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PREFACE

The purpose of the *Population Bulletin of the United Nations,* as stipulated by the Population Commission, is to publish population studies carried out by the United Nations, its specialized agencies and other organizations with a view to promoting scientific understanding of population questions. The studies are expected to provide a global perspective of demographic issues and to weigh the direct and indirect implications of population policy. The *Bulletin* is intended to be useful to Governments, international organizations, research and training institutions and other bodies that deal with questions relating to population and development.

The *Bulletin* is prepared by the Population Division of the Department of International Economic and Social Affairs of the United Nations Secretariat and published semi-annually in three languages—English, French and Spanish. Copies are distributed widely to users in all States Members of the United Nations.

Although the primary source of the material appearing in the *Bulletin* is the research carried out by the United Nations Secretariat, officials of governmental and non-governmental organizations and individual scholars are occasionally invited to contribute articles.

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Explanatory notes

Symbols of United Nations documents are composed of capital letters combined with figures. Mention of such a symbol indicates a reference to a United Nations document.

Reference to "dollars" ($) indicates United States dollars, unless otherwise stated.

The term "billion" signifies a thousand million.

Annual rates of growth or change refer to annual compound rates, unless otherwise stated.

A hyphen between years (e.g., 1984-1985) indicates the full period involved, including the beginning and end years; a slash (e.g., 1984/85) indicates a financial year, school year or crop year.

A point (.) is used to indicate decimals.

The following symbols have been used in the tables:

Two dots (. .) indicate that data are not available or are not separately reported.

A dash (—) indicates that the amount is nil or negligible.

A hyphen (-) indicates that the item is not applicable.

A minus sign (-) before a number indicates a deficit or decrease, except as indicated.

Details and percentages in tables do not necessarily add to totals because of rounding.

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FERTILITY PATTERNS AND CHILD SURVIVAL: A COMPARATIVE ANALYSIS

*John Hobcraft**

SUMMARY

This article presents information on the impact of fertility patterns upon child survival for 18 countries from the Demographic and Health Surveys (DHS). Results are also contrasted with those from earlier World Fertility Surveys (WFS). The findings generally serve to confirm that children born to teenage mothers, especially those under age 18, experience considerable excess mortality before age 5. More important at the population level is the deleterious effect of short birth intervals for child survival. Data quality, although a problem, is shown not to have a major distorting impact on these findings. Further analysis in 10 categories of family formation is carried out. The more important findings to emerge are that the overall impact of poor timing of births on child survival is substantial in many countries but has been improving over time, probably as a result of increased use of family planning, in a number of cases (e.g., Colombia, Morocco). On the other hand, child mortality gains in Senegal are being inhibited by a worsening pattern of timing of births. The policy implications of these findings are briefly assessed.

BACKGROUND

Results from analysis of the World Fertility Survey demonstrate a consistent and powerful association between fertility patterns, particularly short birth intervals and teenage child-bearing, and the chances of child survival (see, for major comparative analyses, Hobcraft, McDonald and Rutstein, 1983 and 1985; Palloni and Millman, 1986; and Hobcraft, 1987). These findings came at a time when international funding of family plan-

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The work for this article was carried out with support from the Population Division of the Department of International Economic and Social Affairs of the United Nations Secretariat. The author is particularly grateful to Birgitta Bucht and Trevor Croft for their support. The tabulations of mortality for the DHS were derived using a heavily modified version of an Integrated System for Survey Analysis (ISSA) procedure originally written by Guillermo Rojas.

ning programmes was under threat and commanded wide attention as a result. The renewed emphasis on the health rationale (as opposed to a population control or human rights rationale) for family planning was highlighted by the Nairobi Conference on Better Health for Women and Children through Family Planning in 1987, which brought together as co-sponsors an unprecedented range of international agencies—International Planned Parenthood Federation, Population Council, United Nations Children's Fund (UNICEF), United Nations Development Programme, United Nations Population Fund, World Bank and World Health Organization.

The consistency and strength of the deleterious effects of a short preceding birth interval (and, to a lesser extent, of a short subsequent interval) on child survival came as a surprise to many scholars, although findings on this issue date back to at least the 1920s (for a discussion of this literature, see, e.g., Hobcraft, McDonald and Rutstein, 1983; and Hobcraft, 1987). A great deal of effort and concern has gone into the related questions of whether these observed effects are real or artefactual and what the causes of the observed association might be.

The effects of short birth intervals on child survival persist (though they are sometimes attenuated, for example, in the case of controls for sibling survival) in the face of *all* controls that have been tried, including the other bio-demographic correlates of child mortality (sex of child, age of mother at birth, birth order, sibling survival and breast-feeding duration), and the available socio-economic correlates (mother's and her husband's education level, husband's occupation, type of place of residence etc.).

Attempts to clarify the mechanisms involved in causing an association between birth intervals and child survival—if it is indeed a causal one—have tried to distinguish between sibling competition and maternal deprivation, but have usually been inconclusive (e.g., Cleland and Sathar, 1984; Hobcraft, McDonald and Rutstein, 1983 and 1985; for reviews of the evidence, Hobcraft, 1987; and National Research Council, 1989). Evidence is usually lacking on some key variables which might aid an understanding of the mechanisms, including birth weight (although see Da Vanzo, Butz and Habicht, 1983; and Da Vanzo, Habicht and Butz, 1984, for some evidence on persistence of these effects with controls for birth weight and previous obstetric history).

Given the impact of these findings, there is quite reasonable concern as to whether the association between short birth intervals and child survival is a causal one. Most of the major papers which present results on the association are quite careful to point out and address, as far as data limitations permit, the problems subsequently raised. Prematurity almost certainly serves to exaggerate the apparent excess mortality associated with *very* short preceding intervals. Miller (1989) suggests this as an important explanation of the observed association for perinatal mortality in Sweden and Hungary, two countries with low overall child mortality and good compensatory health care, although quite strong differences still remained.

(The question of whether short birth intervals are one of the causes of prematurity is not usually addressed in attempts to assess this issue.) Several studies on developing countries had attempted to address this issue and concluded that the impact of prematurity on the observed associations was quite small (Wolfers and Scrimshaw, 1975; Hobcraft, McDonald and Rutstein, 1985; and Pebley and Stupp, 1987). This conclusion still seems warranted.

Issues of data quality inevitably loom large in trying to assess results from retrospective event histories. Again, most of the major analyses attempted to address these issues and concluded that differential omission of dead children would lead to a *downward* bias in the observed associations. Perhaps the strongest critique of the main analyses on this count has come from Potter (1988b), who constructs possible combinations of reporting errors which might lead to spurious associations, but provides no evidence to buttress his key assumptions (for example, that a mother is likely to date the death of a child as far back as she can, while maintaining the integrity of birth order). Potter does not acknowledge a study that explicitly addresses the issue of differential imputation of dates of birth for dead and surviving children (Trussell, 1987). Trussell's simulation study strongly suggests that differential imputation makes little difference to the substantive interpretation of the estimated effects in a complex regression model, which replicated the model used by Hobcraft, McDonald and Rutstein (1983).

Potter (1988b) also raises the possibility that effects are spurious due to consequences of links between child-spacing and access to and use of health care, particularly contraception. Once again, the discussion is hypothetical and does not quantify the potential biases discussed. Potter assumes that "contraception is surely the most important proximate determinant of interval length" and that, given the association of contraceptive use with other characteristics associated with child survival, including use of health-care facilities, the observed correlations of child survival with interval length may be spurious. Since most contraceptive use in most of the WFS countries was for stopping purposes, it is surely true that the most important proximate determinant of the proportion going on to have the next birth is contraception. It is by no means self-evident that closed intervals for contraceptive failures are necessarily longer. Equally, Potter does not address the issue as to how it can be that the findings on the association between short birth intervals and child survival are so persistent across societies with such widely differing health service provision. Lantz, Partin and Palloni (forthcoming) take up the issues raised by Potter in more detail and rebut, with empirical evidence, many of Potter's assertions and hypotheses, although the nature of the WFS data does not always permit conclusive analysis.

A further interpretational problem arises with the clustering of child mortality within families. Hobcraft, McDonald and Rutstein (1985) argue that the survival status of the preceding child is a proxy variable for the family mortality environment and suggest that the deleterious effects of a

short preceding interval are approximately the same, regardless of the survival status of the preceding child, once this is taken into account. Aaby (1988) reviews evidence that measles transmission and case fatality are associated with overcrowding and thus with a concentration of young children in the family. This suggests a possible mechanism associated with crowding, rather than short intervals *per se*. As yet, the empirical assessment of the consequences of clustering of child deaths for the observed associations between birth intervals and child survival is scant (although see Da Vanzo, Butz and Habicht, 1983, for an attempt to control for clustering of young children; and Curtis, McDonald and Diamond, 1991, for an illustrative analysis of the use of random-effects models to allow for the statistical biases that might be induced by ignoring clustering within families).

A further series of findings suggest that teenage child-bearing is also disadvantageous for child survival (e.g., Hobcraft, McDonald and Rutstein, 1985; and Hobcraft, 1987, who further refines the excess risk as being greater for children born to mothers aged under 18). These authors were well aware of the possible limitations of inferring a direct bio-demographic causality, stressing in the 1985 conclusion that "there are sound biological explanations, as well as the likely association of teen-age pregnancy and socio-economic disadvantage in societies whose members marry later in life". Yet Geronimus (1987) was inclined to be dismissive of any biological link, even for third world societies, on the basis of evidence from the United States of America. The degree of social selection for disadvantage with early child-bearing is inevitably much greater in the United States (and other societies with normally delayed child-bearing) than in (at the other extreme) Bangladesh, where fully 88 per cent of all first births occurred to teenage mothers (Hobcraft, 1987).

Beyond the issue of whether the observed associations between birth intervals and teenage child-bearing and child survival are real, there are a number of questions concerning the potential policy implications. The possibility of gains in overall survival chances of *up to* 30 per cent was first raised by Trussell and Pebley (1984). This issue was again addressed in some detail by Hobcraft (1987), who also stressed that shifts to less favourable fertility patterns in sub-Saharan Africa could lead to increases in overall child mortality. Both the aforementioned studies were careful to stress that these possible gains in child mortality from improved child-bearing patterns were *maximal* estimates, on the assumption that all the observed associations were real.

Debate about this issue of possible gains in child mortality from improved family planning was focused by Bongaarts (1987), who suggested that likely changes in child mortality with fertility decline would be upwards, rather than downwards. Bongaarts's analysis (1988) has been taken to task by a number of authors on several counts (see Potter, 1988a; Trussell, 1988; Hobcraft, 1988; and Palloni and Pinto, 1989): overstatement of excess mortality associated with first births; the assumption that the proportion of short birth intervals was likely to rise with increased fer-

tility control, based on the predicate that all high-fertility societies have long birth intervals (as in sub-Saharan Africa) and the assumption that all low-fertility societies (typified by Latin America) have short birth intervals (this ignores the evidence to the contrary from within the Latin American WFS programme, and runs directly counter to Potter's assumption quoted earlier); and the assumption that the Matlab thana in Bangladesh, with its reliable data, represented a useful quasi-experiment in this context, whereas birth intervals were already long enough to ensure that family planning could not provide gains from further lengthening of intervals (except, possibly, where the preceding child died).

However, it is clear that such policies would need to be applied selectively, depending on the existing child-bearing patterns. For Latin American, Middle Eastern and North African countries for which relevant data are available, the prevalence of short and very short birth intervals suggests that family planning policies targeted on improving birth-spacing may help to lower child mortality. For many sub-Saharan African countries, the proportion of short birth intervals is low, mainly from traditional fertility restraints; in these countries the potential of family planning for child-spacing might serve to prevent a rise in child mortality from changing patterns of child-bearing during the onset of their fertility transition.

