true that there has been a deflation of sickness. Diseases familiar before the mortality decline have become unfamiliar and a few, such as smallpox, have disappeared. That is a larger matter than may appear at first glance. Smallpox was the leading cause of death in eighteenth-century Europe, accounting for ten per cent or more of all deaths. Virtually the entire population was exposed to smallpox and most people – and in cities nearly everyone – contracted the disease. Across much of the nineteenth century tuberculosis was equally widespread as an infection and a cause of death. To withdraw merely these two diseases from the profile of maladies that people suffer in the late twentieth century – taking smallpox completely out and counting tuberculosis only by the small proportions of people who have died in recent years or have tested positive – is to withdraw two diseases that in their heyday were nearly universal in occurrence. If maladies as commonplace as these have disappeared, or at least become uncommon, then how can sickness rates have increased?

How sickness rates have increased

Recent research has explored an array of forces that have contributed to rising sickness. The mortality decline itself changed the composition of the population (Alter and Riley 1989). Mortality has often been a weaker force than fertility in shaping the population's age structure, but in recent decades

declining mortality among the old has far outstripped fertility as a force behind population ageing. In the aggregate, today's population demands more health services and spends more time in sickness because it is older and thus at a stage of life where the risk of sickness is much higher and where also the propensity and the ability to pay for health services are greater.

The point of particular importance is that, and at specific ages, the sickness rate has increased as mortality has declined. The average age-specific duration of sickness episodes among insured males in Britain increased between the 1870s and the 1890s, a period in which the mortality of adults fell sharply (Riley 1987). In the era of health surveys in Japan, the United States and Britain, sickness prevalence has increased as mortality has decreased (Riley 1990b). An inverse association has obtained also in Hungary, the one country where disease prevalence has been surveyed across a period of increasing mortality. Health surveys undertaken there in 1981 and 1986 show that the prevalence of chronic sickness among adults decreased while mortality increased (Riley 1991). If the addition of new survivors augmented sickness rates in populations experiencing a mortality decline (Verbrugge 1984), Hungarian experience suggests that rising mortality promotes a withdrawal of non-survivors whose earlier death contributes to lower sickness rates.

A striking feature of comparisons of mortality and morbidity trends is that each one so far reported shows an inverse association: when mortality falls, morbidity rises and *vice versa*. Another striking feature is that this inverse association occurs in comparisons across short and long periods. It is plausible to suppose that cultural forces might have changed in Britain between the early 1870s and the mid-1890s or in Japan between the early 1950s and the mid-1980s, between which mortality decreased and morbidity increased. Hence it is plausible, even without specifying which cultural forces operated, and how they operated, to suppose that changes in behaviour and belief pushed morbidity upward. But the surprising finding is that inverse shifts in mortality and morbidity as large in proportion have occurred across short periods in these two societies and elsewhere. What are the grounds for supposing that cultural forces may have promoted a substantial decrease in sickness in Hungary between 1981 and 1986, or a substantial increase in the United States and Britain during the 1970s? What specific beliefs and behaviours changed so substantially in those brief periods? Johansson calls attention to imposing changes in behaviours and beliefs that occurred over decades, even centuries. But the increase of sickness has not required the passage of decades or centuries.

Another non-cultural factor that has been shown to have contributed to rising sickness rates is the successful treatment but not the successful resolution of diseases, a phenomenon Gruenberg designated the 'failures of success' (Gruenberg 1977). Many diseases cannot be cured. Some of these will eventually result in death. Others will not, but the people suffering them will die from other causes before they recover from the original malady. In treating the chronic diseases of adulthood, Gruenberg points out, modern medicine has been especially adept at finding ways to push back the point at which death occurs. This has added substantially to the sum of sickness time because sickness episodes have been prolonged so much. The same force has added yet more substantially to spending on medical services.

Many of the sicknesses associated with rising morbidity do not threaten survival. One recent estimate suggests that, even in disabling diseases, only 36 to 41 per cent of sickness occurs because of diseases that may cause death (Chapman, LaPlante and Wilenski 1986). Most disabling diseases do not cause death. Since it is disabling, this sickness is serious. In terms of individual and social costs, this kind of sickness is the modern equivalent of the often fatal diseases of the high-mortality regime.

To these explanations may be added other economic, social and medical forces that have contributed to rising sickness rates. As the discussion above indicates, it cannot be shown that the ratio of medical practitioners to people increased between the eighteenth and the twentieth century. But it

can be shown that the proportion of their incomes that people spent on health services and health insurance increased, with particular force beginning in the nineteenth century. It can also be shown that the earlier detection of disease has added to the length of time during which people acknowledge that they have a disease, although not necessarily to the length of time for which they suspend ordinary activities. These and other contributors have a cultural component, but that component is not by itself sufficient to explain why sickness has increased.

Trends

It is too early to know what will eventually be learned about sickness trends that will shed light on the power behind each of the biological, medical, economic, social, demographic and cultural forces contributing to sickness increase. But enough is known already to formulate an hypothesis (Riley 1989). In the high-mortality regime that existed before the health transition began, sickness was commonplace. But it was also mostly brief in duration. Measured by incidence, the pre-transition society lived under a heavy burden of sickness, one that over time would shrink. Measured by sickness duration, however, the burden would grow rather than shrink. It would grow especially in this form: both the incidence and the average duration of sicknesses suffered by infants and children would diminish. People in those age groups have come to be better protected from the risk of falling sick and, especially since the 1930s, they have benefited also from new medications that shorten the course of many childhood maladies. In the developed countries today's children are sick much less often than were their counterparts in the eighteenth century. But the reverse is true of adults. In the pre-transition scheme adults, like children, suffered often from infectious diseases having an acute course, as well as from chronic diseases. Since then their susceptibility to acute diseases has diminished, but their susceptibility to chronic diseases has increased. Most importantly, the sickness time added in the form of more and longer chronic diseases far exceeds the sickness time subtracted in the form of fewer acute diseases. For the individual, and for society, the burden of sickness has increased chiefly because the average duration of sickness episodes has expanded.

That, in itself, is an important insight. Discovering that the average duration of sickness episodes has increased weakens the force of any argument that cultural factors alone are responsible for rising sickness. That is so for the simple reason that what has increased is not the number of experiences that people count as sickness, but the duration of those experiences.

Conclusion

The point of investigating the reasons why sickness rates have risen is to discover how to create a low-morbidity regime. That evidently will require different strategies from those followed to reduce mortality. To identify strategies appropriate for reducing sickness requires that the causes of rising sickness be understood. To allocate resources among strategies requires that the relative importance of all of the forces contributing to rising sickness be understood. Johansson wishes to assign to cultural forces a leading or even an exclusive role without first showing how far changes in behaviour and belief go toward explaining rising sickness rates. The evidence now available suggests that sickness rates have risen and are continuing to rise chiefly because of an extension in the average duration of the chronic maladies of adults. Cultural forces may have played a role in prolonging the average duration of sickness episodes, but that role appears to be much smaller than the part played by medical advances, which have deferred death in fatal diseases and in old age, and by the wide-ranging forces that have added new survivors to the population.

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Measuring the cultural inflation of morbidity during the decline in mortality

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Barriers to measurement: the standardization of meaning as a pre-condition for measurement

James C. Riley takes issue with the generalization that the inflation of morbidity during the decline of mortality was primarily a cultural phenomenon. As Riley points out, most recent research, including his own, has been 'directed toward identifying non-cultural forces and assessing their effect'. He correctly raises issues related to measurement, and to the possibility of estimating to what extent non-cultural changes, largely of a biological nature, are responsible for the rise in reported morbidity levels which appears to accompany the decline of mortality during development.

But in pointing out my apparent failure to measure morbidity, Riley seems to have overlooked the major concern of my essay, the conceptual preconditions that make meaningful measurement possible. 'Morbidity', or 'sickness' as it is currently used in the literature is too vague a term to permit the meaningful measurement of historical trends. Apparently, I should have made it clearer that in discussing the concept 'morbidity', and at such length, I was making a point often made in the philosophy of science: standardizing a definition for research purposes constitutes a conceptual problem which must be solved within a research community before scientifically meaningful measurement can proceed, and experts can begin to converge on the most likely explanation for a trend.

My essay was an attempt to explain how historians or health scientists could move towards meaningful measurement by reducing the natural vagueness which attaches to the concept 'morbidity' (or sickness) by explicitly addressing its multi-dimensional complexity, and separately considering how cultural and biological influences impinge on each dimension over time. From my perspective, Riley wishes to continue treating the history of morbidity trends as if that history were no more conceptually problematic than the history of mortality trends or heights. But since being sick or healthy is not as biologically clear-cut as being dead or alive, or being five feet eleven inches rather than six feet tall, morbidity history is inherently conceptually confusing in a way that mortality history and height history are not.

Without reaching some agreement on how to convert vague vernacular concepts into technical terms, historians of health will continue to talk past one another, as James Riley and I seem to be doing. Therefore I am grateful to Riley for raising questions that once again take us back to the most fundamental issue in health history: why it is so difficult to define morbidity or sickness in a way that makes it simple to measure, or to interpret quantitative data, in either the present or the past.

Judging by Riley's reply, I did not get my main points across. I will therefore try to make them in another way, and then conclude by demonstrating how cultural influences on morbidity data could be quantified by treating them demographically,as if they involved various forms of mental migration which could be measured historically.

Measuring trends in height, mortality and morbidity

Consider the following three generalizations about the historical experience of Western Europe, and its offshoots: (1) the level of mortality fell as development proceeded; (2) the mean height of mature males has risen over the last two hundred years; (3) the level of morbidity/sickness has risen (or fallen) in the last two hundred years during the course of economic development.

The first two are informative but vague generalizations about trends over time; well established conventions exist for converting these implicitly quantitative generalizations into a more precise form for analytical or historical purposes. In the case of the third generalization, about a possible trend in morbidity levels, it is much more difficult to go from a level of colloquial vagueness to a precise form permitting quantification, because 'morbidity' is a complex, multi-dimensional concept, that has several possible, quite different meanings. No metric encompasses all of these meanings.

When historians do height history, for example, it is relatively easy for them to establish whether or not mean heights of various populations have risen or fallen over time, in one country or in several, because the ordinary-language meaning of 'height' is already one-dimensional.[1] Since 'height' has only one dimension, it is immaterial whether we use inches or centimetres to measure it, so long as both are standardized units which can be converted into one another. Moreover, contemporary historians

[1] Floud, Wachter and Gregory (1990) discuss the problems of measuring height using the various available sources. Most of the problems are technical in nature and have little or nothing to do with cultural influences.

share the same meaning of height and use the same rulers as the people who actually gathered and reported height data in the past. Thus the height data gathered by past army recruiters in one country can be analysed by present social scientists who live in another. Height historians do not need to figure out which of several possible physiological characteristics the army recruiters in country X were trying to measure when they recorded data on the height of potential recruits. Historians do not need to cope with the fact that centimetres and inches may have been of different lengths in different countries, nor do they need to consider the extent to which the real length of an inch stayed the same over time, even in one country. Moreover recruits were never asked to say how tall they felt, or thought they looked. So long as the recruiters used normal rulers and had no incentive to misrepresent the heights of potential recruits (if they did, then cultural influences on height reporting would have to be considered) measuring trends in the mean height of recruits is a relatively straightforward social-scientific undertaking in which the role of cultural factors can be consigned to the margins of research. (This assumes that the data are published in a precise form, that vague categories like 'short', 'medium' and 'tall' are not used, and that data about recruits are age-specific, and representative of the total population of adult males.)

Similar observations could be made about mortality and its history. Like height, mortality is a one-dimensional concept. Once a system of vital registration has matured sufficiently to encompass most deaths, the cultural barriers to measurement are minimal. Throughout human history the concept 'dead' did not change over time, and virtually everyone everywhere could tell the difference between a living body and a dead one.[2]

But when historians want to measure trends in morbidity over time, they are forced to start by using a very fuzzy colloquial term which has more than one meaning. These meanings can be conceptualized as the separate dimensions of sickness. Epidemiologists have succeeded in standardizing the measurement of each dimension to a certain extent, but they have not and cannot standardize the concepts involved in each dimension because everything associated with health and sickness is so intrinsically cultural.

Cultural considerations are about the learned forms of human behaviour associated with the biological states that are classified as disease. Cultural considerations determine how much disease or sickness is perceived as illness (particularly in its non-extreme forms), and how much perceived illness is reported. Because we live with others from whom we expect help, we must learn to tell the difference between being sick and being well in a culturally acceptable manner which is variable over time and space. Learning to identify sickness is also linked to community-level (or government-level) incentive systems. Various institutions embody those incentives through the distribution of rewards and punishments related to sickness. Incentives are culturally constructed from the specific standpoint of discouraging or rewarding the decision to perceive oneself as sick, to report oneself as sick, or to report that someone else is sick and needs treatment. Therefore reported morbidity, which is what we observe directly in all the morbidity data we have, and which is not based on something like a physician's examination or a laboratory report, has to reflect, first and foremost, how people were educated to identify and report disease, and the extent to which they are responding to the incentives associated with sickness. The reporting process is linked to the biology of disease, but not in a way that is as clear as being dead and being reported as dead, or being tall and being measured as having a height of six feet.

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[2] As noted in Johansson (1991) the progress of modern medical technology has begun to blur the formerly crisp boundary between life and death, and thus to make the definition of death into a problem which requires a culturally negotiated solution, since not everyone agrees that brain death should be preferred to other forms of biological failure.

The weak links between biology and behaviour in morbidity reporting force health historians to become cultural historians to an extent that height and mortality historians need not be. I apparently did not convey this message clearly in my previous essay. Nevertheless, the obvious point remains: cultural considerations are fundamental to the production of the morbidity data that health historians use. In a very real sense they take precedence over biology in the interpretation of data. There is simply no way to explain observed trends in sickness without first understanding the reporting system that produces morbidity data and how it reflects the way people and doctors are trained to perceive, detect and report sickness, and the differential incentive systems (social and economic) that shape reporting behaviour over time at the individual or institutional level.

Therefore, when an historian talks about sickness as if it were a biologically obvious state (like being dead) and proceeds to assume that some form of morbidity data can be used to measure biological sickness (as if measuring the biological extent of sickness over time were just like measuring height) and then attributes an observed trend to biological factors, he or she cannot be doing health history in a meaningful social-science sense. Since all the available quantitative data on sickness are defective in some way and come in a number of inconsistent and incomplete forms that chronically confuse the biology of disease with the sociology of illness, the facts, even the quantitative ones, can be made to say anything the historian wants them to say, once he or she has fallen into the habit of treating morbidity as a simple, one-dimensional concept (like mortality or height) and the associated data as a direct reflection of the biology of disease.

The social-scientific history of health begins with confronting the problems created by the cultural complexity built into concepts like ‘health’, ‘morbidity’, ‘sickness’ and ‘illness’. These problems precede meaningful measurement or the interpretation of quantitative data.

Barriers to measurement: measuring the separate dimensions of morbidity during the decline of mortality, given that the available data are defective or incomplete

Epidemiologists have long recognized that the study of diseases, as biological phenomena, is complicated by individual and institutionalized influences on the identification and reporting of disease. Thus it is common to distinguish the study of the epidemiology of sickness, as the biology of disease, from the sociological study of sickness as illness, or perceived and reported disease. Nevertheless, even when it seems legitimate to assume that some type of morbidity data is accurately reflecting real biological changes, quantifying those biological changes remains a complex demographic undertaking.

Approaching sickness and disease as purely biological phenomena which can be measured demographically begins with understanding that four separate dimensions must be measured, namely, frequency, duration, severity and depth (or co-morbidity). The demographic biology of disease is concerned with trying to measure (1) how frequently individuals get sick; (2) how long sick individuals stay sick; (3) how sick they are; and (4) how many identifiable diseases they are sick with at one time, always assuming we can hold the cultural meaning of sick constant while using a particular data set.