Data and methods

The Demographic and Health Surveys provide a further opportunity to address these issues. The evidence from the World Fertility Survey typically covered the 1960s and part of the 1970s. Since there have been major changes of emphasis in child survival programmes and, indeed, family planning programmes during the 1980s, there is a need for a more up-to-date assessment of the associations. It is probably still too early to assess whether a greater stress on child-spacing through contraception has had much impact on birth intervals or, consequentially, upon child survival. The data from the Demographic and Health Surveys, which we shall consider here, typically span the 1970s and the 1980s (see table 1). Hardly any information is available after the time of the Nairobi Conference (1987) and mature assessment of actual possible impact in most countries will have to await the emergence of studies during and covering the 1990s. Nevertheless, it is clearly desirable to examine more recent information in order to assess whether there has been any change in the relative risks of child mortality associated with child-bearing patterns, and whether such patterns have changed for the better or worse, in terms of their likely impact on child mortality.

The approach adopted here is to replicate the straightforward tabular analysis presented by Hobcraft (1987), for 18 WFS countries. By chance, the results from 18 DHS are considered here (see table 1 for details of the dates, sample sizes and overall child mortality). Moreover, eight of the countries covered in the earlier study are also included here, enabling direct comparisons of change over time to be made. The opportunities for

TABLE 1. DATA FROM DEMOGRAPHIC AND HEALTH SURVEYS IN 18 COUNTRIES AND WORLD FERTILITY SURVEY IN 8 COUNTRIES

Country	*Survey year*	*Total births of children to mothers aged under 35*	*Total deaths of children to mothers aged under 35*	*Death rate by age 5 (per 1,000)*	*Period covered by DHS*	*WFS birth cohorts*	*Overlap (years)*
Brazil	1986	8 874	877	99	1971-1986		
Colombia	1986	7 121	419	59	1971-1986	1960-1975	5
Dominican Republic	1986	11 439	1 016	89	1971-1986		
Ecuador	1987	7 721	751	97	1972-1987		
Peru	1986	8 323	971	117	1971-1986	1961-1977	6
Trinidad and Tobago	1987	4 878	173	35	1972-1987	1961-1976	5
Burundi	1987	8 263	1 590	192	1972-1987		
Ghana	1988	8 846	1 357	153	1973-1988		
Kenya	1988/89	17 773	1 625	91	1973-1989	1962-1977	4
Liberia	1986	10 851	2 512	232	1971-1986		
Mali	1987	8 210	2 421	295	1972-1987		
Morocco	1987	15 310	1 946	127	1972-1987	1964-1979	8
Senegal	1986	9 889	2 206	223	1971-1986	1962-1977	7
Tunisia	1988	10 683	819	77	1973-1988		
Zimbabwe	1988	8 104	682	84	1973-1988		
Indonesia	1987	24 683	2 865	116	1972-1987		
Sri Lanka	1987	10 699	471	44	1972-1987	1959-1974	3
Thailand	1987	11 035	558	51	1972-1987	1959-1974	3
Total		192 702	23 258				

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

change to appear are somewhat circumscribed by the overlap between the periods covered by the analyses of the WFS and DHS results, as indicated in table 1. Life-tables have been calculated covering the mortality experience over 15 complete years plus the year of the survey (this contrasts with the earlier analysis, which only considered the experience of birth cohorts born 1-16 years before the WFS programme, thus excluding about half of the mortality experience during the period 11-16 years before the survey). In total, the study uses information from the DHS concerning the survival chances of about 193,000 children and some 23,000 deaths. In order to keep the amount of material manageable and to summarize overall patterns, we shall only consider chances of survival to age 5 and not disaggregate experience by age of the child.

The analyses are presented as simple tabulations and the lack of controls for other variables must be borne in mind. The robustness of findings from WFS to such controls gives some reassurance that the results should not be unduly misleading. However, the magnitude of the observed associations might well be altered by the introduction of further controls (multivariate analysis is planned). The comparisons with the same tables for WFS should prove reasonably robust to the lack of further controls.

As a first stage of the analysis, the effects on child survival of mother's age in isolation, first for the DHS surveys and then a comparison with earlier WFS results, will be considered, followed by an examination of the association of child mortality with length of the preceding birth interval. This is followed by a digression to examine the robustness of the findings to date imputation. The results on age of mother and preceding birth interval are then brought together, using a 10-category classification of "family formation" pattern, which was developed by Hobcraft (1987) and has subsequently been used in multivariate analysis by Bobadilla, Schlaepfer and Alagon (1990). Finally, the possible gains in child survival that have occurred over time and might be made in the future as a result of alterations in family-formation patterns are considered briefly.

The categorization of family-formation pattern is achieved as follows, for all births to mothers aged under 35 at the time of the birth (births at higher ages are excluded because of the heavy censoring of experience during the 15 or so years before the surveys for this group). Births are divided into first and higher-order groups. The first births are subdivided into those to teenage mothers and the rest. Later births (a term used here to connote second and higher-order births) are divided according to whether the preceding birth interval is less than two years ("poor" spacing or "short" intervals) or not ("well" spaced or "long" intervals). Each of these two categories of later births is further subdivided into four; births to teenage mothers, and three categories of births to 20-34-year-old mothers—namely, those who are "slow", "medium" and "fast" reproducers up to the time of the birth in question. "Slow" reproducers have two births if aged 20-24, two or three if aged 25-29, and two to four births if aged 30-34. The "medium" group contains 20-24-year-olds with three births, 25-29-year-olds with four or five births, and 30-34-year-olds with five or six children. The "fast" category comprises 20-24-year-olds with four or more births, 25-29-year-olds with six or more, and 30-34-year-olds with seven or more births.

Age of mother

Table 2 presents the information on differences in child-bearing patterns and mortality risks by age of mother for the 18 DHS surveys. Only 6.8 per cent of births to mothers aged under 35 were to teenagers in Tunisia, with this proportion also being low (about 10 per cent) for Sri Lanka and Burundi. This proportion was around 15 per cent in Peru, Brazil, Morocco and Thailand. For the remaining 11 countries, fully 20 per cent of births considered here were to teenage mothers, with this proportion reaching 28 per cent in Liberia. More than 10 per cent of all births to women under age 35 during the period considered were to mothers aged less than 18, the age group identified in earlier studies as being at high risk of child death, in the Dominican Republic, Kenya, Liberia, Mali and Senegal.

Turning to the extent of excess mortality risk, table 2 also shows the relative risks of death for children born to each age group of mother, with

TABLE 2. CHILD-BEARING PATTERNS AND MORTALITY RISKS FOR 18 DEMOGRAPHIC AND HEALTH SURVEYS COUNTRIES, BY AGE OF MOTHER

Country	Births[a]					Relative risk[b]					Mortality rate q(5) for births to mothers aged 20-34	Population attributable risk
	Under 18	18-19	20-24	25-29	30-34	Under 18	18-19	20-24	25-29	30-34		
	(Age of mother)					(Age of mother)						
Brazil	66	95	358	312	169	167	122	102	102	91	93	107
Colombia	84	115	364	272	165	162	105	96	88	130	56	106
Dominican Republic	106	127	348	262	157	161	100	99	99	104	83	106
Ecuador	79	117	353	276	175	141	94	106	101	87	95	103
Peru	66	100	325	300	208	158	102	99	95	109	112	104
Trinidad and Tobago	75	125	376	274	148	210	131	109	98	81	32	112
Burundi	33	83	347	322	216	108	126	103	102	92	188	102
Ghana	89	108	334	279	190	151	105	104	98	96	146	105
Kenya	107	121	331	265	177	146	119	111	92	91	85	107
Liberia	154	127	327	242	150	138	121	110	92	91	213	109
Mali	129	113	306	265	187	130	129	103	97	99	275	107
Morocco	57	90	337	306	211	163	127	102	101	95	120	106
Senegal	129	117	326	253	175	125	104	102	97	101	215	104
Tunisia	12	56	339	351	242	205	102	102	103	92	76	101
Zimbabwe	94	119	340	269	178	135	126	106	91	102	79	106
Indonesia	95	119	347	264	176	150	118	102	98	99	109	107
Sri Lanka	30	71	331	343	225	158	96	96	110	90	43	101
Thailand	53	105	361	300	180	113	132	98	96	110	49	104
Mean 18 DHS	81	106	342	286	185	151	114	103	98	98		106
Mean 18 WFS	91	104	324	284	197	141	112	99	97	105		105

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS.

[a] per 1,000.

[b] 20-34 = 100.

the overall rate for children born to women aged 20-34 being used as the reference category. Taken overall, the average across all 18 DHS countries implies considerable excess mortality risks for children born to mothers aged under 18, at 51 per cent. There is a small (14 per cent) average excess associated with the mother being an older teenager. The excess risk for younger teenage mothers is perhaps a little higher in Latin America and a little lower for the sub-Saharan African countries, although several of the latter group have apparently greater excess mortality risks for the children of older teenage mothers, perhaps indicative of data quality problems. For the younger teenage mothers (under 18) the pattern of excess risk is nevertheless fairly consistent, with only four countries falling outside the range of 125 to 167, and all but one (Thailand) of these exceptions being based upon the experience of fewer than 500 births in the

group. Overall, the results also agree quite well with the average pattern for the 18 WFS countries considered by Hobcraft (1987), despite the two groups of countries having only eight in common and being a happenstance selection (although with some purposive choice for the 18 WFS countries).

The final column of table 2 presents the risks attributable to population for each country. The risk is simply a calculation of how much excess mortality is observed in each country as a consequence of the combined impact of the observed excess risks and the child-bearing patterns. It is identical to the ratio of the overall death rate by age 5 for all children included here (mothers under 35) to the death rate for the baseline group (restricted to mothers aged 20-34 at the time of the birth). It is thus an estimate of the mortality that might be avoided if all births occurred to mothers in the 20-34 age range *and* all the excess mortality for teenage mothers was simply due to their being teenagers and not a consequence of other characteristics selectively associated with teenage motherhood. As always, this means that the potential gains from delaying child-bearing are almost certainly overstated by this simplistic analysis. Clearly, the potential for population-level reductions in child mortality from delaying child-bearing to, at least, age 18 and, perhaps, to age 20 is not massive. This is not surprising, since usually well over 70 per cent of all births are in the reference group for all 18 countries considered. Although the excess risks are substantial, the proportions of births experiencing the excess risk are not massive. The potential maximal gains range from 1 to 12 per cent.

We note, though, that the potential gains for first births would be considerably greater, since the proportion of first births which are to teenage mothers exceeds two thirds in Kenya, Liberia, Mali and Senegal, and is well over a third in most other countries (the only exceptions being Tunisia and Sri Lanka). Indeed, the maximum potential gain in survival chances for first births might be about 40 per cent in the Dominican Republic and Peru; around 30 per cent in Brazil, Colombia and Liberia; minimal, at approximately 5 per cent, for Tunisia and Sri Lanka; and between 10 and 20 per cent elsewhere. This potential gain for first births would also be of increasing significance as fertility levels fall, since first births then constitute a higher fraction of all births. Note also the implications for the discussion by Bongaarts (1987), who suggests an effect in the opposite direction.

Table 3 provides a comparison of results for the eight countries where we have results available from both WFS and DHS. The fraction of births to teenage mothers has remained remarkably constant over time for the first five countries, but there appears to have been a significant reduction in the fraction of births to teenage mothers in Morocco and Sri Lanka, and a significant rise in Thailand, perhaps as a consequence of reduced fertility at older ages. The excess risk of child death associated with births to younger teenage mothers appears to have increased over time in all but Sri Lanka, where the excess risk was already high and the numbers of births to such mothers is anyway low. The excess risk for children of very young

TABLE 3. COMPARISON OF DATA FROM WORLD FERTILITY SURVEY AND DEMOGRAPHIC AND HEALTH SURVEYS ON CHILD-BEARING PATTERNS AND MORTALITY RISKS IN 8 COUNTRIES, BY AGE OF MOTHER

Country	Births[a] (Age of mother)					Relative risk[b] (Age of mother)					Mortality rate q(5) for births to mothers aged 20-34	Population attributable risk
	Under 18	18-19	20-24	25-29	30-34	Under 18	18-19	20-24	25-29	30-34		
Colombia												
WFS	82	111	334	279	194	127	125	100	100	101	96	105
DHS	84	115	364	272	165	162	105	96	88	130	56	106
Peru												
WFS	68	90	317	294	231	115	104	96	100	106	158	101
DHS	66	100	325	300	208	158	102	99	95	109	112	104
Trinidad and Tobago												
WFS	78	112	361	278	171	152	80	98	89	122	46	102
DHS	75	125	376	274	148	210	131	109	98	81	32	112
Kenya												
WFS	116	117	324	259	184	142	117	96	101	105	137	107
DHS	107	121	331	265	177	146	119	111	92	91	85	107
Senegal												
WFS	127	121	302	261	189	116	105	102	100	96	256	102
DHS	129	117	326	253	175	125	104	102	97	101	215	104
Morocco												
WFS	90	100	320	287	203	146	122	104	96	99	142	106
DHS	57	90	337	306	211	163	127	102	101	95	120	106
Sri Lanka												
WFS	58	83	309	319	231	164	109	100	104	96	75	105
DHS	30	71	331	343	225	158	96	96	110	90	43	101
Thailand												
WFS	44	83	325	310	238	103	123	98	96	107	105	102
DHS	53	105	361	300	180	113	132	98	96	110	49	104
Mean												
WFS	83	102	324	286	205	133	111	99	98	104	127	104
DHS	75	106	344	289	186	154	115	102	97	101	89	106

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS.

[a]Per 1,000.

[b]20-34 = 100.

mothers in Thailand is strikingly low in both the WFS and the DHS; it is also low for Senegal. With the exception of Trinidad and Tobago, where sample sizes in both surveys are small, the risks attributable to population do not alter much over time. Whatever the potential gains from delaying motherhood, they are not yet being realized in those eight countries.

LENGTH OF PRECEDING BIRTH INTERVAL

Table 4 gives results on survival chances to age 5 for later births (order two and above) and on the distributions of births by length of the

TABLE 4. SURVIVAL CHANCES TO AGE 5 AND BIRTH DISTRIBUTION FOR 18 COUNTRIES, BY LENGTH OF PRECEDING BIRTH INTERVAL

Country	Death rate[a] (Preceding interval[b])					Relative risk[c] (Preceeding interval[b])					Birth distribution[a] (Preceeding interval[b])					Population attributable risk
	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+	
Brazil	185	105	72	69	38	259	146	100	96	53	295	179	363	97	66	151
Colombia	102	68	51	33	25	200	132	100	64	48	231	198	375	121	75	121
Dominican Republic	143	88	73	55	90	195	120	100	75	123	262	186	400	93	58	128
Ecuador	178	94	82	50	46	218	114	100	61	56	234	178	417	108	64	123
Peru	201	148	103	63	30	195	144	100	61	29	216	191	444	92	57	121
Trinidad and Tobago	60	29	28	36	24	216	105	100	128	85	238	181	341	127	112	130
Burundi	297	223	176	99	80	168	127	100	56	45	103	177	605	89	27	106
Ghana	210	187	145	120	107	144	129	100	83	74	89	136	594	133	47	104
Kenya	156	97	80	51	36	195	122	100	64	45	141	197	537	89	36	113
Liberia	311	262	207	140	88	150	126	100	67	42	170	189	487	103	51	107
Mali	469	320	235	143	133	200	136	100	61	57	196	177	486	96	45	120
Morocco	220	127	102	53	56	216	125	100	52	55	182	190	513	81	34	120
Senegal	287	240	212	141	150	135	113	100	66	71	90	174	636	75	24	102
Tunisia	154	83	55	34	34	278	150	100	62	62	216	188	471	90	34	143
Zimbabwe	170	106	71	52	64	238	149	100	72	90	90	154	603	112	41	116
Indonesia	199	132	105	71	75	188	125	100	67	71	142	162	502	127	67	111
Sri Lanka	76	50	38	36	46	198	131	100	92	121	132	174	492	129	73	119
Thailand	117	51	46	38	44	253	112	100	82	96	147	162	455	144	92	121
Mean 18 DHS						203	128	100	73	68	176	177	484	106	56	118
Mean 18 WFS						199	131	100	73	83	217	205	478	72	28	125

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.
NOTE: 15 years before DHS.
[a]Per 1,000.
[b]Months.
[c]24-47 = 100.

preceding birth interval. The average relative risk of child death, compared with an interval of 24-47 months, is about double for very short intervals (1-17 months, where prematurity may contribute to the excess risk). There is around a 30 per cent excess risk for intervals of 18-23 months. The agreement with the average relative risk patterns shown across the 18 WFS countries in the earlier study is remarkable.

The low relative risks for longer preceding intervals (of four years or more) occurred consistently for the WFS too, and have sometimes been at least partially attributed to the impact of possible omissions of dead children, which may also lead to a downward bias of relative risks for short intervals. An alternative interpretation of the low risks associated with long intervals might be Potter's suggestion (1988b) of longer intervals being associated with higher use of contraception and concomitant access to and use of health services. Excess risks are particularly high for children born less than two years after the previous birth in Brazil, Tunisia and Zimbabwe, and for very short intervals in Thailand.

The excess risks of death are atypically low for children born after very short birth intervals in Burundi, Ghana, Liberia and Senegal. These four are also predominantly the countries where the fraction of such births is very low (with Liberia being an exception with respect to the fraction of such births, and Zimbabwe being an exception in terms of relative risks). It is at least plausible that underreporting of deaths at very short intervals is occurring for these countries, leading to a downward bias in the observed excess mortality. We also note that imprecision of dating is a prevalent feature in those four countries (although not exclusively). With the exception of Mali (where we shall be indicating severe data quality problems later), the four countries with low excess risk of death for children born after very short intervals are those with the highest overall levels of child mortality before age 5 (more than 15 per cent of children dying—see table 1). The relatively small excess risks may, perhaps, be genuine, reflecting the fact that differentials emerge most strongly at moderate levels of overall child mortality.

Not only are the excess risks of child mortality associated with short preceding birth intervals high; the proportions in the high-risk groups are also high. Over 40 per cent of all later births occur less than two years after the previous one in all six countries from the Americas and in Tunisia. This replicates the earlier findings from the World Fertility Survey. About 35 per cent of all later births occur at short intervals in Mali, Morocco, Liberia and Kenya; the fraction is around 30 per cent for the Asian countries considered here; and fewer than 30 per cent of births are poorly spaced in Burundi, Senegal, Zimbabwe and Ghana.

The combined impact of the high excess risks and the distribution of children across the categories of previous interval length is summarized once again through the population attributable risks. These are strikingly high for Brazil (51 per cent excess) and Tunisia (43 per cent), suggesting considerable room for improved child-spacing patterns or compensatory health interventions to alter the pattern of relative risks (whatever such

interventions may be), to reduce overall levels of child mortality for births other than the first. Indeed, a target of reducing these population attributable risks to somewhere around 20 per cent (a level attained in several countries) ought to be achievable.

Table 5 also compares experience from the WFS and DHS programme for eight countries. Although there is some evidence of change in the pattern of relative risks over time, the most striking changes occur in the distribution of births by length of previous interval. The fraction of short intervals (under 24 months) declines by 7-9 percentage points in Peru, Kenya, Morocco, Sri Lanka and Thailand, and by a massive 13 and 15 percentage points, respectively, in Trinidad and Tobago and Colombia. The child-spacing pattern worsens in Senegal, with an increase of 4 percentage points in the proportion of poorly spaced births. The population attributable risk for Colombia suggests a significant 12 per cent reduction in overall child mortality for later births, primarily as a result of improved spacing patterns. The gain for Trinidad and Tobago is even larger, being about 16 per cent, although an improvement in the relative risk for births at intervals of 18-23 months contributes to this. There are also significant gains in the population attributable risk for Morocco (14 per cent), Thailand and Kenya (both 7 per cent). A widening of the relative risks for Sri Lanka negates the improvements in child-spacing, and the population attributable risk worsens over time (by 9 per cent) as a consequence. The worsening of the child-spacing pattern in Senegal is not sufficient to make a major difference to the population attributable risk. These quite substantial changes in child-spacing patterns over a short period (typically about 10 years), especially with a significant overlap in the period covered between the successive surveys, do strongly suggest that the gloomier views about the worsening of these patterns with fertility decline (typified by Bongaarts, 1987) are not tenable. On the contrary, significant improvements in child-spacing patterns are occurring, and child mortality is probably being modestly reduced as a consequence. We cannot be sure, on the basis of the information presented here, that it is indeed family planning which is responsible for the changes observed.

More detailed distributions of births by preceding interval are presented in table 6 and the corresponding relative risks are shown in table 7. Perhaps the most striking finding from these tables is, once again, the remarkable extent of agreement for two arbitrary selections of 18 countries, with some overlap, concerning the detailed average pattern of relative risks. The confirmation of a progressive worsening of risk of death before age 5 with shorter intervals for a later period is valuable.

A DIGRESSION ON DATA QUALITY

One of the regularly recurring concerns of most analysts of data on retrospective events is the quality of date reporting. It is often supposed that dates of birth for dead children are less reliably reported than are dates for surviving children. The recode files produced for DHS have a major advantage in this respect over those produced for WFS. For each

TABLE 5. COMPARISON OF DATA FROM WORLD FERTILITY SURVEY AND DEMOGRAPHIC AND HEALTH SURVEYS ON SURVIVAL CHANCES TO AGE 5 AND BIRTH DISTRIBUTION IN 8 COUNTRIES, BY LENGTH OF PRECEDING BIRTH INTERVAL

Country	Deaths[a] (Preceding interval[b])					Relative risk[c] (Preceding interval[b])					Birth distribution[a] (Preceding interval[b])					Population attributable risk
	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+	
Colombia																
WFS	141	111	79	53	80	179	140	100	67	101	347	232	337	60	24	135
DHS	102	68	51	33	25	200	132	100	64	48	231	198	375	121	75	121
Peru																
WFS	243	185	138	72	92	176	134	100	52	67	265	214	422	69	29	123
DHS	201	148	103	63	30	195	144	100	61	29	216	191	444	92	57	121
Trinidad and Tobago																
WFS	71	46	32	24	56	222	143	100	75	175	329	219	332	78	42	151
DHS	60	29	28	36	24	216	105	100	128	85	238	181	341	127	112	130
Kenya																
WFS	224	151	119	84	80	188	127	100	71	67	194	234	492	60	21	121
DHS	156	97	80	51	36	195	122	100	64	45	141	197	537	89	36	113
Senegal																
WFS	356	224	269	252	188	133	83	100	94	70	66	155	686	68	25	99
DHS	287	240	212	141	150	135	113	100	66	71	90	174	636	75	24	102
Morocco																
WFS	249	165	110	73	40	227	150	100	66	36	235	216	462	62	25	137
DHS	220	127	102	53	56	216	125	100	52	55	182	190	513	81	34	120
Sri Lanka																
WFS	112	80	72	59	54	155	111	100	82	75	179	207	487	91	35	109
DHS	76	50	38	36	46	198	131	100	92	121	132	174	492	129	73	119
Thailand																
WFS	196	107	85	49	118	231	125	100	58	139	205	195	509	68	23	130
DHS	117	51	46	38	44	253	112	100	82	96	147	162	455	144	92	121
Mean																
WFS	199	133	113	83	88	189	127	100	71	91	228	209	466	70	28	124
DHS	152	101	82	56	51	201	123	100	76	69	172	183	474	107	63	117

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.
NOTE: 15 years before DHS.
[a]Per 1,000.
[b]Months.
[c]24-47 = 100.