By extension, if morbidity history is to be approached as the history of disease and not just the history of reported illness, the historian has to reconstruct simultaneously its four separate dimensions, and thus produce four separate grand trends. Epidemiologists have devised standard measures for each of the dimensions of disease. Frequency is usually measured with cross-sectional incidence rates in some form; duration with measurement of person-days spent in the sick state during some time period; severity with time-specific case-fatality rates; and depth with cross-sectional co-morbidity rates.

At the beginning of the mortality transition no one anticipated that a multidimensional morbidity transition would accompany declining death rates, and no country therefore institutionalized morbidity

reporting to the extent that mortality registration was institutionalized.[3] Morbidity data have been gathered fitfully for various biological and cultural purposes, by various institutions none of which coordinated their efforts.[4] Thus we have no historical equivalents in the history of morbidity that are as good as our estimates for death rates, or as continuous, nor do we have a continual series of physician-generated assessments of the health status of a European population during the health transition based on detailed examinations supplemented with laboratory reports. Most extant data concern reported illness, and roughly approximate the biology of disease in a certain time and place. Morbidity data must therefore be approached by historians as the product of the cultural forces that affect reporting behaviour. The data might also tell us about the underlying biology of disease, if we carefully consider the four separate dimensions.

All historians of health can do is to use whatever defective or incomplete data happen to have been generated, for whatever purposes, in order to estimate the most likely historical trends in each of the separate dimensions of morbidity. Inevitably, various assumptions about missing data must be made. Riley's critique of my essay presents as disconcerting or problematic that, in discussing the history of morbidity, I made such assumptions. The only difference between me and other historians of health is that I made explicit assumptions. When historians do not fully specify their own assumptions, they may conceal from themselves, and others, their naivetŽ about the defective data that history forces them to use.

Because reported morbidity data do not primarily represent the changing biology of disease, both of the following historical generalizations are true: (1) morbidity levels have risen over the past two hundred years; (2) morbidity levels have fallen over the past two hundred years.

The first generalization will be true if by 'morbidity' the generalizing historian is referring to data which pertain to the frequency and duration of all *reported* forms of morbidity. The second generalization will be true if the historian is implicitly referring to the diminishing extensiveness of the very *severe* infectious diseases, exposure to which significantly raised the probability of dying over a short time period.

Obviously, a rise in the frequency with which non-severe forms of illness have been reported can produce an extremely rapid inflation of morbidity even as death rates continue to decline, or while they remain stable. Such inflation has been observed in all or most of the developing countries in the past few decades. But earlier, as mortality declined, there was clearly a parallel decline in the severity of the most frequently reported diseases. It is because exposure to the most severe infectious disease was reduced that mortality decline occurred.

Changes in the level of co-morbidity, particularly concerning severe diseases, have not been measured at all during the mortality transition. Historians can do little but make assumptions about this dimension of sickness history since even now we know so little about the extent of co-morbidity in modern societies,or how to standardize its measurement. For example, I assumed that as the rapidly lethal infectious diseases were brought under control, the extent to which the average person was simultaneously assaulted by two or more severe diseases also declined. Thus the underlying extent of severe co-morbidity must have declined. The desire to do more than make assumptions about the

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[3] Mercer (1990) reviews the evolving attempts made by various European countries to gather more, and more complete data for specific infectious diseases in the course of the eighteenth and nineteenth centuries. Mercer is careful to distinguish real trends in disease from reported trends based on changes in the completeness of reporting.

[4] Even now, cross-national coordination remains only an imagined possibility, and cross-national comparisons using morbidity data are hazardous.

history of European co-morbidity set me to using data from other places that still have relatively high levels of mortality. At present such societies are non-Western.

Although Riley objected to my using material from African societies, what is biologically true of African bodies when mortality remains high is likely to have been biologically true of European bodies when mortality used to be high. This does not mean that the same diseases would have ravaged both sets of bodies, or that both populations would have had the same genetic adaptations to each specific disease. What is similar between the two sets of bodies is that when mortality is high,the ordinary person is likely to have more than one form of disease at once, especially more than one form of relatively severe disease. That is the most obvious, if unobserved, biological foundation for observed high mortality wherever it exists.

As discussed in my earlier article, ordinary people living in high-mortality regimes who have been examined by modern physicians do suffer from high levels of co-morbidity including relatively severe diseases which would be considered disabling in a modern society. Moreover,anthropologists who observe the same non-Western populations have also noted that people who would be sick by modern standards continue to work, or otherwise live normal lives,even though, if they lived in a modern Western culture they would disrupt their ordinary routines in response to such conditions. We have no biological reason to assume that the same was not true of peasant populations, or even high-mortality upper-class populations, in Europe at the beginning of the mortality transition.

It was interesting to note that, having criticized my use of biological data about human bodies from other places to reconstruct the probable history of co-morbidity levels in Europe, Riley used some qualitative 'data' produced by privileged Europeans in the eighteenth century to generalize about European illness-reporting behaviour as found in diaries and journals. Eighteenth-century literate Europeans who kept journals and diaries were members of high-income, upper-class or middle-class families. Because they already enjoyed incomes and lifestyles that placed few constraints on the translation of pain or discomfort into socially supported forms of illness, they were as free to complain about their health or disrupt their routines as modern people who live in contemporary high-income countries. Sociologically, exceptionally privileged eighteenth century Europeans had more in common with ordinary modern Europeans than they did with the poor struggling peasants and landless workers who constituted 80 to 90 per cent of Europeans alive in the eighteenth century.

To use class-specific specific qualitative data, produced by literate, middle or upper class eighteenth-century Europeans, in order to generalize about illness behaviour among all Europeans, is to make an assumption so ahistorical as to deny the past and strip the European health transition of its transitional character. In effect, Riley is using a cultural source to try to hold culture constant, a move that implicitly takes the history out of human behaviour. If we could hold culture constant during the health transition, then it would be legitimate to interpret morbidity data as if it described changes in the biology of disease. I see this assumption as pervading Riley's work on the history of morbidity, whereas I assume that both culture and biology co-evolved as the health transition unfolded in Europe.

If anything is obvious or factually certain about the long health transition that occurred in Europe and its offshoots it is that the meanings, norms, conventional behaviour patterns,and policies associated with illness and reporting illness have changed dramatically over time, and that these changes are intrinsic to studying quantitative data. This salient fact should prevent historians of health from equating real sickness with reported illness during the health transition, or from writing as if cultural changes could be marginalized. The co-evolution of culture and biology means that the various measures devised by epidemiologists to study disease involve measuring rates with a rubber ruler, at least whenever we deal with time periods longer than a decade in the twentieth century, and several decades in the nineteenth century when scientific progress was not so rapid.

To study the co-evolution of both culture and biology during Europe's health transition,historians must be more than narrow empiricists who are prepared to observe and measure only what can be observed or measured using the limited range of data for the time or place in which they specialize. As John Caldwell (1990) has been trying to demonstrate, the health transition is a world-wide process which has certain similarities, despite its country-level and regional differences. Everywhere, fundamental cultural problems must be solved before the health transition succeeds in eradicating or reducing the severe infectious diseases which are so closely linked to high levels of mortality. These cultural problems involve changing how people are trained to conceptualize disease, and what kind of health practitioner they prefer to consult.

Modern health scientists, who think of science as quantitative, everywhere resist acknowledging the important role that such cultural changes have in actually changing the biological extent of disease, just as they resist the idea that cultural influences are fundamental to the production of morbidity data. Epidemiologists are happy when they tackle complex measurement issues related to the various biological dimensions of disease, because these dimensions are, in principle, measurable. But changes of a sociological nature, based on socially constructed meanings, norms, conventionally acceptable behaviour patterns that differ by age, sex, social class, ethnic group, religious group, and government policies, look unmeasurable, and hence are shunned.

So, as a sort of last try to make demographers, epidemiologists or any quantitatively oriented health historian more comfortable with the importance of cultural influences on morbidity history, let me briefly explore the extent to which cultural changes during the health transition in Europe can be approached demographically,and measured, as if they were about mental migration patterns from one sector of a population to another. From this demographic perspective, the health transition concerns the individual's relocation from a perceived state of health into a perceived state of illness, given that there are culturally constructed rules that control the issuing of visas in the form of the rules governing the allocation of health resources. These visas, which can be measured in various ways using statistics about the economics of health, permit the outmigrant to leave the free and productive state of health and to enter the costly state of illness for a given time period at the expense of others.

Overcoming barriers to measurement: treating the culturally driven inflation of morbidity as a form of mental migration

At least two centuries ago, at the beginning of the health and mortality transition in modern Europe, almost everyone (let us estimate that as meaning 85 to 95 per cent of the total population) lived in the sector of the population that did not report being sick to anyone prepared to record that report and link it to other comparable reports that could be counted, summed and stored. When ordinary people felt sick they complained about it to their friends, families or diaries (if they could write and afford to buy a diary, which most people could not) or they took to their beds and medicated themselves,or they simply carried on in various degrees of pain and with impaired levels of function. If they could not manage to perform their usual routines at all they consulted a European folk practitioner who did what he or she could. Nothing in the informal institutions associated with European folk medicine encouraged keeping morbidity data in written form. So much the worse for the health historian who wants to quantify the real level of disease at the beginning of the health transition! Most real disease went unmeasured and most illness behaviour went unreported.

Even in the eighteenth century, at least 5 to l5 per cent of the European population came into regular contact with doctors who already considered themselves scientifically trained. (Whether or not they were by modern standards is another story.) Most of the physicians already had a professional commitment to reducing the real level of disease in their societies, and Riley (1987) has written very

informatively on that subject. Towards that end they began to gather and report some morbidity data for their communities, the hospitals they were connected with, various private insurance companies or employers, or various governments who were trying to improve the public's health; but such data encompassed only a fraction of the total population. Gradually, physicians began actively to sponsor a form of mental migration from the folk-medicine sector of the population, where traditional theories of disease still governed the explanation of causation, and the use of traditional folk remedies still dominated treatment. This migration took less educated, uneducated and illiterate Europeans out of the traditional mental sector, in which disease was a punishment from God or the result of witchcraft, into the modern mental sector of the population in which disease had scientific causes and remedies.

If we had data on this mental migration from one sector to another we could observe a gradual, but not necessarily regular, rise in the proportion of Europeans who mentally resided in the medically modernized sector of the population, and who were thus at an increasing risk of having their illness behaviour reported or recorded in some way.

After two hundred years, at least 95 per cent of the population had been transferred to this mentally modernized sector. Among other things, this sector continues to be responsible for producing morbidity data, and continues to produce more and more of it. Holding biological factors constant, this culturally-sponsored mental migration would have greatly inflated the incidence of reported morbidity given that the entire population was already being enumerated. This inflation of reported morbidity is demographically similar to urbanization, which was a physical transfer of the majority of the population from one geographic sector to another. Medically modernizing a population which is already completely enumerated will inflate the apparent extent of reported morbidity.

But of course we cannot hold everything else equal during the mental modernization of the European population. As the health transition proceeded, the scientifically trained doctors who populated the modern health sector became more and more scientifically sophisticated. They were constantly retrained to detect and report new diseases, and institutionally required to engage in more and more diverse forms of illness reporting. If we could count the number of named diseases that scientifically trained doctors were prepared to report at the beginning of the health transition, and prepared to teach their patients about, I would guess that at the very least this list of named diseases doubled in size over two hundred years.

This increase in the population of named diseases contributes to both the incidence-related inflation of reported morbidity, and the decline in the severity of the most frequently reported diseases. For the most part the diseases formerly reported were quick killers, that is they had a high level of severity, that took the lives of many people. The newly discovered diseases afflicted fewer people, and killed them more slowly. The last development contributes to the biological rise of any measure of prevalence, but, once again, this rise is also a function of better knowledge (most of the new diseases existed previously but were undiscovered), more diligent diagnosis and society's willingness to pay for the treatment of extended forms of disease. But this increasing population of reported diseases was not such as to interfere with the fact that a small but select list of reported diseases was being brought under public control. Declining incidence rates for a few severe infectious diseases were linked to the modern decline of death rates. The slow addition of new, less severe diseases to the list of reportable disease kept up the pressure for a gradual inflation of reported morbidity, while gradually divorcing that rise from trends in mortality.

Perhaps the most profound change which took place in the modern medical sector was the increasing extent to which its ordinary residents were institutionally encouraged, indeed invited, to migrate from well to sick,and generously rewarded for staying that way for longer and longer periods. Imagine going back to the beginning of the health transition and calculating the economic cost of each

reported illness (as the amount of money it cost to pay a doctor, keep someone in a hospital, or pay for time off work and so on). Because income was so much lower than now societies could not afford to sponsor much migration to the state of illness, and the scarcity of resources strictly limited the number of visas that permitted lengthy stays in the state of sickness. Even people who had mentally migrated to the medically modernized sector of the population had to limit their costly visits to the doctor, hospital stays,or their expensive time off work. By so doing they limited the amount of reported morbidity and the apparent duration of sickness associated with each individually reported incidence of sickness, however sick they really felt and for however long.

One of the biggest changes in human welfare over the past century has been the increasing propensity of developed countries to allocate resources to the support of those afflicted with either infectious or chronic diseases. Society no longer limits care to the terminal phases of any disease but increasingly tries to cure or care for the afflicted person from first diagnosis to recovery or death, while detecting disease in earlier and earlier phases. Few controls on the length of care or the cost of trying to cure disease have ever been institutionalized.

Doctors, who benefit economically from society's generosity to the sick, have no professional incentives to control the cost of paying for curing or caring, and thus no incentives to limit the amount of routine migration from wellness to sickness, or the number of visas they issue that permit people to reside in the sick state for long periods. As long as the payments for being sick approach the ordinary wage, people who do not feel in perfect health can use sick leave to leave the state of health whenever they feel mildly under the weather. To people who feel well, generous sick-leave policies constitute a free visa for vacations in the reported state of illness.

Even when we are dealing with the retired population, the generosity of society encourages more frequent and longer departures from the state of wellness. Ageing has always been associated biologically with increasing sickness and disability. In former times society simply refused to pay for this real, age-related increase in sickness and disability and older people were forced to tolerate increasing levels of pain, and their families or villages to cope with caring for them as best they could. Home care went unreported and the cost of it was privately borne. But who would doubt that there was once a huge reservoir of socially unreported, unsupported disease among the aged?

Society's recent and commendable generosity with respect to the welfare of elderly people gradually drains this reservoir of unreported sickness, and sponsors the transformation of unreported forms of real sickness, into reported forms of illness. Even if real sickness were declining among the aged, especially among younger old people, reported levels of illness would rise as society issued the economic visas which permitted the old to migrate to the state of illness and reside there.

The cost of paying for morbidity among the elderly is certainly influenced by the increasing percentage of the population over age 65, but if only demographic changes were at work the costs of paying for disease among the elderly would rise only as fast as their representation in the total population. Everyone knows that the cost of paying for medical care for the elderly is soaring much faster than would be demographically justified if everything else stayed constant. Determining the extent to which this rise is biologically real or sociologically induced is not productive. The biological problems of the elderly have always been real. The main question is, to what extent was sickness among the elderly underreported, even two decades ago? After that question is answered we can begin to estimate to what extent the elderly may or may not be more biologically frail than their counterparts at an earlier date. My guess is that the cultural propensity to support frailty is probably dominating trends, not an increase in real biological frailty. But this is a hypothesis. It cannot be proved or disproved by assuming that morbidity data are a straightforward reflection of the biology of disease among the elderly.

So long as little or no institutionalized controls are imposed on the collection of generous benefits for not being in perfect health, people of all ages will readily migrate to the expensive state of being sick, and stay there as long as it is comfortable, or advantageous, to do so. Limiting this process of migration means culturally negotiating how to draw an ethically acceptable line between being sick and being well on a health or sickness continuum that, as explained earlier,has no naturally identifiable, biologically obvious, break point. Limiting the migration to the socially expensive state of illness is a problem in applied ethics which a confusion of the biology of disease with the sociology of reported illness will do nothing to clarify.