TABLE 6. DISTRIBUTION OF BIRTHS (PER 1,000) BY LENGTH OF PRECEDING BIRTH INTERVAL

Country	Total births	Preceding interval (months)									
		Under 12	12-14	15-17	18-20	21-23	24-35	36-47	48-59	60-71	72+
Brazil	6 699	59	120	116	96	83	238	124	60	37	66
Colombia	5 589	39	91	101	101	97	251	124	78	42	75
Dominican Republic	9 325	61	108	93	92	95	273	127	62	31	58
Ecuador	6 450	68	88	78	87	92	289	128	71	37	64
Peru	7 419	52	82	82	92	99	306	138	60	32	57
Trinidad and Tobago	3 609	36	102	100	94	87	215	126	80	47	112
Burundi	7 556	24	37	42	55	122	419	186	65	24	27
Ghana	7 899	17	32	40	56	80	367	227	93	40	47
Kenya	16 235	37	51	53	79	118	389	148	63	26	36
Liberia	9 173	32	65	74	82	106	344	143	66	37	51
Mali	7 613	70	58	67	83	94	327	159	66	30	45
Morocco	14 428	48	72	63	78	112	374	139	54	27	34
Senegal	8 745	19	29	41	54	120	459	177	51	24	24
Tunisia	9 497	48	90	77	81	107	334	137	61	30	34
Zimbabwe	7 039	21	32	37	62	92	421	182	74	38	41
Indonesia	20 573	34	52	56	72	90	328	173	83	45	67
Sri Lanka	8 234	18	51	64	81	93	326	167	84	45	73
Thailand	8 170	33	54	59	77	84	292	163	90	54	92

Source: Demographic and Health Surveys standard recode files.
NOTE: 15 years before DHS.

recorded date of birth of a child, an indicator variable shows how the date was reported (e.g., year and month, year only, age only, year and age, season and year, and no information). From this information on the nature of the report used and the imputed date of the event, it is possible to recover the range within which imputation took place, where the month and year were not both reported. For each birth included in the present analysis, a minimum date and a maximum date from this information were created. Given these estimates, the maximum and minimum possible interval between each pair of births can also be derived. If the maximum possible interval is less than 24 months, the interval is treated as being "firmly" assigned to a short interval; if the minimum possible interbirth interval equals or exceeds 24 months, the interval is treated as being "firmly" assigned to a long interval; for all remaining intervals, the possible range spans the 24-month threshold and these intervals are referred to as being "fuzzily" dated.

Table 8 provides basic information on the extent of fuzzy or imprecisely determined intervals, simply trying to distinguish long and short intervals. The proportions of births per 1,000 where the assignment to long or short intervals is fuzzy are quite small for many of the countries considered here. Moreover, the fuzzy intervals are usually concentrated among the two categories close to the critical 24-month boundary. For

TABLE 7. RELATIVE RISK OF DEATH BY AGE 5, BY LENGTH OF PRECEDING BIRTH INTERVAL

Country	Rate after 24-27-month interval	Relative risk[a] (Preceding interval[b])									
		Under 12	12-14	15-17	18-20	21-23	24-35	36-47	48-59	60-71	72+
Brazil	72	420	233	202	145	148	114	71	101	87	53
Colombia	51	307	210	149	137	128	105	89	73	47	48
Dominican Republic	73	291	189	135	142	98	98	104	78	67	123
Ecuador	82	295	215	153	128	102	107	83	69	46	56
Peru	103	258	188	162	149	139	111	75	70	45	29
Trinidad and Tobago	28	484	245	89	118	91	102	97	88	194	85
Burundi	176	198	163	157	160	112	106	87	61	43	45
Ghana	145	185	130	137	155	111	97	105	87	72	74
Kenya	80	214	179	199	146	105	109	76	72	44	45
Liberia	207	161	155	141	141	115	109	76	77	50	42
Mali	235	230	197	170	138	134	104	91	63	57	57
Morocco	102	288	212	165	138	115	105	87	60	35	55
Senegal	212	129	137	137	123	108	104	89	72	53	71
Tunisia	55	462	247	197	168	137	104	90	67	53	62
Zimbabwe	71	349	184	224	113	173	105	88	72	73	90
Indonesia	105	236	190	158	115	133	109	83	66	69	71
Sri Lanka	38	404	178	153	103	155	109	82	105	69	121
Thailand	46	443	210	187	124	100	101	98	81	83	96
Mean		297	192	162	136	123	106	87	76	66	68
18 WFS countries		295	193	166	141	121	104	90	73		83

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS.

[a]24-27 = 100.

[b]Months.

example, in Morocco, 12.8 per cent of birth intervals assigned to the category 18-23 months on the basis of the imputed dates are actually of uncertain status with regard to being short or long when the imputation ranges are taken into account. Similarly, 10.8 per cent of the birth intervals assigned to the category 24-47 months in Morocco on the basis of the imputed dates could be shorter than two years. But much lower proportions of births assigned to the intervals which are not adjacent to the two-year threshold are doubtfully assigned to short or long intervals. Thus, in Morocco, 98.4 per cent of births assigned to preceding birth intervals of 1-17 months are almost certainly short intervals, in the sense that their range of possible dates does not allow the interval to be longer than two years.

Taken over all interval categories, 42 per cent of all assigned interval lengths in Mali are of uncertain or fuzzy classification, with respect to the two-year divide. The imputation procedures used for Mali were, in fact, extreme. Sullivan, Rutstein and Bicego (1989) indicate that information was missing on the month of birth for 58 per cent of surviving births. For

TABLE 8. PROPORTIONS OF BIRTHS AND DEATHS (PER 1,000) IN IMPRECISE INTERVALS SINCE PREVIOUS BIRTH

Country	Proportion of births					Proportion of deaths					Ratio: deaths/births				
	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+
	(Preceding interval[a])					(Preceding interval[a])					(Preceding interval[a])				
Brazil	9	23	13	1	1	28	144	87	12	0	3.1	6.2	6.6	15.3	0.0
Colombia	1	18	5	0	0	0	138	49	0	0	0.0	7.5	9.6		
Dominican Republic	2	20	9	0	0	7	74	35	0	0	4.9	3.7	4.0		
Ecuador	10	45	25	1	2	19	98	98	31	0	1.9	2.2	3.9	21.3	0.0
Peru	7	18	9	0	0	22	65	44	0	0	3.2	3.7	4.7		
Trinidad and Tobago	2	5	2	7	5	19	158	30	63	0	8.3	34.4	18.4	9.6	0.0
Burundi	51	206	91	4	1	61	337	232	29	14	1.2	1.6	2.5	7.0	14.3
Ghana	202	306	97	5	3	254	350	221	9	26	1.3	1.1	2.3	1.9	9.9
Kenya	15	31	13	6	8	31	85	53	58	76	2.0	2.7	4.1	9.5	9.5
Liberia	43	133	79	18	18	58	200	173	59	121	1.4	1.5	2.2	3.2	6.6
Mali	451	646	399	141	208	392	610	470	428	627	0.9	0.9	1.2	3.0	3.0
Morocco	16	128	108	6	16	30	198	157	54	154	1.8	1.5	1.4	9.0	9.4
Senegal	85	242	103	5	14	93	350	245	13	37	1.1	1.4	2.4	2.8	2.6
Tunisia	37	43	25	10	27	104	172	187	74	364	2.8	4.0	7.3	7.1	13.2
Zimbabwe	8	3	2	1	7	19	9	10	0	0	2.4	3.2	4.9	0.0	0.0
Indonesia	37	200	85	1	0	52	359	222	5	0	1.4	1.8	2.6	5.4	
Sri Lanka	41	73	32	8	10	116	235	151	176	56	2.9	3.2	4.8	21.7	5.7
Thailand	112	84	50	27	34	284	531	397	312	333	2.5	6.3	7.9	11.4	9.9
Mean	63	124	64	13	20	88	228	159	73	100	2.4	4.8	5.0	8.6	6.0

Source: Demographic and Health Surveys standard recode files.
NOTE: 15 years before DHS.
[a]Months.

Mali, the combination of age and year of birth was never used in these cases, and imputation alternated between using one or the other, with each implying a full year of range of imputation.

There is also considerable imprecision in the simple categorization of preceding birth intervals into short and long intervals (a division at two years) for several other countries, with about 12 per cent of intervals being "fuzzy" for Ghana and Senegal, 10 per cent for Burundi, 8 per cent for Morocco and Indonesia, 7 per cent for Liberia, and 6 per cent for Thailand; levels of imprecision were much lower elsewhere, at 3.5 per cent or less. Over 20 per cent of births classified as being at intervals of 18-23 months, according to the imputed dates, might be at intervals longer than two years in Mali, Ghana, Senegal, Burundi and Indonesia, when the imputation ranges are taken into account. This proportion also exceeds 10 per cent in Liberia and Morocco. For a handful of countries, significant percentages of births recorded as being less than 18 months after the preceding birth, on the basis of imputed dates, are of uncertain status with respect to being at intervals definitively shorter than two years: Mali (45), Ghana (20), Thailand (11) and Senegal (8). Significant fractions of the preceding birth intervals which would normally be assigned to the 24-47-month (reference) category might conversely be below the two-year threshold; Mali is again the extreme case, and the percentage ranges from 8 to 11 for Liberia, Indonesia, Burundi, Ghana, Senegal and Morocco. At the other extreme, the degree of apparent uncertainty about assignment to short or long intervals is strikingly (and probably surprisingly) low for Zimbabwe.

While the results considered so far, on the extent of imprecision in classification of births, are sufficient to cause concern, the extent of imprecision in the dating of births of deceased children is much greater (table 8). Over 20 per cent of all dead children cannot be unequivocally assigned to short or long intervals for Mali, Thailand, Ghana, Senegal and Burundi; 12 per cent or more of dead children are also potentially misclassified in Indonesia, Sri Lanka, Tunisia, Liberia and Morocco. The proportion is 6 per cent or less in the remaining eight countries.

The final panel of table 8 serves to emphasize some of the worst fears with respect to data quality: dates of birth and preceding intervals are much more likely to be imprecise for deceased children than for those still alive.

How damaging are these findings for the study of associations between length of preceding interval and child survival? Perhaps surprisingly, table 9 shows that these very strong associations between imprecise dating and child survival have very little impact upon the estimated patterns of relative risk. The relative risks of child death are shown for an analysis based only on firmly dated preceding intervals (those which could unequivocally be assigned to one side or the other of a boundary at two years), and for the analysis based on all intervals. Clearly, the selective association of fuzzy dating with child deaths will mean that the estimated *mortality rates* based only on the firmly dated intervals will be too low.

TABLE 9. RELATIVE RISKS OF DEATH BY AGE 5 FOR FIRMLY DATED AND FOR ALL PRECEDING INTERVALS, BY GROUPED LENGTH OF INTERVAL

Country	Firmly dated intervals (Preceding interval[a])					All intervals (Preceding interval[a])					Ratio: all/firmly dated (percentage) (Preceding interval[a])				
	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+	1-17	18-23	24-47	48-71	72+
Brazil	274	139	100	102	57	259	146	100	96	53	94	105	100	93	92
Colombia	210	122	100	67	51	200	132	100	64	48	95	109	100	96	96
Dominican Republic	199	116	100	77	126	195	120	100	75	123	98	103	100	97	97
Ecuador	233	117	100	64	61	218	114	100	61	56	93	97	100	95	92
Peru	200	142	100	64	30	195	144	100	61	29	98	101	100	96	96
Trinidad and Tobago	218	91	100	124	87	216	105	100	128	85	99	115	100	103	97
Burundi	193	125	100	63	52	168	127	100	56	45	87	101	100	89	87
Ghana	155	140	100	94	82	144	129	100	83	74	93	92	100	88	90
Kenya	200	120	100	64	44	195	122	100	64	45	97	102	100	101	103
Liberia	164	130	100	71	43	150	126	100	67	42	91	97	100	94	100
Mali	239	168	100	42	29	200	136	100	61	57	84	81	100	144	195
Morocco	224	121	100	52	50	216	125	100	52	55	97	103	100	100	111
Senegal	157	116	100	77	80	135	113	100	66	71	86	97	100	86	88
Tunisia	310	156	100	69	48	278	150	100	62	62	90	96	100	89	129
Zimbabwe	238	149	100	73	91	238	149	100	72	90	100	100	100	99	98
Indonesia	217	118	100	78	83	188	125	100	67	71	87	106	100	86	86
Sri Lanka	207	124	100	88	131	198	131	100	92	121	95	106	100	105	92
Thailand	321	89	100	90	106	253	112	100	82	96	79	125	100	90	91
Mean	220	127	100	75	70	203	128	100	73	68	92	102	100	97	102

Source: Demographic and Health Surveys standard recode files.
NOTE: 15 years before DHS.
[a]Months.

However, the *relative risks* of child death by length of the preceding interval seem remarkably robust. Indeed, the likely inferences concerning the broad patterns of relative risks would only change in one country, Thailand, where the already weak excess mortality for children born 18-23 months after the previous birth disappears. That Thailand should be most affected by this restriction to firmly dated intervals should come as no surprise, since dead children are about six times more likely to be removed from the analysis than survivors (37 per cent of dead children are removed from the analysis, and only 6 per cent of all births). Otherwise, there is a slight and very consistent tendency for the relative risk for the 1-17-month interval category to rise with the restriction to firmly dated intervals (perhaps an artefact of the concentration on the division at two years for defining fuzzy intervals, which leads to a greater proportionate removal from the 18-23-month and 24-47-month categories in most countries).

The scope for empirical examination of imprecise dating which the ability to recover the imputation range for all dates reported in the DHS allows is important. The limited use of the information here suggests that findings on relative risks of mortality by length of the preceding birth interval are surprisingly robust, even to fairly extensive imputation. To the best of our knowledge, this is the first empirical confirmation of the simulation results presented by Trussell (1987), which also suggested such robustness to selective imprecision of dating by child survival status. Of course, machine imputation of dates may only be the tip of the iceberg. DHS files, unsurprisingly, are mute concerning the extent of bargaining between the respondent and the interviewer and the amount of field imputation of dates.

AN ANALYSIS BY "FAMILY FORMATION" PATTERN

Having examined patterns of child-bearing by age of the mother and by length of the preceding interval separately, we now try to put the whole picture together, using the 10 family-formation patterns described earlier. We can now separate out first births, teenage births and poorly spaced births; moreover, any combination of these categorizations can be identified. Furthermore, the classification includes a crude attempt to distinguish the concentration of all previous births for mothers aged 20-34, by classifying women into pace of previous reproduction groups. This classification proved useful in our earlier, related analysis (Hobcraft, 1987) and has been found helpful in multivariate analysis for Mexico (Bobadilla, Schlaepfer and Alagon, 1990).

Table 10 provides the distribution of births across these 10 categories for each of the 18 DHS countries considered. The overall proportion of first births is obviously strongly inversely correlated with the fertility level, being highest (at about one third of all births to women under age 35 during the 15-year period covered) in Thailand, Trinidad and Tobago and Sri Lanka. First births constitute only one fifth of the total sample for Kenya, Mali and Morocco. There is striking variability in the fractions of

TABLE 10. DISTRIBUTION OF BIRTHS (PER 1,000) TO WOMEN AGED UNDER 35 IN 10 FAMILY-FORMATION CATEGORIES

			Later births							
	First births		Well-spaced				Poorly spaced			
Country	Teen	20-34	Teen	Slow	Medium	Fast	Teen	Slow	Medium	Fast
Brazil	108	197	14	192	93	58	39	112	84	105
Colombia	134	175	20	183	111	68	45	99	80	84
Dominican Republic	147	126	30	152	114	94	57	83	90	107
Ecuador	129	137	23	177	139	80	44	95	81	95
Peru	109	135	21	167	146	97	37	99	92	97
Trinidad and Tobago	136	190	15	212	105	44	50	108	77	64
Burundi	87	149	13	246	214	65	15	83	82	45
Ghana	142	109	32	235	223	84	23	44	61	46
Kenya	137	62	49	129	195	147	41	48	82	110
Liberia	176	91	54	158	148	102	52	55	73	92
Mali	137	72	48	137	166	133	57	54	87	109
Morocco	92	119	19	150	180	125	35	90	95	94
Senegal	154	74	55	157	210	136	37	40	66	71
Tunisia	53	185	2	203	166	64	13	152	101	60
Zimbabwe	149	99	38	198	218	107	26	47	64	53
Indonesia	144	120	36	205	176	87	34	69	69	62
Sri Lanka	75	249	11	275	128	39	15	126	58	25
Thailand	124	226	13	261	128	35	21	111	56	25
Mean 18 DHS	124	140	27	191	159	87	36	84	78	75
Mean 18 WFS	118	106	31	152	157	107	46	78	91	115

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS.

first births which occur to teenage mothers: about two thirds for Kenya, Liberia, Mali and Senegal; and under one fourth for Tunisia and Sri Lanka (in our previous study, using WFS data, these fractions ranged from 88 per cent for Bangladesh down to only 9 per cent for Korea).

The overall fractions of poorly spaced births also vary considerably, being below 20 per cent in Ghana and Zimbabwe, and about one third for Brazil, the Dominican Republic, Peru and Tunisia. Well-spaced later births, the remaining broad category, constitute a large proportion of all births. Over half of the births fall into this category for Burundi, Ghana, Indonesia, Kenya, Senegal and Zimbabwe. Fewer than 40 per cent of all births to women under age 35 in Brazil, Colombia, the Dominican Republic and Trinidad and Tobago are well-spaced later births.

It is sometimes regarded as desirable for all births to be to 20-34-year-old mothers and for none of them to be poorly spaced. There would be a long way to go towards achieving this target in any of the countries examined here. By this criterion, Sri Lanka has the best family-formation pattern, with just over two thirds of all births before age 35 being in this advantaged category. Fewer than half of all births occurring in the Dominican Republic and Liberia meet this criterion.

The fractions of women who are classified as fast reproducers at the time of a birth is determined by a combination of the timing of the initiation of child-bearing and the overall fertility level. As might be expected, short preceding intervals are much more common among this group and even more so for teenage later births (perhaps the ultimate fast reproducers).

These family-formation patterns can also be examined for the eight countries where we have results from both WFS and DHS. Table 11 shows the distributions of births across the 10 categories. Despite the fact that the periods of observation overlap, often considerably, the changes in family-formation patterns are striking. Without exception, the fraction of first births has increased, although the change is small for Kenya, Morocco and Senegal, and only moderate for Peru.

TABLE 11. COMPARISON OF DATA FROM WORLD FERTILITY SURVEY AND DEMOGRAPHIC AND HEALTH SURVEYS ON DISTRIBUTION OF BIRTHS (PER 1,000) TO WOMEN AGED UNDER 35 IN 10 FAMILY-FORMATION CATEGORIES

			Later births							
	First births		*Well-spaced*				*Poorly spaced*			
Country	*Teen*	*20-34*	*Teen*	*Slow*	*Medium*	*Fast*	*Teen*	*Slow*	*Medium*	*Fast*
Colombia										
WFS	113	111	21	107	99	99	59	102	116	172
DHS	134	175	20	183	111	68	45	99	80	84
Peru										
WFS	98	109	19	143	145	105	40	101	112	127
DHS	109	135	21	167	146	97	37	99	92	97
Trinidad and Tobago										
WFS	117	142	18	132	97	88	56	100	100	151
DHS	136	190	15	212	105	44	50	108	77	64
Kenya										
WFS	134	62	46	111	166	137	53	62	102	127
DHS	137	62	49	129	195	147	41	48	82	110
Senegal										
WFS	156	51	60	138	261	159	33	26	51	67
DHS	154	74	55	157	210	136	37	40	66	71
Morocco										
WFS	105	83	25	105	159	156	59	68	100	140
DHS	92	119	19	150	180	125	35	90	95	94
Sri Lanka										
WFS	84	140	24	173	168	111	33	96	86	86
DHS	75	249	11	275	128	39	15	126	58	25
Thailand										
WFS	92	139	11	191	188	71	24	108	105	71
DHS	124	226	13	261	128	35	21	111	56	25
Mean										
WFS	112	105	28	138	160	116	45	83	97	118
DHS	120	154	26	192	150	86	35	90	76	71

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS and WHS.

The four countries with big changes in fractions of first births to mothers aged under 35 years, and thus in levels of fertility, are Thailand (23-35 per cent of the births, an increase of 12 percentage points, or about 50 per cent), Sri Lanka, Colombia and Trinidad and Tobago. Each of these four countries also shows a big reduction in the proportion of births which are poorly spaced (8-14 percentage points, or about 30 per cent reductions). The extent of fertility decline is also evidenced by the shifts in the distribution of later births across the pace of reproduction categories: the fractions of later births to 20-34-year-olds which are to slow reproducers go up by 15-24 percentage points; the proportions attributed to fast reproducers decline by 9-18 percentage points. In general, then, these changes in family-formation patterns for the four countries with the largest changes are towards patterns which would be expected to be more favourable to child survival. The only minor exception is that the proportion of births which are teenage first births has gone up (by about 2 percentage points) in all but Sri Lanka.

Changes in Senegal are mainly in the opposite direction, although neither the overall proportion of first births nor the distribution of later births across the pace of reproduction categories has changed much, suggesting that fertility levels have barely altered. But the fraction of births which are poorly spaced has risen noticeably (by 4 percentage points, or 21 per cent), suggesting that traditional post-partum restraints on spacing of births are eroding.

Having examined the family-formation patterns themselves, we now turn to the relative risks of child death associated with these categories. Table 12 shows them, using well-spaced births to 20-34-year-old mothers as the reference category.

Starting with first births, there is clear evidence of excess risks associated with teenage motherhood everywhere. Taken overall, the average excess risk for teenage first births is 46 per cent. For first births to 20-34-year-old mothers there is a small excess risk compared to well-spaced births for the same age group. This latter excess is larger for Kenya, Mali and Tunisia, at about 30 per cent, and there is an apparent clear relative advantage for first births to 20-34-year-old mothers in Colombia, the Dominican Republic and Peru.