Achieving consensus about the history of morbidity

Riley asks: is culture everything in sickness? Epidemiologically, sickness is about the biology of disease not the sociology of illness-reporting behaviour. But most morbidity data from both the present and the past continually confound illness and sickness. Our present ability to estimate the extent of real disease, by distinguishing it from non-biological changes in the propensity to identify and report illness, is still imperfect. Distinguishing between the two in the past is even more difficult. If quantifying historians want to disentangle the two, they must first understand the cultural mechanics of illness reporting during the health transition. That was the main point of my previous article. Such an understanding is prior to meaningful measurement.

Moreover to quantify the health transition in Europe the historian must use both biological theory and data from comparable cultures that are in the early stages of the worldwide health transition. Our historical data cannot tell us much about the biology of disease, with the possible exception of those data series involving the accurately recorded number of cases of severe infectious diseases which attracted so much attention during the mortality transition.[5]

Although measurement-minded morbidity historians cannot escape from the frustrations of having to deal with rubber rulers, their efforts to measure the biological aspects of disease will be most successful if it is assumed from the start that cultural forces are responsible for most forms of rising levels of reported morbidity during the health transition. If death rates are declining as morbidity rises, some form of culturally influenced reporting is most likely to be inflating morbidity. Only when both morbidity and mortality rise together, and to approximately the same extent, is it safe to assume that the biology of disease is driving the reported rise of morbidity, and that changes in the mechanics of reporting can be analytically marginalized.

In those developing countries currently in the middle of the health transition, issues pertaining to the measurement of morbidity are particularly pressing since they are so closely tied to health policy. Today,the health transition can proceed so rapidly that nothing can be held constant from year to year – neither the proportion of people in the medically modernized sector of the population, nor the diseases which most doctors are prepared to report, nor the kinds of sickness that institutions are prepared to support. In the midst of so much change there is one rule of thumb. If mortality levels are falling as development proceeds, but reported morbidity levels are rising, check whether the reported diseases are increasingly less severe, that is, that they have a lower probability of causing death in a short period of time than reported diseases used to do. If so, the historical experience of Europe is probably being reenacted, and the cultural inflation of morbidity has begun. This is a good thing from a welfare perspective. If, as appears in some Central European countries, both disease-specific incidence rates

5 Szreter (1988) considers the extent to which the probable overreporting of tuberculosis as a cause of death in the middle of the nineteenth century accounts for much of its apparent decline in the late nineteenth century. Most health historians have assumed that the observed decline was real, and not the result of reporting practices related to the changing level of expertise of those reporting causes of death.

and mortality rates are rising together, then some form of biological deterioration is likely to be at work, especially if the apparent severity of the most frequently reported diseases is rising. The historical experience of Europe, properly conceptualized, can illuminate present-day illness-reporting behaviour,just as the modern history of developing countries can be used to illuminate Europe's dim biological past.

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Forum: On the demography of South Asian famines

This issue of Forum is devoted to a discussion of Tim Dyson's articles 'On the demography of South Asian famines' which appeared in *Population Studies* 45 last year.

Age-patterns of famine-related mortality increase: implications for long-term population growth

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Abstract

In this paper, we reaffirm Watkins and Menken's (1985) conclusion that there is 'little likelihood that famines will be a major determinant of population growth in the future, any more than ... in the past'. We find that age and sex-specific patterns of famine mortality change that have markedly different proportional change in group-specific mortality can nevertheless lead to similar trajectories for population size. Subsequent growth is related more to relative reductions in survival by age than to relative increases in mortality. We also comment on the use of Dyson's conception index as a measure of the onset of famine.

On the basis of careful analysis of three nineteenth-century famines in the Indian subcontinent, the 1943–1944 Bengal famine and the 1974–1975 Bangladesh famine, Dyson (1991a,b) challenges accepted thinking on the short-term effects of famine in South Asia and argues that many notions about the consequences of famine may be incorrect. His work illuminates our pictures of the evolution of the response of birth and death rates to famine conditions, of the interaction of famine conditions and epidemics, and of the age and sex composition of famine deaths. In this paper, we comment on his claim that conceptions provide a sensitive index of the development of famine and conclude that they do so only in retrospect. We next discuss the controversy over age and sex differentials in the impact of famine and attempt to recast the issue in terms of the impact first on survival rather than deaths, and then on resumption of population growth. We use a computer-simulation model to provide illustrations of the effects of different age and sex patterns of mortality change during famines on subsequent population growth.

Conception rates

Dyson (1991a:22) contends that the fertility of a population was 'affected at a far earlier state in the build-up to famine than was its mortality' and that the 'level of conceptions...constitutes a reasonably sensitive index of the development of famine'. We are puzzled by these statements for several reasons. First, in these populations, conceptions were not observable until the birth occurred nine months later, by which time death rates were, in most cases, increasing. For the 1943–44 famine in Bengal, the increases in his conception index appear to precede price increases (Dyson, 1991b, Figure

6). As he discusses, conditions other than food shortage may have led to the early change in the conception index.

An alternative explanation for the observed patterns of birth and death rates is that both the fertility and the health of these populations were affected by changes in the availability of food. In both cases, the effects were not observable until some time later, in that deaths do not occur immediately, but rather follow cumulative decline in health and may await the renewal of an infectious disease, as Dyson suggests. The difference is that poor health can be reversible, while a conception that failed to occur is always reflected in the births nine months later. Like death rates, birth rates may also reflect cumulative effects of food shortage since, according to Pebley et al. (1985), intrauterine mortality is affected by poor nutrition.

Therefore, we agree only to a limited extent with Dyson's claim that conceptions can provide a sensitive index of the evolution of famine. In the past, they were not observable while the famine was developing, so could not be used to predict famine. This situation is likely to hold today. In addition, without an increase in mortality, it is doubtful that a crisis would be classified as famine. Therefore, the usefulness seems to be circumscribed: when there is a famine, the change in health and fertility conditions can be traced back by shifting birth rates back nine months to serve as a proxy for live-birth conceptions.

We are far more persuaded by his description of the difference in concentration of the effects. Live-birth conceptions may be affected over an extended period of rising concern about food conditions, even before the mortality crisis has begun. The peak in deaths, however, is likely to be much more sharply delineated if most deaths occur when, after an extended period of deteriorating health, infection strikes. It may, as Dyson suggests, even coincide with improvement in food intake.

A final set of comments returns to Dyson's use of the birth rate as a proxy for the conception rate nine months earlier. By so doing, he implicitly assumes, first, that intra-uterine mortality is not affected by famine conditions, secondly, that registration includes all live-born infants, even those who die soon after birth; and thirdly, that if births are under-registered, the level of underregistration is constant through time, even during famine. While the assumptions regarding birth registration may hold for the data that Dyson uses, they are less plausible when applied to other historical populations, for example China's, so that conception indices may be of limited utility in different circumstances. Where many births were never registered, especially if the child died while still very young, reliable rates of birth and conception are difficult to estimate, especially on a monthly basis as required by Dyson's method. Fluctuations in the conception rate would be impossible to distinguish from the effects of variations in infant and child mortality in later months.

Even in the data that Dyson employs, conception rates during the famine may be underestimated by birth rates displaced by nine months. Pebley et al. (1985) show that intrauterine mortality in Matlab increased as the nutritional status of the mother deteriorated, especially when nutritional status was measured by weight gain in the previous month, but also when measured by pre-conception weight. What may appear to be fluctuation in the conception rate nine months previously could in part reflect variation in intrauterine mortality in the intervening months.

Age and sex pattern of famine mortality change

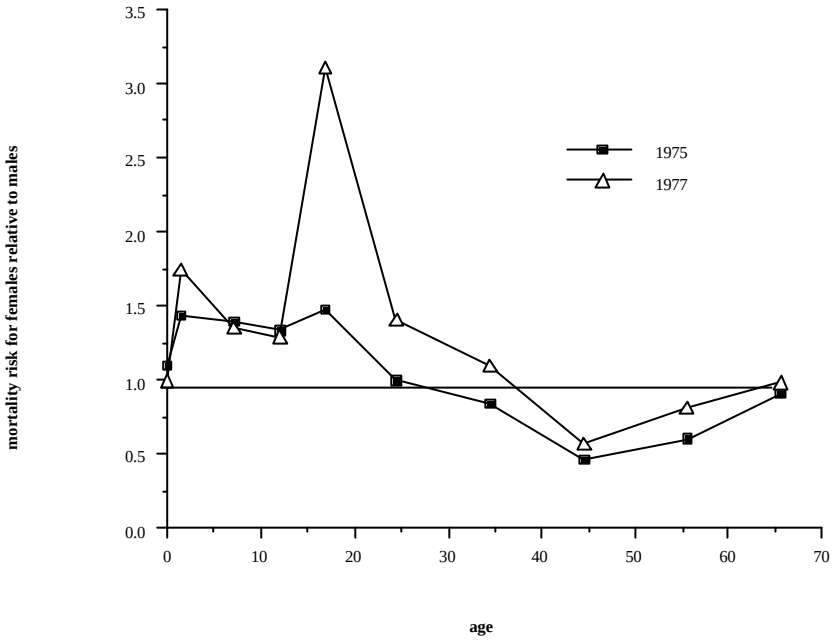
According to Dyson (1991b), use of flawed or incomplete data has been the major culprit leading to faulty conclusions about the mortality impact of famine. Analyses of the effects of the 1943–1944 Bengal famine by Sen (1981) and Greenough (1982) are both problematic because they relied on data for West Bengal alone, which at the time of the famine included only one-third of the population of Bengal. Dyson found that the data were questionable because they were adjusted before they were published, and the assumptions built into the adjustment procedure were later treated as findings. He

employs new data on this famine that cover the entire country and are available by month rather than by year. Dyson also presents evidence that Chen and Chowdhury's (1977) analysis of the 1974–1975 Bangladesh famine was flawed. Some age-specific death rates used in the analysis seem to have been incorrectly taken from years other than the ones listed in the tabulation. In addition, in order to calculate the longest possible time series, they relied exclusively on data from the 132 villages that were part of the Demographic Surveillance System from its inception. In many age groups, rates were based on only a few deaths, and may vary considerably from year to year. These data have been widely used to represent the age and sex pattern of the mortality impact of famine (see for example Watkins and Menken 1985, Caldwell and Caldwell 1989, Menken and Phillips 1990). In this new analysis, data from 228 villages are used, so that the number of individuals in the sample is almost doubled.

Dyson (1991b:289) somewhat surprisingly suggests that the change in mortality for males in Matlab has been 'obscured by a general concern with excess mortality of females'. Yet as early as 1978, Ruzicka and Chowdhury (1978:5) commented on the fact that male mortality for 1975 exceeded female mortality from age 25 on and Watkins and Menken (1985:656) discuss the mortality advantage of women during famines. Figure 1, which uses Dyson's (1991b, Table 9) data, shows the relative risk of dying for females compared to males in 1975 and 1977. The famine in 1975 sharply reduced the female disadvantage for teenagers and reduced the age at which women began to have lower mortality than men. Based on similar data, Watkins and Menken (1985:656) concluded that women are favoured in famine situations. Therefore, we find no disagreement between Dyson and a number of earlier analysts on this issue.Dyson's conclusion that relative mortality increases during famine were highest in the age range which normally enjoyed the lowest mortality, older childhood and young adulthood, contradicts the findings in Chen and Chowdhury (1977). Dyson shows that their claim that relative increases were highest in the age groups that already had high mortality, that is among children and the elderly, was based on faulty data.

In their review of information on the age pattern of mortality response to famine, Watkins and Menken (1985) did not find a universal pattern of mortality increase that characterized all societies. Variations in response to famine could reflect the effect of social and cultural norms governing the allocation of resources during crisis, as well as region-specific age patterns of cause of death. While Dyson's findings may be valid for South Asia, it seems quite possible that other age patterns could characterize the response of other historical populations.

Figure 1
Age-specific relative risk of dying for females compared to males: Matlab, 1975 and 1977



Even for South Asia, it may be too soon to reach some of these conclusions. Dyson's results about the most recent Bangladesh famine depend heavily on his decision to compare the average death rates of 1975 and 1976–1977. Razzaque et al. (1990) compared three cohorts of children born during the famine, conceived during the famine, and conceived after the famine in villages that were on the riverside, and therefore expected to be vulnerable to flooding. They found that children born during the famine experienced higher mortality through the second year of life, while those conceived during the famine had higher infant mortality. The 1976 rates in the youngest age groups are therefore influenced by the famine-related risks – whether they came from measles, as Dyson speculates, or are carryover effects from the famine experience. Dyson used the average rates for 1976 and 1977, so his results are surely influenced by this problem.

The inconsistency between Dyson's results and the results in Chen and Chowdhury (1977) in fact point to the need for recalculation of the Matlab time series of vital rates, using consistent geographical areas, time periods, and computer programs. The original rates were estimated when computer facilities were less than ideal; it is not surprising that errors are now being found in these data. Since all records are still available, it should be possible to correct the time series. This task is not a simple one, however, since there are at least three periods of varying geographic coverage.

In 1966, the Demographic Surveillance System was established and operated in the 132 villages used in the Chen and Chowdhury (1977) analysis. In 1968, 101 villages were added, a few of which were merged or dropped; Dyson uses figures that come from the remaining 228 villages. In 1978, data

collection ended in 79 villages, leaving 149 villages for which more recent information is available. Thus, one important need is for three, or possibly four, parallel series: (1) the 132 villages from 1966 either to the present or until 1978 if some of them were dropped; (2) the 228 villages from 1968 until the present or until 1978 if some of them were dropped; (3) the current 149 villages from 1968 until the present, and (4) that subset of the current 149 villages that has been continuously observed since 1966.

Even these series may not resolve the problem of estimating the age and sex differentials in the impact of the famine. It may be necessary to use time periods other than calendar years. Razzaque et al. (1990) claim that the famine ran from July 1974 to June 1975. For this reason, years that begin in May, such as Chen and Chowdhury used, may reflect the famine conditions better than the calendar year. There is, however, a cautionary note: D'Souza and Chen (1980) suggest that rates for adult males may be biased upward because able-bodied men 'migrated to cities in search of food and work' leaving the less healthy in Matlab.

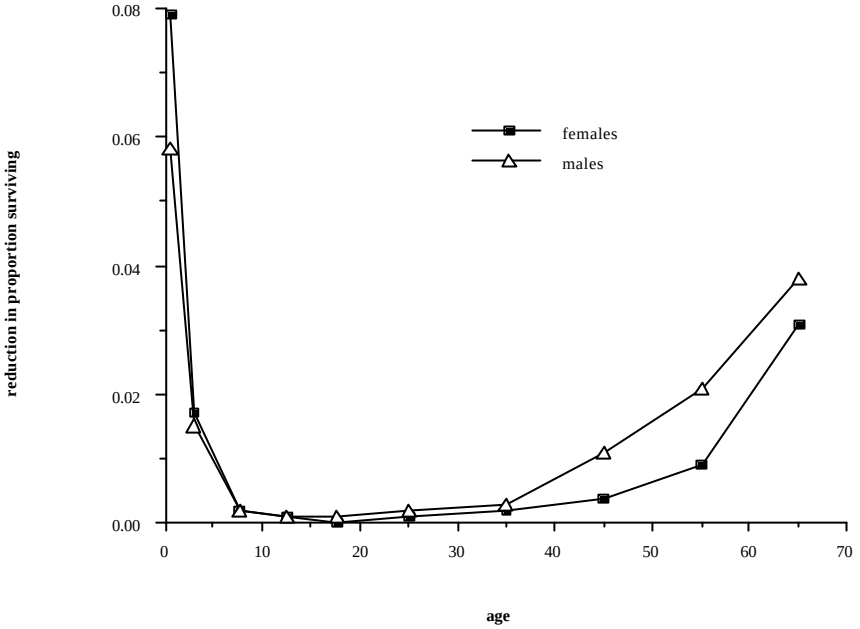
We strongly suggest that these recalculations be carried out, along with an examination of migration so that consistent time series are available on which to base firmer conclusions about age and sex differentials in famine impact.

Why are we interested in *proportional* increase in mortality?