For well-spaced later births to 20-34-year-olds, the relative risks must average 100. For teenage mothers the sample sizes are usually small, and the estimated relative risks need to be treated with caution since they are typically subject to large sampling variability. Nevertheless, an average excess risk of about one third (over all 18 countries) emerges for this group. For the well-spaced births to 20-34-year-olds, some differences do emerge. Relative risks are usually a little lower for the slow reproducers (8 per cent, on average). The fast reproducers show evidence of excess risk, with the average excess being 24 per cent, and several countries showing bigger excess risks. The gradient across the pace of reproduction categories for well-spaced later births to 20-34-year-old mothers is quite clear and sizeable for Brazil, Colombia, Ecuador and Peru; perhaps such

TABLE 12. RELATIVE RISKS OF DEATH BY AGE 5 FOR 10 FAMILY-FORMATION CATEGORIES

Country	Well-spaced rate, 20-34-year-old mothers[a]	First births		Well-spaced				Poorly spaced				Population attributable risk
		Teen	20-34	Teen	Slow	Medium	Fast	Teen	Slow	Medium	Fast	
Brazil	61	173	96	245	86	104	137	313	176	227	337	161
Colombia	46	128	77	106	68	105	172	255	132	161	235	126
Dominican Republic	68	148	88	107	109	104	82	211	119	135	226	130
Ecuador	72	138	107	74	88	104	119	213	128	218	217	134
Peru	86	145	75	143	79	99	137	226	120	246	229	135
Trinidad and Tobago	26	162	117	205	96	90	140	282	139	163	172	136
Burundi	166	134	104	125	98	95	123	161	150	157	141	115
Ghana	132	135	108	154	94	101	114	142	172	141	146	116
Kenya	70	154	127	153	96	91	116	199	96	156	203	132
Liberia	184	150	105	133	98	90	118	173	134	159	152	126
Mali	208	164	128	136	93	103	104	216	167	185	197	141
Morocco	95	168	114	108	96	101	104	240	154	166	201	134
Senegal	203	120	101	117	97	101	102	136	100	120	144	110
Tunisia	51	158	129	82	92	101	119	283	167	229	360	150
Zimbabwe	65	149	114	148	91	98	120	225	146	172	256	129
Indonesia	94	136	98	134	95	92	125	235	124	161	198	122
Sri Lanka	37	126	107	149	95	107	103	163	117	228	279	119
Thailand	39	149	105	109	90	94	203	241	164	223	322	130
Mean 18 DHS		146	106	135	92	99	124	217	139	180	223	131
Mean 18 WFS		150	108	126	93	100	113	203	135	171	208	134

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS.

[a]Well-spaced births to 20-34-year-old mothers are baseline group (= 100).

differences in the Latin American countries arise from very high socio-economic inequalities, which also coincide with the current differentials in pace of reproduction (at an intermediate stage of the demographic transition). This finding should once again caution against the lack of socio-economic controls in this analysis.

Poorly spaced later births typically experience considerable excess risks of death before age 5. Even for slow reproducers there is a 35 per cent average excess risk for poorly spaced births, compared with all well-spaced births to 20-34-year-old mothers, or a 51 per cent excess compared with well-spaced births for slow reproducers. For those poorly spaced later births which occur to women with a medium pace of reproduction, the average excess risk is 80 per cent. If the relative risk for poorly spaced births to fast reproducers is compared to that for well-spaced births to fast reproducers, the excess is also 80 per cent; compared with all well-spaced later births to 20-34-year-olds the average relative risk is 223, an excess of 123 per cent. Poorly spaced teenage births also experience high risks of death, being more than twice as likely on average to die before age 5 than children born to women in the reference group; compared with teenage well-spaced births, the average excess risk is 61 per cent.

Brazil stands out as having extraordinarily high excess risks, both for teenage and for poorly spaced births. Tunisia and Thailand also show extreme excess risks for poorly spaced births. Excess risks are small in Senegal, and generally a little lower in the other high-mortality countries (Burundi, Ghana and Liberia), although Mali (perhaps because of its severe dating problems) is an exception. A 40 per cent excess risk leads to many excess deaths when applied to a baseline rate of about 200 per 1,000, raising the mortality by 80 per 1,000. It is thus not surprising that the relative risks are a little more modest in such circumstances. Only Mali provides a puzzle in this respect, with much higher excess risks and the highest baseline mortality rate.

The comparison of mean values for the relative risks derived from 18 WFS countries and 18 DHS countries shows very broad agreement. If anything, the excess risks associated with short birth intervals are a little higher for the more recent DHS.

Implications at the population level

The combined impact of the family-formation patterns and their associated risks is summarized by the population attributable risks given in table 12. Brazil and Tunisia have severe problems, with combinations of high relative risks and unfavourable family-formation patterns leading to major excess child mortality (population attributable risks are 161 and 150, respectively). Experience elsewhere suggests that these might be reduced to around 130 if all or most of the excess is indeed attributable to the factors considered here. Better family planning might lead to a 30 per cent reduction in child mortality for Brazil, and possibly more, although very likely less.

The combination of low relative risks and fairly favourable family-formation patterns for Burundi, Ghana and Senegal results in quite low population attributable risks (PARs). This contrasts with Mali, where the PAR is 141, and overall child mortality by age 5 is 295 per 1,000. If the results for Mali were correct, and the PAR could be reduced to that of Ghana or Burundi, the overall child mortality rate might conceivably be reduced to about 240 per 1,000; this would require both a significant reduction in the fraction of births which are poorly spaced and in the relative risks, implying the need for both family planning and health interventions. Of course, it is highly questionable to pursue this example too far in view of the data-quality problems observed earlier for Mali.

A further concern, which is more likely to be of importance, arises from the following scenario. With the onset of fertility transition in Burundi, Ghana or Senegal, family-formation patterns may well become less favourable for child survival; the erosion of traditional post-partum fertility constraints is the concern here. As a result, more births could well occur at short intervals, with a possible concomitant rise in fertility (see Dyson and Murphy, 1985). Moreover, as we have already indicated, the relative risks for these high-mortality countries are unusually low. As

overall levels of mortality are reduced, it seems quite plausible that these relative risks will rise (the scope for differential survival being greater at moderate levels of child mortality). It thus seems highly likely that population attributable risks associated with family-formation patterns are set to rise in these societies over the next 10-20 years and thus make overall mortality gains harder to achieve. There is thus a need for family planning programmes in these countries to emphasize child-spacing.

With these arguments in mind, we now turn to a comparison of results for WFS and DHS, and begin by focusing on results for Senegal (see table 13). The child mortality rate for the baseline group in Senegal has declined from 261 per 1,000 to 203, a significant gain. But the relative risks for the typically higher-risk groups have increased. We had already

TABLE 13. COMPARISON OF DATA FROM WORLD FERTILITY SURVEY AND DEMOGRAPHIC AND HEALTH SURVEYS ON RELATIVE RISKS OF DEATH BY AGE 5 FOR 10 FAMILY-FORMATION CATEGORIES

Country	Well-spaced rate, 20-34-year-old mothers[a]	First births		Well-spaced[a]				Poorly spaced				Population attributable risk
		Teen	20-34	Teen	Slow	Medium	Fast	Teen	Slow	Medium	Fast	
Colombia												
WFS	70	135	97	174	115	91	92	238	153	156	202	143
DHS	46	128	77	106	68	105	172	255	132	161	235	126
Peru												
WFS	127	122	76	102	85	96	126	183	141	166	204	128
DHS	86	145	75	143	79	99	137	226	120	246	229	135
Trinidad and Tobago												
WFS	34	148	99	102	80	103	127	165	92	153	264	139
DHS	26	162	117	205	96	90	140	282	139	163	172	136
Kenya												
WFS	110	159	127	124	89	96	114	198	131	161	171	133
DHS	70	154	127	153	96	91	116	199	96	156	203	132
Senegal												
WFS	261	107	79	104	94	103	100	123	66	102	103	101
DHS	203	120	101	117	97	101	102	136	100	120	144	110
Morocco												
WFS	102	176	112	97	110	84	110	233	151	194	212	145
DHS	95	168	114	108	96	101	104	240	154	166	201	134
Sri Lanka												
WFS	70	139	84	114	95	94	118	169	94	141	167	113
DHS	37	126	107	149	95	107	103	163	117	228	279	119
Thailand												
WFS	75	132	118	120	105	95	102	228	129	208	222	132
DHS	39	149	105	109	90	94	203	241	164	223	322	130
Mean												
WFS		140	99	117	97	95	111	192	120	160	193	127
DHS		144	103	136	90	99	134	218	128	183	223	129

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: 15 years before DHS and WFS.

[a]Well-spaced births to 20-34-year-old mothers are baseline group (= 100).

noted a worsening of child-spacing for Senegal over time. The combined effect of the worsening family formation patterns and relative risks associated with them is to raise the population attributable risk from 101 to 110. The actual overall gain in child survival over the mere eight years between the surveys (and with some seven years of experience in common) is from 263 per 1,000 to 223 per 1,000, a 15 per cent reduction (see table 14). But mortality for well-spaced births to 20-34-year-old mothers was reduced from 261 per 1,000 to 203 per 1,000, a 22 per cent reduction. Without the negative impact of family-formation differences, the overall mortality rate might possibly have been reduced by a further 20 per 1,000, or a 50 per cent increase in the mortality gain. Such considerations are clearly not of negligible importance. Of course, we urge some caution over these results, since the assumptions involved in these illustrative calculations are strong: the estimates from both surveys are taken as accurate, or at least similarly deficient; all changes in relative risks and all excess mortality observed for family-formation groups are taken to be caused solely by the child-bearing patterns and not by associated variables.

Table 13 also shows comparisons for the other seven countries. Population attributable risks have also increased modestly over time for Peru and Sri Lanka. Yet the distribution of births across the family-formation categories in both these countries had become more favourable, slightly so for Peru and significantly so for Sri Lanka. Closer examination of the patterns responsible for these changes does not provide a coherent story. For Sri Lanka, the overall rise in PAR is mainly due to a worsening of relative risks for two categories which gained a considerable fraction of births: the relative risks for 20-34-year-old first births and for poorly spaced births to slow reproducers both rose from noticeably low levels to more normal ones. For Peru, the increase in the fraction of first births to teenage moth-

TABLE 14. ACTUAL CHANGES IN MORTALITY RATES UNDER AGE 5 AND COMPONENTS OF CHANGE

Country	Observed mortality rate		Ratio of mortality rates		Ratio of population attributable risks (percentage)	Percentage change due to changes in:			
	WFS	DHS	All births (percentage)	Well-spaced births (percentage)		Fertility structure (using DHS relative risks)	Relative risk (using WFS structure)	Fertility structure (using WFS relative risks)	Relative risk (using DHS structure)
Colombia	100	58	58	66	88	85	104	92	96
Peru	162	116	72	68	106	94	112	96	111
Trinidad and Tobago.....	47	35	75	76	98	94	105	87	113
Kenya.....	146	92	63	63	99	97	102	97	102
Senegal	263	223	85	78	109	100	109	99	111
Morocco	148	127	86	93	92	94	97	94	98
Sri Lanka	79	44	55	53	105	88	120	91	117
Thailand.....	99	50	51	52	99	88	113	94	105
Mean.....			68	69	100	93	108	94	106

Sources: Demographic and Health Surveys and World Fertility Survey standard recode files.

NOTE: Comparison for 15 years before DHS and WFS.

ers was combined with a slightly higher relative risk, and the relative risk for poorly spaced births to medium-paced reproducers unaccountably rose. We doubt that these slightly quirky changes are of real significance.

The largest change in population attributable risks between the WFS and DHS over the 10 family-formation categories occurs for Colombia. Over an effective 10-year period, the child mortality rate for the well-spaced births to 20-34-year-old mothers went from 70 per 1,000 to 46 per 1,000, a reduction of 34 per cent. But overall child mortality (for the entire samples considered here) fell from 100 per 1,000 to 58 per 1,000, a reduction of 42 per cent. If there had been no gains in child mortality associated with family-formation patterns, the overall rate might have been reduced to only 66 per 1,000. The extra reduction corresponds to about a quarter of the observed gain, and might be attributed mainly to improvements in the family-formation pattern and perhaps thus due to family planning. Once again, we urge caution in such interpretation.

One other curious feature of the changes for Colombia is the alteration in the pattern of relative risks across the reproductive-pace categories for well-spaced births. For the 15 years preceding the WFS programme, there appears to be little differentiation in these relative risks. But the DHS data show a massive differential by reproductive pace for these well-spaced births.

Table 14 is a final attempt to examine the patterns of change over time for the eight countries where information was available from both WFS and DHS. The first two columns show the observed overall child mortality rates for the entire samples (restricted to births to women under age 35 and, in different ways for the two data sources, to experience covering the 16 years before the surveys). The next column shows the DHS rate as a percentage of the WFS rate and thus indicates the extent of overall decline in mortality. This can further be subdivided into two components: the percentage ratio of the change in the baseline rate for well-spaced births to 20-34-year-old mothers; and the percentage ratio of the risks attributable to population for the two periods. The product of these components is precisely the ratio of the overall rates.

Thus, we can observe that the 42 per cent overall decline in Colombia resulted from a 34 per cent decline for the baseline group, reinforced by a 12 per cent reduction in the population attributable risk. The 14 per cent overall decline in Morocco was about equally attributable to declines in the baseline rate and in the PAR, whereas the entire declines in Kenya and Thailand are attributable to across-the-board mortality changes and not associated with family-formation patterns.

Table 14 also shows two alternative partitions of the ratio of the PARs into a component due to changes in the structure and one due to alterations in risk patterns across the family-formation groups. In most cases the alternative partitions tell the same story (e.g., Kenya, Morocco, Peru and Sri Lanka). For Peru and Sri Lanka, both considered earlier, the rise in the PARs is clearly attributable to the changes in the patterns of relative risks, which can be seen to more than counterbalance gains from

improvements in the structures. As noted before, the structural gains in Sri Lanka were not negligible: on their own they would have accounted for about a 10 per cent overall reduction in child mortality.

These partitions tell a consistent story. With the exception of Senegal, where structural change was about neutral, there were improvements in the distribution in terms of the likely mortality impact for all countries, with the net effect being around a 10 per cent overall gain in child survival from structural change in Colombia, Trinidad and Tobago, Sri Lanka and Thailand. But in most places these structural gains were offset, partially or completely, by a worsening of the relative risk patterns. Only Morocco shows evidence of a small overall improvement in relative risk patterns by family-formation group; there is doubt for Colombia, depending upon which partition is used.

Conclusion

The detailed examination of results of DHS for 18 countries and extensive comparison with both summary results and matching countries from WFS have provided several opportunities to enhance our understanding of the links between child-bearing patterns and infant and child mortality. We have emphasized the complexity of the relationships involved and brought to bear some new empirical results on the impact of date imputation on our analyses.

The associations of early fertility (especially before age 18) and of short birth intervals with the chances of child survival are as strong 10 years on (using DHS data) as they were shown to be in earlier studies (based overwhelmingly on WFS). Patterns of relative risk associated with various child-bearing patterns have been shown to be broadly the same, although a few interesting differences over time do emerge.

The changing patterns of child-bearing over time have also been considered in some detail, along with the possible consequences for child mortality. Among the more provocative conclusions, the possibility that mortality in Senegal might have fallen by a further 50 per cent over the eight-year effective observation period, if only child-spacing and associated relative risks had not changed for the worse during that period, is worth highlighting. However, the caveats associated with that possible analysis must also be stressed again.

The very real possibilities of mortality gains in several sub-Saharan African countries being slowed down by worsening family-formation patterns and widening relative risks have also been discussed at some length.

The changes observed for Colombia suggest that evidence implying that family planning does indeed help save children's lives is beginning to become more clearly available. Such gains can never be huge, since the population attributable risks rarely exceed 40 per cent and are unlikely to get much lower than 15 per cent. Thus, the possible gains in child survival from improved, targeted use of family planning are always likely to be modest in the long term. Such gains are nevertheless worth realizing.

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SENSITIVITY OF AGGREGATE PERIOD LIFE EXPECTANCY TO DIFFERENT AVERAGING PROCEDURES

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SUMMARY

When aggregating the observed period life expectancies of smaller units (countries) into larger ones (regions or world), one can either merge the populations at risk and deaths according to age groups and recalculate life expectancy from the joint age-specific mortality rates, or one can simply calculate a weighted average of the individual life expectancies, where usually births are taken as the weights. The Population Division of the United Nations Secretariat recently switched from the second to the first procedure. This results in a life expectancy for the world total which is 2.5 years higher. For less heterogeneous aggregates, the difference is less. This article gives special attention to the fact that the joint life expectancy calculated by merging the populations may even be outside the range of life expectancies in the constituent populations. Extensive simulations are performed to estimate the empirical relevance of this seemingly paradoxical phenomenon, using various sets or model life-tables. It also shows how the phenomenon depends on the difference between the constituent life expectancies and on the age distributions of the populations concerned.

INTRODUCTION

In *World Population Prospects, 1988* (United Nations, 1989), the United Nations gave a life expectancy of 59.6 years for both sexes for the world total for the 1980-1985 period. The recent 1990 revision gives a figure of 62.1 years for the same period. This "gain" of 2.5 years in global life expectancy is not due to a revision of the empirical input data: life expectancies at the national level for the 1980-1985 period are identical in the 1988 and 1990 revisions. It is rather the consequence of a different way of aggregating national life expectancies into regional and global ones. In the 1988 revision, life expectancies were weighted by the number of births in each population. In the 1990 revision, populations were actually merged according to age groups, and the life expectancy of the joint population was calculated.

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Table 1 lists life expectancy figures for both sexes for the periods 1950-1955 and 1980-1985, as given by the 1988 and the 1990 revisions, using the different averaging approaches. We see that the difference between the two life expectancies is highest for the world total and already much lower when breaking the world into two groups of more developed and less developed countries. Looking at continents separately results in even lower differences between the two revisions. Over time, differences seem to increase at almost every level. Only in Europe do they slightly decrease and in Africa and Latin America they stay the same. Most striking is the increase in Oceania, where there is no difference between the two revisions for 1950-1955 but a difference of 2.1 years for 1980-1985.

As we will demonstrate below, the extent of the difference between the two methods of aggregation has to do with heterogeneity in the aggregated age structures together with different mortality patterns. Table 1 clearly indicates that the more homogeneous the aggregate considered, the smaller the difference. Hence, it is not surprising that the largest difference appears for the world total. In the case of Oceania, it is the increasing heterogeneity of more and less developed countries in the region that caused the strong increase in the difference.

More puzzling to common sense than the change in total life expectancy figures due to different methods of aggregation is the case of the Ukraine, where official data give female life expectancy of 75.1 years for urban areas and 75.0 years for rural areas. Female life expectancy of urban and rural areas together in the Ukraine, however, has a value of 75.2, which is higher than urban and rural areas taken separately. More extreme results appear when merging the Soviet republics of Azerbaijan (male period life expectancy in 1979 was 64.13 years) and Estonia (64.23

TABLE 1. PERIOD LIFE EXPECTANCIES FOR WORLD REGIONS IN 1950-1955 AND 1980-1985, ACCORDING TO THE 1988 AND 1990 UNITED NATIONS REVISIONS

	1950-1955			*1980-1985*		
	1988 revision	*1990 revision*	*Difference*	*1988 revision*	*1990 revision*	*Difference*
World total	45.9	47.5	1.6	59.6	62.1	2.5
More developed countries	65.7	66.0	0.3	72.3	72.8	0.5
Less developed countries	41.0	42.2	1.1	57.6	59.4	1.8
Africa	38.0	37.7	−0.3	49.9	49.6	−0.3
Latin America	51.2	51.9	0.7	64.5	65.2	0.7
Northern America	69.0	69.0	0.0	74.6	74.7	0.1
Asia	41.1	42.0	0.9	59.3	60.5	1.2
Europe	65.3	65.8	0.5	73.2	73.5	0.3
Oceania	60.8	60.8	0.0	68.0	70.1	2.1
USSR[a]	64.1	64.1	0.0	67.9	67.9	0.0

[a] USSR was not affected by changes in the aggregation method because it was considered one country.

Sources: *World Population Prospects, 1988* (United Nations publication, Sales No. E.88.XIII.7) and *World Population Prospects, 1990* (United Nations publication, Sales No. E.91.XIII.4).

years). When the two populations are merged into one by adding up deaths and populations at risk at each age, the period life-table for the joint population yields a life expectancy of 63.73. Hence, the joint life expectancy is about half a year lower than life expectancies in either Azerbaijan or Estonia. How can the joint period life expectancy in a population be lower or higher than the life expectancy in each of the subpopulations?

In the following we will consider this question and study the empirical relevance of this phenomenon.

Non-linear averaging functions

The paradox of aggregate life expectancy being higher or lower than life expectancies in all constituent populations is quite different from the well-known Simpson's paradox. An example of the latter is the rank order of fertility levels by linguistic groups in Canada: in every single province of Canada, the Francophones have higher fertility than the Anglophones, but in the whole country the Anglophones have higher fertility. This paradox can be understood intuitively as an aspect of weighting. Because of the concentration of French in Quebec, the Canadian figure for the French is mostly a reflection of the low Quebec fertility (Keyfitz, 1985). The Anglophones in Quebec, on the other hand, have even lower fertility than the French but they are so few that they have little influence on the national average, which mostly reflects the other predominantly Anglophone provinces with higher fertility levels.

In the case of averaging life expectancies, it is not the question of changing rank orders which leaves unconsidered the extent of the difference and the relative sizes of the populations. What may cause the seemingly paradoxical situation is the non-linearity of the averaging procedure applied when merging populations and calculating a new life-table. With certain sets of weights and rate schedules, any non-linear averaging function may result in a mean that lies outside the range of its components. This will be demonstrated below for the harmonic mean, one of the most simple non-linear averaging functions.

Table 2 shows two groups, A and B, distributed in a certain way over two regions, 1 and 2, assuming no migration between groups or regions. Each member of those groups is exposed to the risk of an event at the

TABLE 2. EXAMPLE OF A SIMPLE CASE WHERE THE WEIGHTED HARMONIC MEAN OF THE AGGREGATE LIES OUTSIDE THE RANGE OF THE TWO INDIVIDUAL MEANS

	Group A			*Group B*			
	Size	*Number of Events*	*Rate*	*Size*	*Number of Events*	*Rate*	*Weighted Harmonic mean*
Region 1	100	95	0.95	300	30	0.10	0.1288
Region 2	200	10	0.05	400	80	0.20	0.1000
Both regions	300	105	0.35	700	110	0.16	0.1911

intensity given by the rate. Hence, within region 1, the 100 members of group A experience the event at a rate of 0.95 while the 300 members of group B have a rate of only 0.1. The weighted harmonic mean[1] of the two groups within region 1 turns out to be 0.13. For region 2, a harmonic mean of 0.10 is calculated. Now, what is the average of both regions? If we sum up populations at risk and events for groups A and B in both regions we get rates of 0.35 and 0.16, respectively, and a harmonic mean of 0.19. Here, again, the mean for both regions is by far greater than the mean of regions 1 and 2 taken separately.