We want to raise this question as a serious issue. Dyson, and many previous investigators including the senior author of this note, have treated the proportional increase in mortality as the significant indicator of the effect of famine: the greater the proportional increase in mortality at a specific age, the greater the effect of famine, regardless of the base or 'normal' mortality risk for that age. Dyson explicitly criticizes some analysts for using the absolute increase as their measure. If, however, we are interested in how a population recovers, in size or along other dimensions, from famine, we may well be concerned with the change in the survival rates, especially in those age groups that will contribute to future generations. Figure 2 gives the reduction in 1975 in the proportion surviving one year when the 1975 mortality rates are compared with the 1977 figures (taken from Dyson 1991b, Table 9). (The shape of this curve changes little when the average of the 1976 and 1977 rates is used as the non-famine baseline.) The reduction in survival is greatest for those age groups that were most at risk under normal circumstances.

Coale (1973), in a classic paper, considered the effects of mortality change on the age structure of a population. He found that age distribution was preserved if each age group experienced the same *absolute* change (positive or negative) in mortality rate, that is, if for each person, regardless of age, the added risk of dying during a famine was the same, so that change in risk was *age-neutral*. Under this regime, each age group contains fewer individuals in the following year than it would have without the mortality change, and the proportional reduction is exactly the same in every age group. That is, if every age group is, say, ten per cent smaller than it would have been, then so is the total number, so that age structure is, therefore, maintained.

Figure 2
Age-specific reduction in survival rates for males and females: Matlab, 1975 compared to 1977



In contrast, if every age group experiences the same *proportional* change in mortality, it is the age distribution of deaths that is preserved. The number of deaths at each age is multiplied by a constant, so that the proportion of the total deaths that occur at age *a* is unaltered. In this case, the population age distribution moves toward greater concentrations in those ages where mortality is usually low, since the age groups with the highest usual mortality rates have the greatest decrease in survival.

Finally, the effect of *disproportional* mortality change depends upon whether or not the highest increases occur in age groups which normally have high mortality. If so, the concentration of the population into low-mortality ages is even greater than in the case of proportional-mortality change. The reverse occurs (increased proportions left in high-mortality age groups) if there is a negative relationship between normal mortality level and proportional increase during the crisis.

From this discussion, we can consider the short-term effect of the different types of mortality change on the population age distribution, assuming that the usual J-shaped relationship between age and death rates holds. We consider three cases:

1. If *mortality increases at all ages by a constant proportion,* the survival probabilities of infants and the older population decline the most. The surviving population is then concentrated more in the reproductive ages.

2. If *mortality increases disproportionately at the older ages*, then the survivors have a younger mean age and are even more concentrated in the reproductive ages than under Case 1.

3. An *age-neutral mortality increase* does not change the age distribution of the population and the same proportion is in the reproductive ages after the famine as before.

If famine affects fertility, the effect is similar to the impact of an increase in infant mortality: the population is more concentrated in the reproductive ages.

The short-term effects on the birth rate after the crisis will differ according to which of these types of famine occurred. If we assume that age-specific birth rates return to their original levels, a population that experiences the age-neutral famine will immediately return to its previous crude birth rate, death rate, and growth rate. A population with proportional mortality change will have a higher crude birth rate, because more are in the reproductive ages. It will, in the short term, grow more rapidly than when famine was age-neutral. A population with disproportionate mortality change will have even higher birth rates right after the crisis and will regain its original size more quickly.

The long-term impact of famine mortality

The earlier work by Watkins and Menken (1985) examined the moderate to long-term effects under two assumptions about the age-specific impact of famine, namely, that there was no age selectivity, and that the age pattern of increase followed roughly that of the Chen and Chowdhury data. There was no difference in the effect of famine on males and females. Simulating famines of different duration (two years and five years) in populations in which the underlying rates of growth ranged from zero to one per cent, Watkins and Menken found that recovery time[1] was faster and the population was larger 90 years after the end of the famine, when the reproductive age group was less affected by famine than in an age-neutral famine.

The age patterns reported by Chen and Chowdhury (1977) and by Dyson (1991b) both affect survival of the non-reproductive age groups disproportionately, leading to population age distributions that favour fertility and, therefore, to shorter recovery times.

To examine the effects of the different assumptions, we rewrote the simulation model to incorporate sex differences in mortality change and simulated the three types of famine, with two versions of non-proportional change. Table 1 shows the assumptions about the relative shape of age-specific mortality change that were used; only case 2b incorporates sex-specific impact. These proportions were adjusted so that the death rate in the first year of the famine was either 110 per cent or 150 per cent of the stable level. In all cases, fertility dropped by one-third during the famine. Immediately post-famine, age-specific fertility and mortality rates returned to their original levels. The original population was based on a West-model life table with expectation of life for females of 27.5 (Coale and Demeny 1983).

1 The number of years from the start of the famine until the population regained its pre-famine size.

Table 1
Age pattern of mortality change due to famine

	Change in age-specific mortality				
	Proportional				Additive
Case	1	2a	2b		3
Age Group	Constant	Chen and Chowdhury	Dyson Male	Female	Age Neutral
0	c	1.35c	1.45c	1.64c	+k
1	c	1.27c	1.44c	1.40c	+k
5	c	1.40c	1.23c	1.28c	+k
10	c	1.03c	1.36c	1.43c	+k
15	c	1.18c	1.90c	1.04c	+k
20	c	1.18c	1.81c	1.36c	+k
30	c	1.18c	1.91c	1.60c	+k
40	c	1.18c	2.32c	1.79c	+k
45	c	1.43c	2.32c	1.79c	+k
50	c	1.43c	2.10c	1.63c	+k
60	c	1.43c	1.56c	1.44c	+k
65+	c	1.43c	1.56c	1.44c	+k

Table 2 gives the two measures of the impact of these famines: the recovery time and the population size 90 years after the famine ends. The pattern is clear, and whether we follow Chen and Chowdhury or Dyson makes little difference. Under both assumptions (cases 2a and 2b), the recovery time is slightly shorter and the population size 90 years later is slightly larger than when all mortality rates change by the same proportion (case 1). When famine is age-neutral (case 3), the population takes longer to regain its original size and is smaller 90 years later than under the other assumptions.

One *caveat* is in order. These results are based on the assumptions that women return to their original fertility rates at the end of the famine. If fertility is affected for a long time after the crisis because male mortality widowed women or made it more difficult for them to find a spouse, then the effects may differ from those shown here. An approach to examining the impact of famine in future studies might be through consideration of changes in reproductive value under these circumstances.

Table 2
Effect on recovery time and long-term population size of different age-specific patterns of famine-related mortality increase

Stable population growth rate		0.0%		0.25%		0.5%		1.0%	
% inc. in CDR	Duration of famine	Recovery time	Pop. size after 90 yrs	Recovery time	Pop. size after 90 yrs	Recovery time	Pop. size after 90 yrs	Recovery time	Pop. size after 90 yrs
No change in death rates									
	2 years		1000		1258		1582		2498
	5 years		1000		1268		1606		2574
Case 1. Proportional increase in each age group									
110%	2 years	***	912	35	1147	16	1442	9	2273
	5 years	***	802	89	1016	44	1285	19	2052
150%	2 years	***	891	46	1120	20	1407	11	2217
	5 years	***	759	95+	960	56	1214	27	1937
Case 2a. Chen and Chowdhury									
110%	2 years	***	917	33	1152	15	1448	9	2282
	5 years	***	812	84	1027	41	1298	18	2071
150%	2 years	***	896	43	1126	19	1414	11	2227
	5 years	***	770	95+	973	53	1230	25	1959
Case 2b. Dyson									
110%	2 years	***	917	32	1153	15	1449	9	2285
	5 years	***	813	83	1029	40	1302	18	2079
150%	2 years	***	897	42	1127	19	1416	11	2231
	5 years	***	772	95+	977	52	1235	25	1969
Case 3. Uniform increase in each age group									
110%	2 years	***	897	43	1128	21	1418	11	2235
	5 years	***	762	95+	965	55	1228	28	1948
150%	2 years	***	870	57	1094	28	1374	14	2164
	5 years	***	705	95+	892	71	1128	36	1798

Discussion

While we have disagreed with some of Dyson's conclusions, we want to comment on substantive aspects of his analysis that merit particular attention. First, it would appear from inspection of his time series (Dyson 1991a,b) that conceptions were more closely tied to price levels during the nineteenth-century famines than during the twentieth-century famines. In all three of the nineteenth-century famines, the conception index tracks the price index quite closely. In the twentieth-century famines, though, the conception index seemed to move more independently, responding only to dramatic shifts in the price level. That the response of conceptions to price differed over time suggests that there was a change in the way individuals decided whether or not to have children or in the reserves available in hard times.

Dyson's results also suggest the possibility that population responses to famine may have been region-specific. In the nineteenth-century South Asian famines, mortality did not rise in tandem with prices. Rather, the most severe mortality response was delayed until the arrival of the next monsoon season, at which point rates increased dramatically. The majority of these deaths could be attributed to diseases like malaria, not starvation.

From this perspective, famine could be considered a distal determinant of mortality, operating through a set of proximate determinants that are likely to have varied by climate and society. In the case of South Asia, these proximate determinants were diseases that already dominated mortality there, namely malaria and to a lesser extent diarrhoea.

In a colder region of the world, the proximate determinants might have been respiratory diseases. It is interesting to speculate on what the famine response would look like in a colder climate: perhaps a winter-time peak in deaths, the result of normally mild-respiratory infections that could become acute when poor nutrition weakened the body's immune response.

The possibility that societies vary in the way that they allocate resources during a crisis may also be a determinant of the mortality response to famine. If Dyson's assessment of the age-pattern of famine mortality is correct, it suggests the possibility that societies in South Asia were characterized by a willingness to devote resources to the young and the aged during crisis, at the expense of young adults. It is also possible that, for biological reasons, increases in the incidence of malaria affected young adults more than children or the elderly.

Dyson's investigation greatly adds to our knowledge of the detailed effects of famine. The results of our simulation are complementary, in the sense that they take the results forward in time and examine the moderate to long-term sequelae. As before, we find that the effects of the extreme famines we simulated are rather small. In addition, especially if one is interested in long-term effects, we suggest that the use of survival rates would be preferred over the use of death rates to assess famine impact. Although Dyson has suggested that the age-pattern of famine-mortality change differs from the one used by Watkins and Menken (1985), his new pattern still leads us to the same conclusion: 'Unless famine intensities exceed those upon which our calculations were based, there is little likelihood that famines will be a major determinant of population growth in the future, any more than they appear to have been in the past' (Watkins and Menken 1985:669).

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Inhibited conception and women's agency: a comment on one aspect of Dyson's 'On the demography of South Asian famines'

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Tim Dyson's review of the demographic character of five major nineteenth and twentieth-century South Asian famines (Dyson 1991a,b) deepens but also complicates our understanding of the societal crisis that results from widespread household-level food shortages. His rigorous comparison justifies itself by bringing into focus several until-now rather hazy famine processes; the most important of these, in my view, is the fall in conception that invariably precedes the onset of famine mortality and that continues well after famine mortality ceases. Although other scholars have commented in passing on this phenomenon, no one has so clearly shown the depth and duration of inhibited conception, which, in the examples reviewed, was more serious (because earlier and longer-lasting) than previously recognized.

In the course of demonstrating lowered fertility during historical South Asian famines, Dyson introduces a statistic he calls the 'conception index' or CI. The CI compares the conception rates in a famine-affected population to the non-famine conception rates in the same population at an earlier, more stable time. Whether or not Dyson's terminology is adopted – seeing that the index is intended to measure a decline in conception, it might be more apt to call it a *non*-conception index – his method, which projects post-famine birth registration data nine months backward against baseline fertility during famine, will surely be adopted by others who are engaged with famine demography and have access to registration data.

Some historians are committed to the term 'mortality crisis' as a synonym for famine, but Dyson's work implies that inhibited conception is a better diagnostic than crisis mortality because it is more consistently present. Note that the Bombay presidency famine of 1896–97 had only a modest mortality spike compared to other Victorian famines but a very substantial fall in conception (Dyson 1991a:17-

* **I am grateful to the editors of *Health Transition Review*** for the invitation to comment on Tim Dyson's two articles. I also acknowledge the facilities of the Center for International and Comparative Studies of the University of Iowa and the South Asia Institute of Heidelberg University, while preparing my comments.

18), while in more recent famines, for example, the Bihar famine of 1966–67 and the Maharashtra famine of 1970–73, there was presumably the fall-off in conception but virtually no famine mortality. Nowadays there is a marked tendency for famines to be relieved by outside agencies to the point that mortality from starvation and disease is effectively suppressed (the Bangladesh famine of 1974–75 was exceptional in this regard), which raises the question whether modern, relieved, low-mortality famines have much in common with historical, unrelieved, high-mortality famines. Dyson shows that the link between the two types is the signature effect of inhibited conception.[2]

In famines where mortality has occurred, it is not evident from Dyson's paper that the number of infants *not* conceived is regularly more or less than the number dying. That is, the magnitude of fertility losses during famines might profitably be compared with the magnitude of mortality losses. The calculation of hypothetical lives lost or saved is a familiar activity in public-health work. Concretely, the mortality from diseases and injuries for every age up to age 65 is sometimes converted into a statistic called the 'potential years of life lost' (PYLL). That is, every death prior to 65 years (this number is arbitrary) is considered premature, and the cost to society as measured in years of forgone activity is allocated to specific causes. (Causes of infant mortality loom large for obvious reasons in such calculations.) The magnitudes of all the PYLL attributable to each cause of death, for example, infections, injuries, congenital defects, environmental exposures, are then scaled and used to prioritize health interventions. This kind of logic could be applied also to conception losses during famines to demonstrate in fuller fashion, by going beyond simple mortality, the toll to society.

Dyson is somewhat vague about the socio-cultural routes to lowered conception associated with past South Asian famines. Or, phrased positively, he shows restraint in exploring the behaviours that lie behind the fertility effects he reveals. Changes in conception *pari passu* with price movements and with the deterioration of exchange entitlements are acknowledged, but how environmental factors like scarcity and unemployment come to affect biological processes like fertility is sketchy. We are told that

> through a variety of well-known mechanisms (e.g., reduction in coital frequency, deferment of marriages, decreases in fecundity, spousal separation through migration) these populations reduced their conception rates as a direct response to increasing levels of distress (the latter here roughly proxied by food price rises) (Dyson 1991a:22).

The idiom of 'mechanism' with its suggestion of autonomous process is misleading in this context; while some direct effects of hunger or reproductive physiology are likely, human decisions with all their contingencies and calculations are also prominently involved. In any case none of the 'mechanisms' Dyson mentions is really 'well-known'. (Who has investigated or would care to demonstrate 'coital frequency' during a famine?) Nor has any of these responses been proved to be as direct as or more direct than any other. I do not fault Dyson for these opacities which are not demographic in nature. It may be possible, however, that a more fine-grained analysis of vital registration records would allow demographers to glean information about the ages, districts of origin, castes, income and occupation, and family composition information that, taken together, would help to discriminate those women who conceived during famine from those who did not. Even so, demographic methods alone cannot recreate the contexts of choice in which fertility was inhibited.

Interestingly, Dyson portrays the fall in conception that occurs before the height of a famine as a *protective* state that enhances women's survival. Pregnancy and childbirth, always hazardous to a woman's health in rural South Asia, are particularly dangerous during a famine. Thus, if conception is

2 Like Dyson, I presume that famine is not a merely historical category and that continuing economic inequities and administrative incompetences will lead to subsistence crises in the future followed by global interventions to provide minimal relief.

suppressed, women who would otherwise be at risk of fatal complications will escape them. This escape is Dyson's principal explanation for the consistent pattern of lower adult female mortality increases than adult male mortality increases during famines. At one point he refers to the inhibition of conception as 'anticipatory', putting the term in quotation marks to express, I presume, an ambivalence about the degree of conscious agency. Yet, in his conclusion, he suggests that 'we might be wrong to rule out an element of conscious planning in explaining such anticipatory behaviour' (Dyson 1991b:293).[3] This is something to sit up and take notice of: the large-scale demographic effect which Dyson's research has highlighted appears to him plausibly to be the result of deliberate efforts taken by scarcity-affected persons, presumably women, to control fertility. In short, altered reproductive behaviour during famine may be something that women help make happen instead of having forced upon them. Are the 'mechanisms' of inhibited conception, then, substantially voluntarist rather than autonomic in nature? Is inhibited conception during famines even appropriately considered a *loss* (an involuntary forfeit), as assumed in prior paragraphs, or should it be considered instead a *boon* (a desired benefit)?

There is a version of South Asian famine history, to which I personally have contributed, that represents rural women as victims of authoritative, indeed, masterful adult males who appropriate decision making about all matters related to the survival of the lineage, the family and its individual members (Greenough 1982:215–225, 245–253). Specifically, anecdotal and numerical data in my study of the 1943–44 Bengal famine suggested that male heads of households regularly seized whatever resources were locally available to secure their own survival, or, alternatively, that they regularly abandoned their wives and children and sought work and relief alone at a distance. These behaviours occurred on such a large scale that they resulted in many thousands of abandoned women remaining homeless after the famine, women who had to be cared for by charity. A controversial aspect of my argument was the assertion that the adult male appropriation of resources and abandonment of dependants was culturally sanctioned. I found no contradiction in Bengali culture between adult male self-preservation and widespread moral values that favoured adult males over females. A newer analysis of the same data by Bina Agarwal, however, emphasizes a conflict-cooperation model of household-level decision making in which scarcity-affected Bengali women 'negotiated' with men according to their 'resource endowments' and 'fall-back positions'. Because women began their negotiations from a disadvantaged position *vis-ˆ-vis* fathers and husbands, the result was still massive impoverishment, abandonment and death, but at least women are represented as having actively participated in deciding their destinies rather than simply submitting to 'despotic' males (Agarwal 1990).

Agarwal (1990), following an analytic track opened up by Amartya Sen, assumes that Bengali men in 1943–44 succeeded in imposing their wills on women and in securing their survival in greater numbers because they held larger resource-bundles and more numerous 'fall-backs' at the beginning of the crisis; hence moral codes that prioritized adult males over females may have provided men with convenient ideological cover during a resource free-for-all but were not materially responsible for their survival. I am no more persuaded now than I was ten years ago that a whole gender category reasons so amorally, that is economistically, but I am attracted by Agarwal's argument that Bengali women were active during the crisis. Her position can be directly related to Dyson's study, because while Agarwal says there are women who 'bargain' for their subsistence during a famine, Dyson suggests there are women who 'plan' to inhibit their conception at the same time. Why should not these be the same

3 Citing the ethnographic evidence of Caldwell, Reddy and Caldwell 1988: 196–219; I have not yet been able to examine this work.

women? And why cannot the verbs be interchanged to hypothesize a South Asian woman who, in crisis situations, bargains with her husband over conception and makes plans for her own subsistence? Linked together, these suggestions give an impetus to a view of rural South Asian women as resolute, self-interested actors rather than passive victims. The problem with this formulation is that there are so few data. Neither Dyson's nor Agarwal's reinterpretation offers new observations on these issues.

It seems evident that we should now explore, both in historical studies and through field work, the hypothesis that scarcity-affected South Asian women take recourse on a large scale to behaviours calculated to save their lives. Existing accounts of women's subsistence-enhancing activities during famine – diet-shifting, lowering intake, foraging, selling off jewellery and utensils, taking up paddy-husking and other crafts, conversion, begging etc. (Agarwal 1990) – can be easily fitted within the hypothesis, but we need also to explore the less visible 'fallbacks' such as hoarding, taking secret loans, stealing, and prostitution, which the hypothesis of women's active agency implies. We also want to know in much more detail about the conception-inhibiting 'mechanisms' already mentioned by Dyson: do women in fact *initiate* the postponement of marriages in times of scarcity? Do married women *refuse* intercourse with their husbands and initiate their separation? What other planning goes into fertility inhibition? Do they, for example, procure abortions? While we need to learn much more about these matters, and while we want to know about the mental resolutions and moral visions of the women most affected, I suspect that such knowledge will only be available from informants who are actually trapped in real-world situations of grave scarcity. Grave ethical considerations will intervene, and these intrusive enquiries can be prosecuted only if they are accompanied by direct relief, and carefully explained to informants; and if the women's consent is secured and the interviews are consistent with their self-interest and dignity.

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A note on 'conscious planning'

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Tim Dyson (1991b:293) writes:

One recalls Caldwell, Reddy and Caldwell's conclusions regarding survival strategies in south India during the drought of 1980–83; the birth rate declined, yet mortality failed to increase. Their ethnography suggests that we might be wrong to rule out an element of conscious planning in explaining such anticipatory behaviour. [70] The Matlab data show that fertility declined among women irrespective of their age or parity; this may have applied in earlier famines (footnote 70 reads, 'See J.C. Caldwell, P.H. Reddy and P. Caldwell, *The Causes of Demographic Change*, Madison, Wisconsin, 1988, especially chapter 9).

Two issues should be taken up.

The first is that the 1980–83 famine was, at least in southern Karnataka (once Mysore), a mild famine by the standards of the great famines discussed by Dyson. People were distressed; many lost their incomes; nearly everyone had to eat less food while most turned to foodstuffs they normally would have regarded as inferior or inedible; and almost all forwent all expenses except the most urgent if only because of the fear that the famine might be sustained and might intensify. The mildness of the famine explained the absence of any impact on mortality. The belt-tightening that was undertaken included the cancelling of nearly all festivals and ceremonial occasions, including religious festivals, for these events incur both direct costs and indirect ones in the form of new clothing which is regarded as essential. Marriages were markedly affected, principally because of the cost of dowry, although wedding expenses also played a role (Caldwell, Reddy and Caldwell 1988:208–209). Around 20 per cent, even of marriages already planned, were deferred, and the level reached 30 per cent among richer peasants who were obliged in normal times to have lavish weddings. Most families simply gave up planning marriages. Our estimate was that the marriage rate fell to one-quarter of its pre-drought level. When those who had deferred planned marriages were cross-questioned about their reasons, everyone spoke of dowry and wedding expenses, and not a single family referred to the subsequent costs and problems associated with pregnancies, births and the raising of children. The important points are the following.

(1) The marriage rate fell.

(2) Subsequently, the birth rate fell, and this was entirely explained by the fall in the first-birth rate arising from the deferment of marriage.

(3) Family-planning acceptance continued to rise slowly, and greater acceptance played an insignificant role in short-term fertility decline.

(4) The mortality level remained steady.

This explains why birth rates may begin to fall before death rates. It does not explain the fact that fertility decline in Matlab was not parity-related. The important point is that the 'conscious planning' related entirely to marriage and not to fertility. This would seem to be likely to occur in any society with arranged marriage, related ceremonial weddings, and either dowry or bridewealth. We were repeatedly told in the 1973 Sahelian famines that marriages could not take place because the bridewealth could not be found.

The second issue is the response of the Indian Tamils on Sri Lanka's tea estates to the 1973–74 famine which affected them so severely that infant mortality rates rose from 70 to over 100 per thousand and fertility fell by one-fifth. During anthropological demographic work in this area in 1987 our team repeatedly asked those of reproductive age during that period, why they had not conceived. By far the most common reply was that they had not had sexual relations. When asked why this was so, they most commonly said that those dire times were not the occasion for such frivolity or that people were weak and sick and that couples just did not do such things at such times. Some referred to the subsequent problems that would be incurred by pregnancy, birth and the addition of an infant, but this was not the common response. The emphasis was far more on the the unlikelihood of sex than on the

problems of fertility even though this was a population experiencing hard times and where a woman's pregnancy might curtail her income from tea-picking. Once again, the 'conscious planning' was not primarily a matter of fertility planning, but this experience conforms well with the finding that the Matlab fertility decline was unrelated to age or parity. It should be added that this was a population among whom sexual abstinence in the marriage after childbirth or terminally is culturally acceptable. It might also be noted that there apparently was some rise in illegal abortion during this period.

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Famine Reactions

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I am grateful to the *Health Transition Review* for the chance to reply to the three valuable 'comments' which have been made about my recent paper on the demography of South Asian famines (Dyson 1991a,b). In each of the three comments there is much which extends the issues I addressed, and much with which I agree. However, here I will mainly address points of disagreement, some of which may have arisen because of a failure on my part to make myself sufficiently clear. Since Menken and Campbell make the most extensive and pointed remarks, I will follow them in discussing first, conceptions, second, mortality, and third, the long-term effects of famine.

Conceptions

I certainly stand by my statement that in the five major South Asian famines which I analysed (those of 1876-78, 1896-97, 1899-1900, the Bengal famine of 1943-44 and the Bangladesh famine of 1974-75) the fertility of the population was affected at a far earlier stage in the build-up to famine than was its mortality: in the sense that conceptions were reduced at a much earlier stage. Menken and Campbell say that they are puzzled by this because 'in these populations, conceptions were not observable until the birth occurred nine months later...'. They go on to state that the observation could not be used to predict famine (for example, because of the nine months lag from conception to birth, and because of the absence of birth registration data in many countries) and that therefore its utility seems to be circumscribed.

I entirely agree with Menken and Campbell that the predictive utility of the observation is circumscribed. In saying that reduced conceptions are often 'a reasonable sensitive index of the development of famine' I did not intend to imply any predictive utility, nor did I use the word 'prediction', for precisely the kinds of reason which Menken and Campbell give. My main point was simply to state something which seems to be strongly suggested from the data provided in the original paper, namely that in each of the five famines conceptions were much reduced before the rise in deaths. Since Menken and Campbell go on to state that 'Live-birth conceptions may be affected over an extended period of rising concern about food conditions, even before the mortality crisis has begun', they do not seem to be far from accepting this position.

Let me give a concrete example of how a decline in conceptions may usefully provide 'a reasonably sensitive index of the development of famine', but in a way which is specifically *not* predictive. Menken and Campbell themselves cite Razzaque et al.'s (1990:147) view that in the Matlab area of Bangladesh the 1974-75 famine ran from July of 1974. But the conception index which I calculated using the Matlab data suggests that the fall-off in conceptions dates from around January of 1974, and perhaps a little earlier (see Dyson 1991b:288). Therefore I would argue on this basis that the famine may have started well before July. Incidentally, this interpretation would accord well with the detailed time profile of the 1974-75 famine provided by Alamgir which starts with the period January-March of 1974 (Alamgir 1980:118-140).

Citing the work of Pebley et al. (1985) which showed that intrauterine mortality in Matlab increased as the nutritional status of the mother decreased, Menken and Campbell point out that what may appear to be changes in the conception rate nine months previously could partly reflect variation in intrauterine mortality. I accept that there may well be some validity to this. But one should also note

that Menken and Campbell themselves only propose this as a partial explanation. Moreover, Pebley et al.'s results were strongest for foetal mortality during the last trimester of pregnancy. But variation in such late intrauterine mortality would not account for the fact that in the nineteenth-century famines, my conception index tracks price movements so closely (though it could point to the relevance of early foetal wastage). In any event, there is certainly no shortage of plausible alternative explanations relating to changes in coital frequency to explain the time trends which are observed. Finally in this context, it is interesting that Chen and Chowdhury have specifically stated that 'Fetal wastage rates in Matlab *thana* ... were not affected by the crises [of 1971 and 1974]' (Chen and Chowdhury 1977:422).

In discussing the issue of conception rates, Menken and Campbell state that 'without an increase in mortality, it is doubtful that a crisis would be classified as famine'. However in India, historically severe food crises, which were certainly officially classified as famines, did occasionally occur without net increases in mortality. In my paper I cited the case of the famine in Bombay Presidency in 1905-06; another example is the famine in Punjab in 1896-97. In both cases ecological conditions associated with drought triggered a state of famine, while at the same time probably leading to a reduction in some infectious diseases, especially malaria, and in Bombay in 1905-06, plague: hot, dry conditions perhaps tending to kill the rat flea *X. Cheopis* (Gottfried 1983:9). The fact that there was no *net* increase in mortality, although there were most of the other signs of calamitous famine including, probably, some deaths related to problems in geting food, hardly seems sufficient reason to me to withdraw use of the epithet 'famine'.

In a very different context here, Caldwell and colleagues describe as a 'mild famine' the 1980-83 drought in Karnataka during which the mortality level remained steady while the birth rate fell. In some cases, perhaps that of Karnataka, the appropriateness of the word 'famine' may be largely a matter of semantics. But I think it would be a serious mistake to assume that a famine must always involve an increase in the population death rate. In this sense I indeed subscribe to Paul Greenough's summary statement here that 'inhibited conception is a better diagnostic [of famine] than crisis mortality because it is more consistently present'. So although a reduction in conceptions may be a poor 'predictor' of a population's experience of famine, *ex post facto* it may constitute a powerful piece of evidence that a famine has actually occurred. Unfortunately, in a world in which national governments are all too ready to deny the existence of conditions of famine, such evidence may be of more than passing interest.

This brings us explicitly to the issue of why the level of conceptions declines in a population that is subject to famine. To Paul Greenough I should explain that I used the term 'anticipatory' mostly to indicate the simple fact that the conception decline occurred before the rise in the death rate. (Incidentally, this is a very different sequence of events from those associated with epidemics, in which the conception decline is usually reactive with the rise in deaths.) The term 'anticipatory' was placed in quotation marks to express my considerable doubt as to whether there was any significant degree of conscious agency involved in accounting for such behaviour. I believe that the major causes of these reductions in conceptions were precisely those I mentioned: spousal separation through migration, general declines in coital frequency, deferment of marriages and decreases in fecundity. However, at the end of the paper, and citing the work of Caldwell, Reddy and Caldwell (1988), I very cautiously suggested that there might be an 'element' (by which I meant a small component) of 'conscious planning' behind such reductions in conceptions.

In this context I am very grateful for Caldwell et al.'s comment here explaining that in the case of the 1980-83 crisis in Karnataka the 'conscious planning' element was simply that marriages were deferred, and as a result the first birth rate fell. But I also note from them that in the case of the 1973-74 food crisis among tea estate Tamils in Sri Lanka, a few respondents did mention the problems of pregnancy, birth and the presence of an additional infant, when explaining why they had not had sexual

relations during the famine. Note also that there may have been some increase in illegal abortions. For my purposes these observations by Caldwell and his colleagues are sufficient to suggest that there may be a residual element of conscious planning apropos fertility during famines. Indeed, Caldwell's observations relate precisely to the area of research which Greenough identifies as important.

Famine mortality

Menken and Campbell are surprised when I say that the fact that male mortality deteriorated most during the 1974-75 famine in Matlab has been obscured by a general concern with excess female mortality in the Matlab data. Clearly, we now all agree that male mortality probably did deteriorate most in 1975. And, of course, a number of other analysts, including Watkins and Menken (1985:656), have indeed concluded that generally male mortality tends to increase more than female mortality during times of famine.

However, I stand squarely by my statement that a general concern with the undoubtedly important subject of excess female mortality in Matlab in normal times has tended to conceal the basic fact that overall (that is, at all ages combined) male mortality deteriorated most in the 1974-75 famine. Nowhere in the extensive literature on the demography of this famine have I found a single statement to the effect that overall it was the mortality of males which deteriorated most in 1975. Even Ruzicka and Chowdhury (1978) – who were the source of the life expectation estimates which I used to illustrate this fact – restrict their comments to excess male mortality beyond age 25. And they go on to suggest that chance variation due to small numbers may be at least partly responsible for the high ratio of male to female death rates at these later ages (Ruzicka and Chowdhury 1978:5). Likewise D'Souza and Chen also present Matlab data which show that male mortality in the age groups 15-44 and 45-64 was much higher than that for females. However the main explanation D'Souza and Chen propose for this (and they propose it twice) is that in 1975

> a higher proportion of males in the adult age groups migrated to the cities in search of food and work; as a result, the population left behind may have been sex-selectively biased in favour of less healthy males staying home and thus at greater death risk (D'Souza and Chen 1980:266).

This suggestion that a 'selection effect' may have been involved is worth comment. Unfortunately, we do not have detailed information on the socioeconomic composition of rural outmigrants from Matlab during this famine. Presumably, people who were too ill to move had no option other than to stay where they were, and some of them must have died in Matlab. On the other hand, members of relatively better-off households, who had or could afford to buy food, may have had no reason to leave the area. It may well have been that the most impoverished and weaker sections of the society were overrepresented in the migration flows out of Matlab. And migration itself could have further elevated their chances of death. In short, it seems quite plausible that out-migrant males were more likely to die than those who remained. However, in the absence of data this is only speculation.

To sum up on Menken and Campbell's 'surprise'. The literature analysing the Matlab data for the famine years of 1974-75 has stressed, again and again, that in the childhood ages the increased mortality was disproportionately experienced by females (see, for example, Chen and Chowdhury 1977:415, D'Souza and Chen 1980:264, Razzaque et al. 1990:153). In contrast, the evidence of excess male mortality at adult ages has received much less attention. And when it has received attention, it has been in ways which have tended to cast doubt on its authenticity. So far as I am aware, no one has looked at overall mortality during the Bangladesh (Matlab) famine and has plainly stated that, in relative terms, males were probably hardest hit. Indeed the impression which has emerged has tended to be precisely

the reverse. For example, to quote Alamgir (1980:145), himself quoting Chen and Chowdhury (1977:415):

> During baseline years, female mortality consistently exceeded male mortality in all age groups except infant deaths. The age-specific differentials were more pronounced in children 1-4 and 5-9 years and in the childbearing years. Disaster tended to accentuate even further these sex differentials, particularly among children.

I agree with Menken and Campbell that the age-pattern of mortality increase which I identified in the three nineteenth-century famines, and in the Bengal famine of 1943-44, is unlikely to apply in all famines, even within South Asia. Perhaps the most important single distinguishing feature of these four famines was that they were all immense disasters: 'great famines' in Caldwell et al.'s phrase. My results apropos the age-pattern of mortality increase in the Bangladesh famine are certainly influenced by the use (following D'Souza and Chen 1980) of average death rates for 1976-77 to represent 'normal' conditions. With the available data there was little else which could be done. However I think the exercise was worthwhile in that it provided some hints (no more) of similarities with the age pattern of increase found for the earlier major famines. In using the Matlab data in this context, I was at pains to stress its several major limitations and the unsatisfactory nature of using the 1976 figures as baseline death rates.

Menken and Campbell provide a concise summary of the considerable task that would be involved in correcting and interpreting the Matlab time-series of vital rates for the period around the famine. They strongly urge that a consistent time series be assembled so that age and sex differentials in famine impact can be examined. To me an even more important issue which such an exercise might help to address is the distribution of famine mortality through time in 1974-75. This is because the Matlab data are unique among the famines which I studied in showing a long 'tail' of elevated mortality, lasting throughout 1975. In other words, the distribution of famine deaths does not seem to have been merely an accentuation of the normal seasonal mortality pattern, which in Matlab peaks around November-December.

If this 'tail' feature is real then it would indeed represent a significant departure from the experience of the earlier major famines. However it seems possible that the 1975 'tail' may be at least partly spurious. This could have happened if some of the deaths which occurred to outmigrants in late 1974 were only recorded (and misdated) by the Demographic Surveillance System (DSS) in 1975 as people gradually returned home to Matlab. Thus a real 'peak' in mortality could have been obscured and dissipated to form a 'tail'. In informal discussions about this possibility with people who were intimately involved with the DSS at the time, I have met with responses which range from complete denial that this could have happened, to acceptance that it could well have occurred.

Menken and Campbell raise the important issue of absolute and proportional mortality increases during famine. They note that I mostly address proportional increases and state that I explicitly criticize some (unnamed) analysts for using absolute measures. In response I make three points. First, my reason for working mainly with proportional increases was because that is what previous analysts have used. Some previous researchers, including Watkins and Menken (1985), seem to have concluded that the greatest proportional increases in death rates happened to young children and the elderly. But I found this a little suspect a *priori* precisely because the absolute base or 'normal' mortality risks at these ages are already high. Secondly, no particular criticism was intended of analysts who have used absolute increases as their measure. I can only assume that Menken and Campbell believe that I was criticizing Greenough's (1982) use of absolute increases in death rates in his analysis of the 1943-44 Bengal famine. But no criticism of him was intended. I was merely pointing out that there is nothing surprising in Greenough's finding that the largest absolute increases in mortality happened to young

children and the elderly. Thirdly, as in many areas of demographic research, we clearly need to consider both absolute and proportional changes in tandem when considering the mortality impact of famine.

Let me give another illustration of this third point. In the major famines which I analysed the proportional mortality increases experienced by males tended to be greater than those experienced by females, especially in the prime adult reproductive years. But one cannot interpret this without cognizance of the fact that the normal absolute levels of female mortality in the reproductive age groups were often markedly higher than those of males. To be sure, other factors such as 'anticipatory' fertility decline (which in this context 'benefits' women), greater female body-fat reserves, and the probable greater propensity of adult men to migrate, may all be relevant to explaining why proportional increase in male mortality exceeded those of females at these ages. But so too is the absolute level of the base from which the proportional increases are being measured. And in South Asia female mortality tends to be particularly high in the reproductive years. In this context Greenough is incorrect to say here that anticipatory decline is my 'principal explanation' for the lower proportional mortality increases experienced by adult females. All of the factors mentioned above are probably important.

Incidentally, similar considerations apply to attempts to deduce inferences about a society's 'preferences' in the allocation of food and other resources during famines from interpretation of the age-pattern of mortality increase. I am sceptical of such attempts, partly because many factors combine to influence the age-pattern of famine mortality, and partly because it is unclear whether the pattern of proportional or absolute increase by age, which can be quite different, should be the subject study.

Finally, on the subject of the mortality impact of famine, Menken and Campbell are right to say that previous analyses of the 1943-44 Bengal famine have arrived at faulty conclusions. In particular, Dyson and Maharatna (1991a) have recently shown that figures of 3 or 4 million excess deaths are significant exaggerations; a figure of about 2.1 million is much more likely. On the other hand, Greenough is incorrect in his assumption here that there was 'virtually no famine mortality' in Maharashtra in 1970-73. Even in what was a relatively well-managed disaster there were probably around 130,000 excess deaths (Dyson and Maharatna 1991b).

The long-term impact of famine

My main interest in famine demography was in the short-term effects. It does not surprise me that Menken and Campbell's new simulations, which incorporate the different age-patterns of mortality increase, make no real difference to the principal conclusion previously propounded by Watkins and Menken (1985:669) namely, that 'Unless famine intensities exceed those upon which our calculations were based, there is little likelihood that famines will be a major determinant of population growth in the future, any more than they appear to have been in the past'. While I agree with this statement, I think it is important to stress two things which it does not mean.

First, it does not mean that famines were never major determinants of population growth in the past. In their original paper Watkins and Menken mentioned Lardinois's (1985) work on the Madras famine of the 1870s and McAlpin's (1983) and Davis's (1951) fine studies relating to India 'when there was virtually no growth between 1891 and 1901, and several major famines occurred' (Watkins and Menken 1985:665). In fact if one looks at India over the fifty-year period from 1871 to 1921, when the average annual rate of population growth was about 0.36 per cent, one can make out quite a strong case that famines were a major determinant of population growth; although this is partly dependent upon what is held to constitute 'major'. The famine of 1876-78 devastated vast regions well beyond Madras Presidency; the north of the subcontinent was badly afflicted by famine in 1877-79; there were then the huge successive disasters of 1896-97 and 1899-1900. Summing the estimates of excess mortality assembled by Seavoy (1986:242) for these famines produces a figure of over 16 million excess deaths

in a population that was roughly 280 million, although the true toll may well have been still greater. Then the widespread famine of 1907-08 also killed millions of people. And the existence of famine conditions in central and northwestern regions of India in 1918 may well have contributed to the gigantic mortality toll associated with the influenza epidemic (Mills 1989:251). Of course, in addition, there were many minor famines during 1871-1921. And one would also need to take account of the fertility loss associated with these various crises. In short, the emerging picture of India's historical demography is one which, *inter alia*, does lend some support to famines acting as a major determinant of population growth. It also provides empirical examples of how special factors could come into play in the immediate post-famine periods which helped to hasten population recovery (see the various studies in Dyson 1989).

Secondly, and unfortunately, acceptance of Watkins and Menken's conclusion does not mean that major famines with large-scale excess mortality are a thing of the past. With, for example, increasing problems in global per capita food levels, especially since the mid-1980s (Brown 1991, FAO 1991), with signs of mounting environmental stress in many parts of the world, and most important of all, with widespread social strife and warfare in places such as the former Soviet Union, and much of Africa, there are few reasons for confidence that Caldwell's 'great famines' can be firmly assigned to the past.

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Book Reviews

The Mediterranean Diets in Health and Disease Edited by Gene A. Spiller. New York: Van Nostrand Reinhold, 1991. xvii+303pp. Hardback A$149.95.

For adults, and adult males in particular, culture can do much more to protect survival chances than can the elaborations of modern medical technologies. The two most notable 'safety zones' in the word today are the Mediterranean basin and East Asia. It is suspected that the protection against chronic disease afforded by dietary traditions is the main reason why male death rates in middle age are lower in Crete and Beijing than in the advanced centres of Western civilization. (Female longevity tends to correlate more closely with *per caput* income and to be less influenced by cultural zone.) This is the background to the burgeoning scientific and popular interest in 'Mediterranean diets' and the rise in olive oil consumption outside its traditional production area.

The book under review is therefore timely. Its 15 chapters are grouped into three sections: overview and history; typical Mediterranean foods and their physiology; and clinical aspects and epidemiology. Of the 29 contributors all 23 with institutional affiliations in the Mediterranean basin are from Italy. Of the other six, five are from north America and one from the UK. It is not surprising therefore that the text is biased towards Italy. Partly, this reflects the distribution of scientific activity, but this cannot explain the exclusion of authors from Greece or 'Jugoslavia', where there has also been considerable work on diet and chronic disease.

The first substantive problem and the necessary foundation of much of the subsequent discussion is the accurate characterization of 'the Mediterranean diets'. This foundation is not well constructed and remains wobbly throughout much of the text.

It is not easy to measure what people are actually eating and data of this kind are in short supply everywhere. In the first chapter Giacco and Riccardi make use of the Food and Agriculture Organization Food Balance Sheets for 1979 to 1981 to describe apparent dietary patterns in 14 countries bordering on the Mediterranean. As they note, this is the only standard data source available for comparing a wide range of countries. In terms of foods, the diets of the Mediterranean countries are characterized by high intakes of cereals and olive oil and low intakes of animal fat and meat. The two most apparent differences in macro-nutrient composition in comparison to the USA, are the lower proportion of saturated fats and the higher proportion of carbohydrates in the Mediterranean countries. There is uncertainty (using this data source) whether the Mediterranean diets really do exhibit higher fruit and vegetable (and fibre) intakes than northern diets, as is widely assumed.

Other available data sources are a Euratom diet survey of west European Countries in the 1960s which included samples in north and south Italy and studies of the Italian, Yugoslav and Greek cohorts in the Coronary Heart Disease in Seven Countries study. Relatively little use is made of the latter. Overall, the book would have benefited from a more rigorous and critical approach to the assessment of dietary intakes.

Against this somewhat uncertain background, the assembled chapters provide many interesting details and a few gems. I particularly enjoyed Thomas Braun's beautifully written and densely packed

chapter on ancient Mediterranean food: I doubt if there is a better brief survey of this topic anywhere. ('The Mediterranean', he writes, 'remained butterless, just as India has always been cheeseless'). The reader moves directly to a longer chapter on cereals, which, apart from some introductory paragraphs, is not explicitly related to the theme of the book: some editorial activity might have helped here. There follow, in the remainder of the second section, chapters on legumes, vegetables and fruits, edible fats and oils, dairy products, on slowly digested carbohydrates and mono-unsaturated fats. In the third section there are chapters on lipids, hypertension, cardiovascular diseases, obesity, diabetes and cancer.

The chapters of the second section contain much of interest: how oil has been extracted from olives, the different qualities of olive oil and its minor constituents, how cheese and yoghurt have been made; all this practical information is blended nicely with a consideration of nutrition. But why nothing on the third part of the classical Mediterranean triad of wheat, olives and grapes? Alcohol is a substantial component of the diet in many Mediterranean populations, supplying over 20 per cent of dietary energy in some. There is little discussion of this in the book and alcohol is often excluded from analyses of dietary composition. The high intake of fish, for example in Spain, is also largely overlooked.

The chapters of the third section consider connections between diet and various health outcomes. One is left with an impression of growing complexity: the connections between dietary fats and cardiovascular diseases now seems more complex than once thought; many dietary factors, not just added salt, influence blood pressure; obesity is not just a consequence of 'overeating'; the cancers of affluence (colon, breast etc.) now seem less related to macro-nutrient composition (high proportions of fat) and more related to micro-nutrients, and perhaps even to non-nutrients, in the diet.

This brings us to the dual attractions of the Mediterranean diets: first, in their consistency with low adult mortality they are a rebuke to the earlier 'northern' idea that the replacement of saturated fats with polyunsaturates was the one true path to reducing mortality risks in middle age (all the more so as the trials to 'prove' this hypothesis have yielded unimpressive effects on all-cause mortality); secondly, in their presentation of a 'non-northern' dietary pattern associated with low mortality they provide multiple stimuli to new hypotheses about diet and chronic disease: the apparent protection against cancer associated with green leafy vegetables is a case in point.

But do the disease-specific details really matter? One cannot eat one diet to prevent heart attack, another to prevent colon cancer and so on. As a guide to dietary choice, what is important is the overall mortality risk and to minimize this, the 'mediterraneanization' of one's diet seems a good bet.

If you have a scientific interest in the matter you will find at least some parts of this book rewarding. If you are after a quick read and recipes, go to one of the more popular accounts, for example: C. and M. McConnell, *The Mediterranean Diet: Wine, Pasta, Olive Oil and a Long Healthy Life,* Norton and Company, New York, 1987; or the earlier work by A. and M. Keys, *How to Eat Well and Stay Well the Mediterranean Way,* Doubleday, New York, 1975 (Ancel Keys was the coordinator of the famous 'Seven Countries' study.)

I reviewed this book during a Christmas spent in Spain. For Christmas dinner our first two courses were fish: their long-chain polyunsaturates might just have managed to flush away the cardiotoxic beef fat in the Christmas pudding which I had brought from England!

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History of AIDS: Emergence and Origin of a Modern Pandemic. By Mirko D. Grmek, translated by Russell C. Maulitz and Jacalyn Duffin. Princeton: Princeton University Press, 1990. xii+279 pp. Hardback US$29.95.

This is an eminently readable history, which has been assembled with considerable scholarship from the overwhelming mass of AIDS reportage and scientific publications. It is no mere chronicle of events but presents powerful argument that only now, in the late twentieth century, could the phenomenon of AIDS have arisen.

Mirko Grmek was a physician and Professor of Medical History at the University of Zagreb. He is now French naturalized and Director of Studies at the Ecole Pratique des Hautes Etudes at the Sorbonne. Despite his French connections, Grmek's account of the priority claims between Montagnier and Gallow over the discovery of the AIDS virus does not, to me, reveal bias. That part, like the rest of the book, is finely referenced and dated to allow the general reader, as well as the scientist, to understand how and why the controversy arose. The forces seen to be at work driving medically important (and hence lucrative) discoveries are revealed to be somewhat baser than naively one might wish to believe.

The history of AIDS is divided into four parts, One: A Calamity of Our Times, Two: The Oracles of Science, Three: A Look Back, and Four: Disaster: Its Extent and Causes. The references used in the text are presented concisely by chapter, number and author) and then in full in the 47-page Bibliography. Each of the four parts can be read independently and an index is provided for those wishing to use this history for reference work.

I yearn for much lengthier discussion in Grmek's Chapter 14, 'The Biological and Social Conditions of the Pandemic'. Here he assembles answers to David Durak's question posed in the December 1981 editorial of the *New England Journal of Medicine*: 'Why now, and why not before?'. Grmek introduces his concept of Pathocenosis, where he argues that 'the frequency and overall distribution of each disease, above and beyond various endogenous and ecological factors, depends on the frequency and distribution of all other diseases in the same population'. Following this he cites the immunological and technological advances in blood banking and the extraction and concentration of trace components from huge donor pools, which combined with the increased demand created by medicine and haemophilia, road trauma and increased surgery to allow an enormous augmentation of a previously negligible pathway for infectious disease transmission. The next technological advance combined with real-life forces, which he chronicles as facilitating AIDS transmission, is the disposable hypodermic syringe and needle, which is reused without sterilization owing to supply problems, money problems or cultural forces. The last section of this thought-provoking chapter briefly assembles the facts known about the 1970s 'coming out' of the American homosexual population. Between 1969 and 1978 nearly 30,000 homosexuals arrived in San Francisco. By 1982 it was estimated there were almost 98,000 homosexuals living in San Francisco, almost half living in a particular part of the city centre. Importantly for the AIDS virus, the new Western male homosexuals combined both the active and passive roles, unlike the historic and Eastern models of male homosexuality. Promiscuity and the search for physical pleasures were trumpeted as 'fundamental expressions of individual rights'. Not unexpectedly, a dramatic rise in classic sexually transmitted and other infectious diseases in male homosexuals preceded the arrival of AIDS. 'It was thus in the crowded ranks of the American homosexual community that the AIDS virus finally passed the point of no return in its epidemic spread'.

History of AIDS is highly recommended reading to all. Jargon is not used, but familiarity with medical-scientific terminology would definitely be an advantage to the reader. For anyone wishing to

understand what has happened and what may be ahead, Mirko Grmek has successfully grappled with the intellectual and geographic frontiers transgressed by AIDS, its science, its medicine and its politics.

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Population and Nutrition: An Essay on European Demographic History. By Massimo Livi-Bacci, translated by Tania Croft-Murray and Carl Ipsen. Cambridge: Cambridge University Press, 1991. xiv–149 pp. Hardback A$85.00.

The Struggle over *Kermis*

At fairs, which they called *kermis*, the people of Flanders indulged themselves extravagantly, eating and drinking to excess. In English translations of titles initially published in Italian, two scholars have reproduced depictions of Flemish fairs on the jackets of books discussing nutrition in the preindustrial era. Piero Camporesi[1] used Brueghel's *Le Pays de Cocagne* to illustrate an argument that Europeans had so little to eat, and were so obsessed with food, that they engaged in autophagy. Massimo Livi-Bacci prefers a more straightforward interpretation of this genre of art, using Jacques Savery's *Kermis* to illustrate an argument that Europeans ate well most of the time, at least in terms of caloric bulk.

Although Camporesi makes an overstated version of the case that early modern Europeans ate poorly to the point of malnourishment and worse, a version that few will credit, it is nevertheless true that many scholars continue to repeat and credit the argument that poor nutrition was the dominant cause of high mortality in the early modern era and that better nutrition played the dominant part in improving survivorship across the period 1750–1900. This argument is perhaps best known in the case made by Thomas McKeown, who studied causes of death in England and Wales in the decades after 1840 with the aim of linking specific groups of diseases to specific means of disease transmission. By narrowing the entrance through which causes of death might be assigned, McKeown opened a residual category, made up of diseases that he associated with poor nutrition rather than with specific means of transmission. Because the causes of death that McKeown associated with poor nutrition, such as tuberculosis, diminished in importance across the nineteenth century, he inferred that improvements in nutrition made the crucial difference in mortality decline in England and Wales after 1840, and more generally in Europe across the period 1750–1900. Although he tested the case for other putative causes of mortality decline, such as medical improvements and public-health reforms, McKeown did not bring the same careful scrutiny to bear on the case for nutrition.

In *Population and Nutrition* Livi-Bacci scrutinizes the claim that high mortality in the preindustrial era should be attributed to poor nutrition, concluding that the effect of poor nutrition has been exaggerated and also that mortality declined for reasons other than improvements in nutrition. 'In the long run ... there appears to be no real relation between nutrition and demographic development' (p. xiii). Livi-Bacci's examination of nutrition as a factor in demographic development considers both biological and historical evidence. From the biological literature he stresses not evidence about the synergy between malnutrition and certain infectious diseases detected among infants and weanling children in some developing countries in the mid-twentieth century, but evidence about the effects of temporary food shortages on people who are ordinarily well fed. Proponents of the case that

1 Piero Camporesi, *Bread of Dreams: Food and Fantasy in Early Modern Europe*, translated by David Gentilcore, Chicago: University of Chicago Press, 1989.

malnutrition mattered a great deal like to draw an analogy between preindustrial Europe and less developed regions in the modern world, an analogy that favours using evidence about health conditions in poor countries today as a way of making arguments about health in early modern Europe. Livi-Bacci, in contrast, prefers to draw an analogy between twentieth-century Europe, which has occasionally experienced dire food shortages, such as in the Venetian territories in 1917–1918 and in the Netherlands in the winter of 1944–45, and preindustrial Europe. Which analogy should be preferred?

The view that Europeans were poorly nourished has rested chiefly on the belief that too few calories were produced. Many scholars have collected evidence about the quantities of foodstuffs available for consumption in certain places and periods, and some of them have also translated quantities of food into estimates of quantities of calories. Livi-Bacci assembles a large body of evidence of this type, all of which tends to understate the supply of foodstuffs by reporting items subject to taxation or to exchange in markets but omitting items of auto-consumption. Even leaving aside that understatement, Livi-Bacci shows that almost all of the evidence about food supply shows that it was, in ordinary times, more than sufficient to provide the calories that the population required in order to lead active lives. Harvest failures occurred, sometimes coinciding with peaks in mortality and sometimes not. Although Livi-Bacci acknowledges that poor nutrition played a role in health, he argues that even in crises the death rate more often rose because changes in social behaviour assisted disease transmission than because food shortage lowered resistance to disease. In this way he provides evidence suggesting which biological analogy should be preferred: in ordinary times Europeans were at least adequately fed.

Studying price histories, Livi-Bacci notices not only that high food prices, which are suggestive of shortage, did not regularly coincide with peaks in mortality, but also and more tellingly that low prices, which are suggestive of plenty, did not have any positive effect on survival. Examining socioeconomic differences in mortality Livi-Bacci notices that elites did not gain an advantage in survivorship until the eighteenth century, though they must always have eaten more than the poor. In terms at least of caloric bulk, nutrition probably deteriorated toward the end of the eighteenth and in the early nineteenth century, but life expectancy improved.

Most specialists in the history of survivorship in Europe will probably find that they had reached many of the same conclusions as Livi-Bacci for many of the same reasons. Nevertheless, this book is an important contribution in two ways. First, for specialists it assembles familiar evidence into a forceful case that exalts the importance of epidemiologic factors and diminishes the importance of nutritional factors in explaining the mortality decline. Second, for the much larger body of demographers who do not specialize in the history of survivorship, this essay shows why it is no longer counted persuasive to explain the health transition in Europe as the consequence of better nutrition.

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From the Fat of Our Souls: Social Change, Political Process and Medical Pluralism in Bolivia. By Libbet Crandon-Malamud. Berkeley: University of California Press, 1991. xi–267 pp. Hardback US$45.00.

In the preface to her book, Libbet Crandon-Malamud writes that ‘It is an argument for medical pluralism, not for medical, but for political reasons. Deriving from research that asked why people

choose different or multiple medical resources, its thesis is that they do so for nonmedical reasons. It builds upon an idea...that people use medicine as a primary resource through which they get access to secondary resources, particularly where multiple resources exist and choice between them becomes socially and politically significant' (p. ix).

She argues that the residents of the town of Kachitu (not its real name) in the Bolivian Altiplano share both a common culture and medical belief system, as well as a common biological heritage, whether they are Mestizo or Indian, and, if Indian, whether Catholic or Methodist. Despite these commonalities, there are important divisions within the town. Usually they are phrased in racial terms, but those are simply markers for social class, that is to say, for access to resources.

The entire stratification system as it had developed in the early decades of this century was first weakened and then overturned by the arrival of Methodist missionaries in the 1930s, and then by a revolution and land reform in 1952. The original system saw Mestizos acting as managers of estates for an often absentee elite, as well as owners of significant resources themselves, overseeing a large population of Aymara Indians. Some of the Indians were enserfed on haciendas, others lived in free communities. The arrival of the Methodists was a threat to the Mestizos, for they taught literacy and a variety of trades, and provided medical care to the Indians who became Methodists. As a result of the land reforms in 1952, the Mestizos lost power and control of land, and the Methodist Aymara began to ascend to a dominant position.

In this fluid situation, Professor Crandon-Malamud argues, the various explanations of disease that people used became metaphors for their changing social relations and class positions. So, for example, dŽclassŽ Mestizos began to invoke Aymara explanations for their disease experiences. She writes that four themes may be distinguished in the national political economy, for each of which there is a corresponding aetiological category in Aymara thought, and that these categories are used increasingly by downwardly mobile Mestizos. She writes:

> These four themes emerged from integral components of the national political economy which have become entrenched institutions in Bolivian society. First, the notion of race has differentiated the elite (or 'whites'), mestizos, and Indians to justify unequal access to political participation, the legal system, and economic resources -- the very resources Kachitunos try to obtain through medical dialogue. Second, unequal access led to the development of an irrational economy dependent on the wasteful exploitation of Indian labor and the allocation of greatest economic risk to the Indian population which, after 1952, shifted to include the rural mestizo in the altiplano as well. Third, the entrenchment of caudillo political structures and use of suppression to maintain civil order has inhibited effective political mobilization of the agriculturally based Indian population and the rural mestizo as social classes. Fourth, a consequential antagonism between Indian and non-Indian has expressed itself among the rural Indian population as aversion to non-Indians, and among the mestizos in the countryside as a terror of Indian retaliation. From these four historical processes, the four metaphors that are expressed in etiology, diagnosis, and medical dialogue emerged as salient to contemporary medical dialogue, and today these metaphors are persistent referents in the strategies Kachitunos employ in that dialogue (pp. 46f).

The translation of these themes into aetiological categories, and their adoption by Mestizos, is as follows.

> They had learned to share with the Aymara a lack of confidence in the national government as an institution serving their interests. The majority described their relationship to the Bolivian government in much the same terms as the Aymara are said to have used to describe their relationship to the vecinos (mestizos) before 1952. As a consequence, the themes of hunger

> (for resources they no longer have), the vulnerability of subordination (now to the urban sector), victimization (to forces now beyond their control), and exploitation (by interests in the La Paz government that has neglected them) exacerbate the trauma of toil that Indians once performed for them but that they must now perform for themselves. These themes are the central metaphors running through their medical dialogue. They restructure Bolivian history as they are perceived to affect all Kachitunos, be they mestizo, Indian, or Methodist Aymara (p. 69).

It is not necessary here to provide details of the various aetiological categories that Professor Crandon-Malamud has described. I shall comment instead on what I view as the very considerable strengths as well as weaknesses of the book. Among the former is the very useful description of the historical changes in Bolivia over the past century; how they have been manifested at the local level; and how individuals have responded to, and coped with, them in their explanations of the misfortunes that have befallen them and their kin and neighbours. On the other hand, the 1:1 equation of historical processes with explanations of disease is not entirely convincing and seems to reflect a straining for consistency that may not be there.

Another strength is the rich detail that we are given concerning a variety of illness episodes. Often these are very well done, and in at least one case very humorous. Sometimes the result is confusion as characters appear and disappear without adequate explanation or reminders of who they are and why they are signficant, but for the most part they enrich the argument enormously.

A third strength is that the author tells us much about herself and the circumstances of her stay in the field. Indeed, this is a deeply personal book: her then eight-year old daughter accompanied her (and has provided the very nice drawings which illustrate the book but do not illuminate the text); she became involved with the lives of a number of her informants, and so on. It is good to know all this, for it reminds us that the observer is not omniscient but has interests and values of her own which contribute to the way she gathered and understood her data.

Along with these strengths, there are some accompanying weaknesses. A common thread runs through most of my criticisms. That is, Professor Crandon-Malamud has a tendency to set up straw men against which she poses her arguments and methods. But on closer inspection, she has done or assumed much that she objects to. For example, at the start, when acknowledging the help and influence of Robert Murphy, she writes: 'More than anything else in anthropology, that book (*Dialectics of Social Life*) helped me realize the significance of the fact that rarely do people mean what they say or say what they mean. That social law is the basic assumption of this book and perhaps the most notable difference between this book and those works in medical anthropology which apply positivist models and rely heavily on survey data' (p. xiii). (Parenthetically, this is undoubtedly why Professor Murphy's blurb on the dust jacket claims that 'the book's theoretical framework is impeccable'.) Having distinguished her work from positivist medical anthropology, she reveals surprisingly that she has relied heavily on a survey that she herself collected. In an Appendix she writes 'To assess any differences in health and disease notions among the cultural and religious segments of Kachitu which might affect medical dialogue and decision, I administered a scheduled interview to thirty-eight individuals representing these social divisions. Significant disparities between groups would render the political interpretation of their discussions and actions false' (p. 211). This was the case because she had to be certain that there was a common repertoire of disease explanations upon which all informants could draw equally. For if there was not, then it would make no sense to say that people were making choices from a common fund of information, and selecting those items which suited their particular (non-biomedical) needs.

The analysis of the survey was done by assuming that the 38 informants knew of 93 diseases. Thus there were 38x93 items of information (actually somewhat fewer since not everyone could answer all the questions). The 38 informants were drawn non-randomly and unequally from five different social categories. The responses were grouped and analysed as if they were independent items. For example, the seven Mestizos gave 399 responses about aetiology (whether magical, natural, or psychological); the three Methodists gave 136 responses; and the 11 Campesinos (Catholic Indians) gave 359 responses. But clearly, the responses are not independent of one another, and it is misleading to analyse them as if they were. It is the 38 respondents who ought to be the units of analysis. Thus, for each particular disease condition, one would have a table like the following:

Social Category	Magical Aetiology	Natural Aetiology	Psychological Aetiology	Total
Vecinos	*	*	*	7
Catholic Cholo	*	*	*	3
Methodist	*	*	*	3
Campesino	*	*	*	11
Yatiri (traditional Aymara healer)	*	*	*	14
Total	*	*	*	**38**

In the body of the table would be the distribution of responses. Almost certainly with five social classes and three aetiological categories, the chances of finding any significant differences are vanishingly small, even if a true difference existed in the population. Thus while the medical repertoire may well have been shared among all classes, Professor Crandon-Malamud's survey does not demonstrate it. But what is the point of criticizing surveys, then saying the results of her survey are vital to the validity of her interpretation, and then doing to small a survey (and analysing it inappropriately) to establish the validity of the point she says is so crucial?

A second example comes from her critique of previous medical anthropological studies which 'construct patient and healer as Rational Man, in the classical economic sense, looking for medical efficacy and understood in positivistic, or scientific, terms' (p. 10). The problem here is that the assumptions underlying the notion of Rational Man in the classical economic sense have to do with the consistency with which preferences are ordered. For example, if I prefer A to B, and B to C, if I am rational I should also prefer A to C. Moreover, it is assumed that I will act to maximize the preferences that are of most value to me. There is no assumption about what the preferences are or ought to be. For instance, if Australians prefer leisure to longer hours of work and higher incomes (as they seem to), then one would expect their choices to be made in such a way as to maximize leisure while still managing to keep body and soul together. Why they might prefer leisure to ceaseless hours of toil is another issue, and not one that is related to the assumptions underlying the construct of Rational Man in the classical economic sense. Furthermore, it is perfectly clear that people may not act rationally in the classical economic sense. They may choose what they don't prefer for any number of reasons: someone is holding them hostage; they have bad information, or low IQs, and so on. I am told that even some economists understand that.

In view of all this, it comes as a distinct surprise to discover on the following page Professor Crandon-Malamud's description of the model that underlies her explanations. 'This model is predicated not only on the assumption that ideologies are conditioned by material conditions and class relations,

but that individuals select from them and use them to accomplish their own ends' (p. 11). Sounds like Rational Man ('in the classical economic sense') to me. Indeed, throughout the book the language of choice, negotiation, and maximization is used to make the point that the Kachitunos behave rationally to achieve the ends that they regard as desirable, which might be described as the acquisition of social capital.

A third example is related to the first, the distinction Professor Crandon-Malamud makes between her work and 'those works in medical anthropology which apply positivist models and rely heavily on survey research'. I have already talked about the problem of survey data. Here I want to say something about the problem of 'positivist models'. My interpretation of what she means is that data correspond to something in the objective world, what is sometimes called the dogma of the immaculate perception. If this is indeed what is meant by positivistic models, then it is a caricature. Positivism does assume there is a world out there which can be known increasingly accurately, though how that happens is an issue to which I shall return. Professor Crandon-Malamud seems to believe that too, for she writes that people don't often mean what they say, or say what they mean, but it is possible for the observer to know what they mean. ('By asking why people entertain various medical beliefs and why they choose different medical resources, we can understand medical behavior and medical pluralism' [p.11].) Indeed, her whole book is meant to be an explanation of what people mean, and why they mean it. And she uses a variety of 'positivist' tools and rhetorical devices to substantiate her claim.

At the end of her book, Professor Crandon-Malamud writes: 'Opinions about health are social commentary about multiple facets of the life of the person under discusssion. Opinions are also a dimension of the author's self; they tell other people more about the author than about the subject being discussed' (pp. 206f). That she has told us *more* about herself than her informants is of course possible. Certainly she has told us a lot about herself, and we may infer that much about her has shaped the report she has given us. This is not something to which a positivist would necessarily take exception. Positivists like Karl Popper (1976) believe that science is a social activity and that through debate, challenge, new methods, and free discussion a clearer picture of the truth will emerge. This may happen only some of the time, but it does speak to a serious problem in anthropology. For much of the time anthropologists work alone in settings where other anthropologists cannot examine their work, debate their methods, or challenge their interpretations. In those instances where two or more anthropologists have worked in the same community on similar problems, debate has occurred: one thinks of the differences between Robert Redfield and Oscar Lewis on the nature of the village of Tepotzlan; of the differences among many anthropologists who have worked with Hopi Indians; and most recently of the Mead-Freeman debate on the nature of Samoan society. It is because anthropologists have so often worked alone that it is especially important that they be rigorous in their methods and honest and complete in their descriptions of how they collected their data (as Professor Crandon-Malamud has been). It may also be for this reason -- among others -- that many anthropologists have argued of late that the field can only produce 'coherent ethnographic fictions' (Clifford and Marcus 1986).

Professor Crandon-Malamud seems to be of two minds in this debate. On one hand she is critical of 'positivist science' and claims to have told us more about herself than about the Kachitunos. On the the other hand she has asserted that it is possible to explain the behaviour of her informants, and she writes as if she believes she has produced something other than a 'coherent ethnographic fiction'. I believe she probably has, and that what she has written is a valuable description of one of the ways in which large historical changes may be refracted in the most intimate details of daily life.

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Taming the Wind of Desire: Psychology, Medicine and Aesthetics in Malay Shamanistic Performance. By Carol Laderman. Berkeley: University of California Press, 1991. xvi+356pp. Hardback US$39.95

In this book, Carol Laderman introduces the reader to the world of the Malay shamanic healer through an account of her own fieldwork in the state of Trengganu. It is a fascinating book in which the author combines her own analysis of healing seances with annotated transcriptions of three seances, as well as music and interview transcriptions.

The author describes how spirit seances (called *main peteri*) have been the subject of study by Westerners for many years. Early writers from the colonial period presented them as examples of colourful exotica of the native mentality; later anthropological accounts stressed the social relations of the seance and the way they revealed the core values and beliefs of Malay culture. Recent research has focused more on analyses of the therapeutic effectiveness of the seances, as a therapeutic system not unlike the Western theory of psychoanalysis. Laderman herself views the Malay seance as a powerful healing ritual which encourages patients to look to their own personality dynamics at the heart of their problems. In this sense, she views the Malay seance as a unique case in the anthropology of non-Western healing in that it 'is, to my knowledge, the sole example of the ethnographic record of an indigenous non-Western method of nonprojective psychotherapy existing within the context of a shamanistic seance' (p.85).

To give weight to this assertion, Laderman introduces the reader to the Malay humoral theory of the 'inner winds' (*angin*) of the person. The chapter on inner winds is placed in the context of the most comprehensive historical and cross-cultural survey of Southeast Asian humoralism that I have come across in the extensive but patchy literature on this topic. The inner winds or *angin* is one sense approximate the Western idea of the 'temperament'. Each person needs the opportunity to express his or her *angin*, or various forms of illness and unhappiness will result. The shaman's role is to provide a ritual space where patients can be put in touch with their inner being through trance. The inner winds become an 'experiential reality' for the patient in the hands of the skilled shaman.

Of the three shamanistic performances transcribed in Part II of the book, only one explicitly illustrates the argument about the seance as a context of non-projective psychotherapy, developed by Laderman in the first part of the book. The other two seances concern cases of the spirit exorcism, not dissimilar to those encountered elsewhere in Southeast Asian seances. In one case, a seance for a sick shaman who was a key informant of Laderman's, there is also a strengthening of the patient's inner winds.

The transcriptions of the seances, while quite long (179 pages in all) make for fascinating reading. The text is well-annotated to convey the complex symbolism that is being drawn upon by the shaman and interpreted by his audience of patient, relatives and onlookers. Laderman's translations give the flavour of the poetic range of the shaman and his assistant as they engage in dialogues while imitating the persona of a vast pantheon of Malay spirits. She justifiably likens their skills to those of the bards of ancient oral traditions in European culture.

In this book, Laderman successfully conveys the powerful systems of meaning evoked by the shamanic healer. She takes issue with scholars who would argue that the efficacy of ritual symbols is primarily non-semantic: in the Malay seance, it is the words that heal, as a primary element of the total context. She takes pains to explain the ways in which the shaman's imagery evokes layers of previously acquired understandings among the audience.

This book will be a valuable resource for a wide readership, as it explores issues of healing, performance and cultural change. Medical anthropologists and practitioners interested in non-Western therapies will be drawn to Laderman's account of the seance as a healing ritual. The quality of the ethnographic research revealed in this study, and the manner of presentation and analysis, make the book accessible to an audience of non-Southeast Asian specialists. Its significance lies not least in the fact that these healing ceremonies have virtually disappeared in the last decade or so, a response to the increasing political power of Islamic orthodoxy on the Malay peninsula.

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Disease, Depopulation and Culture Change in Northwestern New Spain, 1518-1764. By Daniel T. Reff. Salt Lake City: University of Utah Press, 1991. xiii–330 pp. Hardback US$30.00.

About 500 years ago the contact between Western European civilization and the Amerindian cutures was initiated. It marked the onset of one of the most catastrophic population downturns that we know of, not because of the numbers involved – since we either suspect or have recorded vast mortalities elsewhere – but because of its suddenness, its speed and the implacability with which, in a short period of time, it managed to inflict an irrecoverable blow on an extraordinarily rich cultural diversity. Although the data are spotty at best, archaeologists, historians and demographers have, in their own ways and within the boundaries of their study domains, managed to painstakingly reconstruct plausible scenarios that make it possible to evaluate the magnitude of the catastrophe. To date most of the work, however, has been focused on what were not only some of the most densely populated areas but also those for which the recorded information was strongest. These roughly correspond to the main areas of domination of the Aztec and Inca Empires in Central Mexico and Peru respectively.

The book by Reff is a welcome addition, for it documents the nature, dimensions and consequences of Spanish penetration in Northwestern Mexico, an area about which little is known despite its being of great importance not only for the sake of completeness but because it provides a very different ecological, cultural and historical setting to test hypotheses that have emerged from studies in the other, much better known areas. The main objective of the book is to document the demographic and cultural impact of the contact between Spaniards and indigenous people in the area comprising what are now Northwestern Durango, Sinaloa, Sonora and Southwestern Chihuahua (or the

'greater Southwest'). The historical period under study is composed of two distinct time intervals. The first corresponds to the initial contact with Spanish explorers and stretches up to about 1591, a year in which the Jesuits established their first permanent mission in northwestern Mexico. The second period starts with the onset of sustained Jesuit influence and ends in 1767 with their expulsion from the Spanish overseas empire. Not only do these two periods differ in terms of the type of contact with the indigenous population and the nature and magnitude of its effects, but they also constitute two separate historical entities that can only be reconstructed by drawing from very different types of information. For the first period the main sources are a blend of archaeological records with the accounts and chronicles of the Spanish explorers who penetrated the area between 1530 and 1565. Taken by themselves these are not always reliable, accurate or very informative or penetrating accounts of indigenous society; but when used in combination with of the archaeological record and with some of the evidence for the second period, they undeniably shed light on the size and structure of the indigenous population. Information about the second period is in the form of annual reports (*anuas*) and other documents written and filed by Jesuits for the benefit of their Father Provincial; although a great deal of these documents are still preserved, many have been lost or remain to be found. The author assigns to this information much more reliability and accuracy than he does to the Spanish chronicle, since Jesuits had less interest in exaggerating certain conditions and were educated, perceptive and careful observers.

What was the magnitude of the population decline experienced after the initial contacts? How did the decline come about? Was there any variability across communities, depending on their original make-up, their location and their reaction to the initial contacts? How did the structure of indigenous society and its cultural superstructure change as a consequence of the decline? How was the Christian message received and absorbed in the various communities exposed to Jesuit influence and how did it modify indigenous beliefs?

These are important issues that get to the heart of some recurrent, unresolved controversies straddling not one but several fields. First there are problems that belong to the territories of the demographer and epidemiologist: was there a uniform collapse of the indigenous population or were there important variations? And if there were variations, what were the conditions that account for them? To what extent can the spread of infectious diseases be held responsible for the collapse of the indigenous population? What was the impact of violence and malnutrition that almost surely preceded and followed the destruction of infrastructure? Second, there are issues that are more properly dealt with in the realm of the theory of culture: what was the degree of acceptance of Christian values and how did it vary across communities? To what extent did indigenous culture absorb Western values and techniques? These questions, however, cannot be dealt with in isolation from population issues since there is always the possibility that acceptance of missionization or the selected absorption from an extraneous culture is more a function of the population collapse itself and the ensuing social disorganization than an admission of Western superiority.

The treatment of these issues, however, is somewhat uneven and though Reff is suggestive enough, in the end the reader is left with a sensation of inconclusiveness. In Chapter 2 the author reviews and extracts information from explorer chronicles to define what we could call the 'initial conditions' of the indigenous population: the size, age, sex and spatial distribution, their political and social organization, and their economic infrastructure. Unfortunately the 80 or so pages provide more detail on the itineraries of the various explorers than elaboration on the items enumerated above. There is a fair amount of repetition as each chronicle is dealt with separately but almost no view of ensemble emerges at the end of the review. The main message that one can draw from these pages is that the cultural, social and economic organization was quite sophisticated, that despite the absence of large

concentrations of population in city-like agglomerations, there were areas of high population density, and more importantly, that these initial conditions were highly variable in a fairly circumscribed territory. The reader is given few clues to discerning the nature of the variability and its geographic representation in the area under study. What one would have liked is to be able to recreate, by estimation from the chronicles, the various population settlements, their estimated size, their physical location, their characteristic culture, social organization, economic system, and lines of mutual contact, and then to piece them together in a less atomized view of the area.

An articulated view of settlements is of profound importance in understanding the information that Reff presents in Chapter 3, and it is accordingly missed. Here he presents the chronology of epidemics that were unleashed with full force shortly after the first contacts and surely long before the contacts with the Jesuits. One has to be prepared to accept at face value the rather rough criteria by which the nature of the various diseases is identified, although frequently it is difficult to single out one disease as the main culprit. It is very likely indeed that postulating the occurrence of clusters of diseases would represent better the actual epidemic episodes. Reff organizes his accounts along a time axis but neglects an equally if not more important part of the story, its spatial dimension. He acknowledges this when he invokes the existence of transport routes (*El Camino Real*) but, apart from a faint effort apparent in the drawings of several maps, there is no organized and coherent treatment of the spatial spread of epidemics. This is a difficult endeavour, requiring the deployment of sophisticated analytical techniques, perhaps more detailed information and surely a more articulated view of the patterns of settlements.

In addition, Chapter 3 does not rest solidly on a sound epidemiological foundation. The account given by Reff invokes somewhat haphazardly factors related to nutrition, immune function, pathogenecity, infectivity, lethality, relation between infections, and rapport between host and agent of disease to explain the presence or absence of this or that disease. Admittedly a more coherent treatment with the information that Reff has at hand is a tall order. But it is possible and even though it may leave glaring gaps at crucial points, it could have resulted in something more illuminating that a year-by-year account of sequels of epidemics.

The magnitude of the population decline and approximate assessment of the time scale in which it took place are presented in Chapter 4. Although the method used by Reff to arrive at his figures varies depending on the information that is available to him, a fairly typical application involves the estimation of the size of the initial population, usually from the Spaniards' chronicles or accounts, and estimates of the size of the same group or population from Jesuits' accounts. The difference is attributed to losses experienced during the period of maximum impact of epidemics. One can point to problems everywhere, from the use of household counts and approximate estimates of average number of members per household (or by estimating first the number of adults and then applying a guessed value of the fraction of the total population which is at adult ages) as a technique to derive total population, to the precise identification of the boundaries separating the group from others. It would be unfair to Reff and to the nature of the undertaking to demand absolute rigour or accuracy. But what one lacks is a sense of the magnitude of errors involved: by how much could estimates change if some assumptions are changed? Providing information on the robustness of the estimates ought to be an important part of the undertaking itself. Finally, I raise the issue of articulation yet again. One would think that population losses, frequency and intensity of epidemics and pre-existing conditions – the themes of Chapters 4, 3 and 2 respectively – are related; and yet the author does not give the reader a sense of how they are related at all. The only exception to this is the extraordinary case of the Tarahumara, a people who suffered relatively little after withdrawing to an inhospitable terrain. But surely initial density and pre-existing lines of contacts as well as the social, political and economic organization of the group are

related to the magnitude of depopulation. The connections are made here and there – reference to Apaches, Seri and Navaho nomad existence and Tarahumara post-contact reclusiveness – but never in any systematic way to shed light on the existence of possible regularities.

The nature of the cultural transformation is dealt with in Chapter 5. This, and the conclusion of the volume, are engaging if tentative sections of the book. More speculatively but also displaying a very good sense for what the data reveal, Reff disparages the hypothesis of cultural adaptation as a result of indigenous recognition of Western superiority. He goes to great lengths to show that there was no such thing as technical superiority and argues, in my mind fairly convincingly, that the acceptance of religion, in all sorts of syncretic forms, can probably be explained by the population debacle itself: the sudden disappearance of the adult and older generations, the disruption of the process of socialization and the complete dissolution of the indigenous political system weakened the basis of indigenous religiosity and created a vacuum promptly filled by an assortment of indigenous adaptations of Jesuits' Christian teachings.

The book ends abruptly, for in its final pages Reff poses a tantalizing puzzle that is left unresolved. Archaeologists have postulated that a cultural collapse affected northwestern Mexico and the American Southwest during the late fourteenth and fifteenth centuries. It is widely assumed that this was the result of climatic conditions, resource mismanagement and warfare. Reff offers a different interpretation, namely, that these cultures vanished as a result of the impact of diseases brought about by their contact with Spaniards. To do so he must first of all dispute the precise timing of their disappearances: collapse should have occurred closer to the middle of the sixteenth century, not in the fourteenth or fifteenth century. Reff invokes a combination of fairly well accepted archaeological evidence and a mistaken interpretative framework as the main culprit for the error in the dating of these peoples'disappearances. But his argument is not forcefully compelling. He succeeds, however, in planting the doubt and in showing that his and future work on these cultures may have important implications for the manner in which we conceive the unfolding of history not just after but possibly before the initial contact with Western Europe.

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