Cohort and period life expectancy

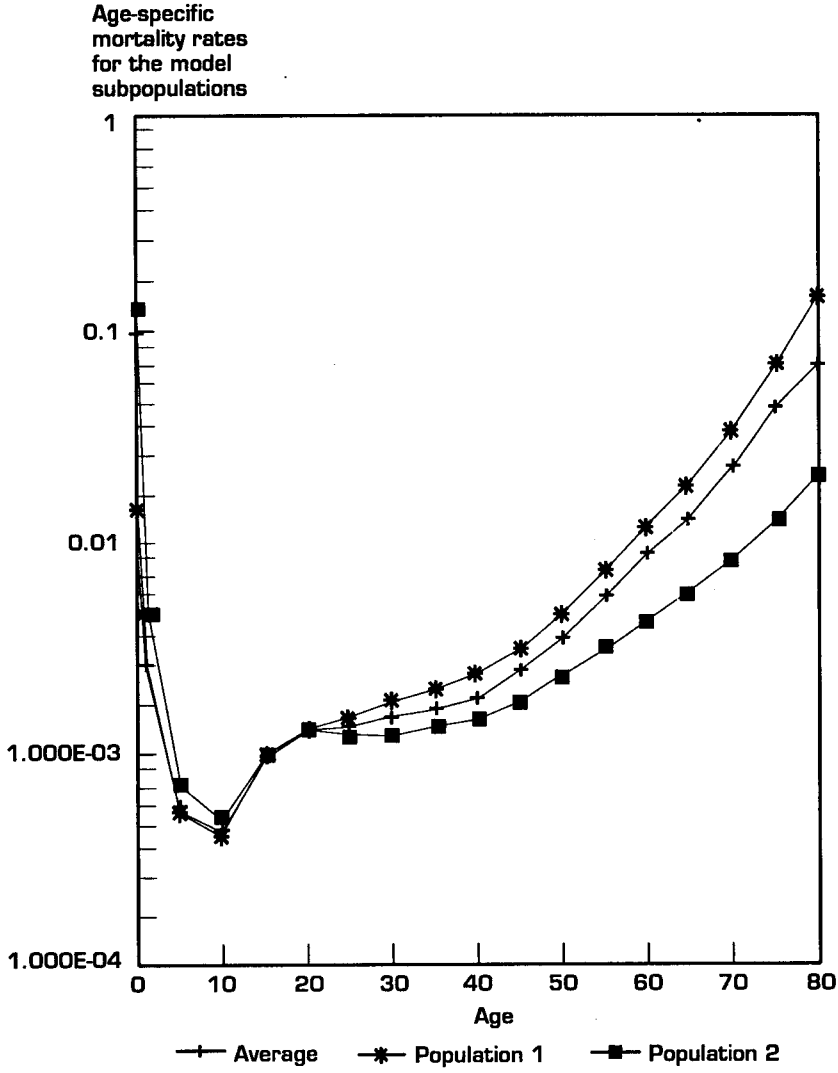
For demographers it is difficult to accept that age structures (the weights for averaging) should play a role in the calculation of life expectancies. Certainly, for cohort analysis, age structures do not influence the result regardless of the aggregating or averaging procedures. One simple proof for the insensitivity of cohort life expectancy to the phenomenon described above is the well-known fact that in a stationary population (and the cohort life-table may be viewed as such), life expectancy is identical to the mean age at death. Averaging the mean ages at death of two groups is clearly a linear function. For two populations, 1 and 2, the arithmetic mean of the two mean ages of death is always equal to the mean age at death of the joint population. In the case of cohort life-tables, this also applies to life expectancies.

A period life-table, although isomorphic to the cohort life-table, does not have the same properties. Because the period age-specific mortality rates that make up the table do not refer to one cohort that monotonically decreases with age but rather to different cohorts that might arbitrarily differ in size, age structure plays a role in averaging period life expectancy. Especially in the case when mortality curves and age distributions of the populations considered crossover, the joint period life expectancy calculated by summing up age-specific deaths and populations at risk might lie outside the range of the life expectancies in its constituent populations.

Illustrating the phenomenon in the case of two stable populations might make the underlying dynamics clearer than a set of analytical formulas.[2] Assume a stable shrinking population with an intrinsic growth rate of -0.005 (population 1) and another with a growth rate of $+0.010$ (population 2). Population 1, which might stand for an aged European society, has relatively higher mortality above age 20 and lower under age 20 than population 2, which might resemble an Asian pattern. The mortality schedules chosen result from the Brass logit life-table with the following parameters: $\alpha = -1.100$ and $\beta = 1.162$ in the case of population 1, and $\alpha = -0.627$ and $\beta = 0.230$ in the case of population 2. The force of mortality functions is plotted in figure I(a); they cross at about age 15. Figure I(b) gives the resulting stable age distributions that are also crossover.

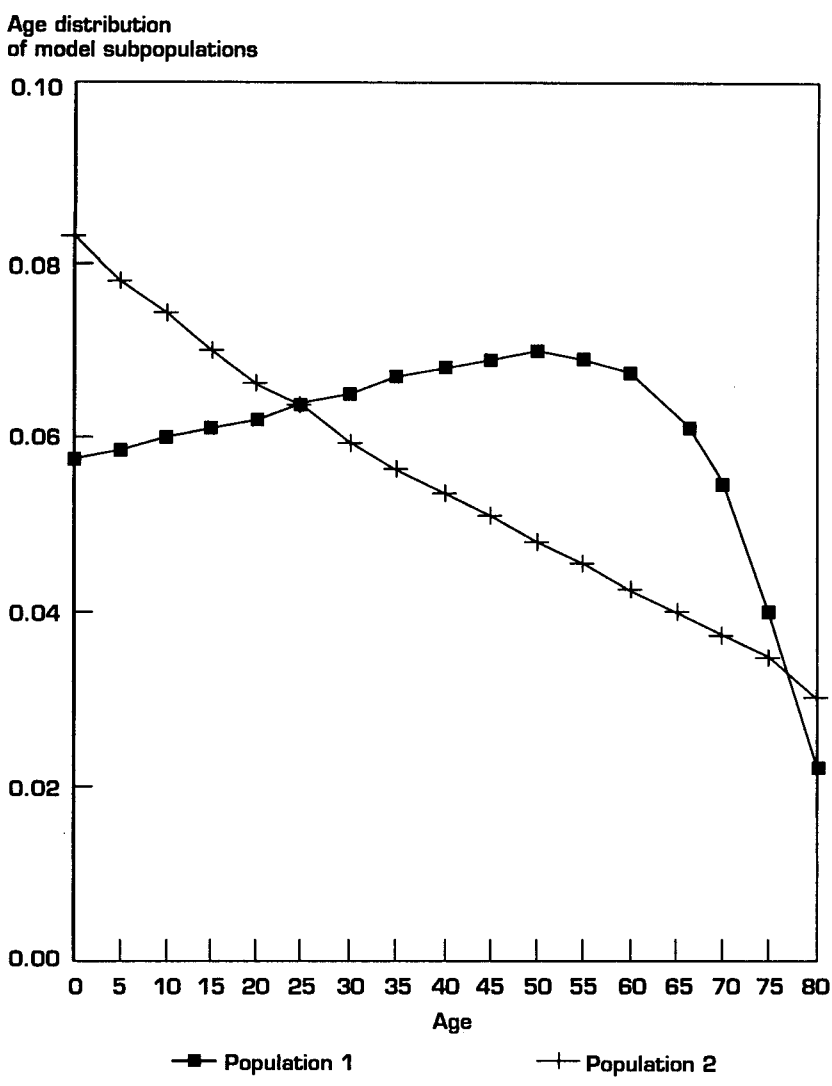
In this example the life expectancy for population 1 is 71.14 years, and for population 2, it is 73.08 years, while the joint life expectancy of

Figure I(*a*). Two selected mortality schedules and their average



both populations resulting from summations of deaths and populations at risk in each age group lies, with 69.26 years, far below the expectancies of both individual populations. Loosely spoken, we may say that the combined population suffers from bad features of both subpopulations: below age 20, the joint force of mortality function is closer to the high child mortality in population 2, and above age 20 it is closer to the higher adult mortality of population 1. This follows directly from the age-distributional weights plotted in figure I(*b*), which are greater for population 2 at the young age and for population 1 at ages 25-75.

Figure I(*b*). Stable age distributions for the two populations



EMPIRICAL RELEVANCE

In the remaining part of this article we will have a closer look at the actual empirical relevance of the above-described phenomenon. It might be useful to distinguish between two cases: one in which average life expectancy calculated by merging populations is different from the arithmetic mean but lies within the range of the means of the subpopulation, and one in which the joint life expectancy lies outside the range. Both cases are a consequence of the same structural properties of the non-linear averaging

function described above, but the second case is more extreme. It is counterintuitive to most people, and even most demographers, and therefore deserves special attention.

Concerning the first case, it is obvious and clear from table 1 that the (weighted) arithmetic mean of life expectancies and the mean life expectancy calculated by merging populations are generally not identical. Their difference would be nil or insignificant only in the few specific cases of averaging very similar populations or of differences in age structures and mortality schedules that happen to cancel out the difference between the two modes of averaging. In table 1 we find this only for North America and Oceania for 1950-1955.

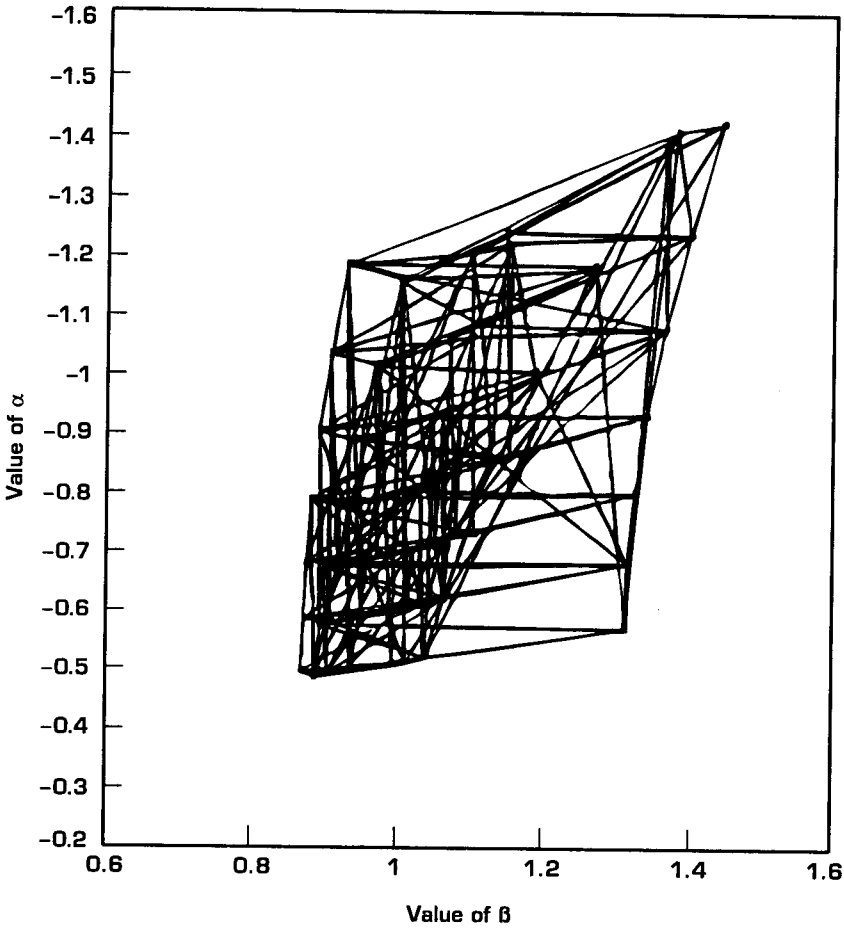
It may also be interesting to see how strongly even the arithmetic mean may be influenced by alternative weighting schemes. If life expectancies in more developed and less developed countries are not weighted by the number of births in both groups (as has been done in the 1988 revision and earlier) but by the total population, the joint life expectancy for the world in 1980-1985 turns out to be 61.4 years instead of 59.6 years, when weighting by births. Hence, for the world total, weighting by population comes much closer to the life expectancy of a merged population than weighting by births. This is caused by the simple fact that in 1980, the more developed countries had 25 per cent of the world population but only 14 per cent of all births in the world. Hence, weighting by births gives more weight to the lower life expectancy in the less developed countries.

No weighted arithmetic mean (using births or population sizes or any other weights), however, can ever come to lie outside the range of its components. This may only happen with non-linear averaging procedures, such as calculating life expectancy from a joint life-table of the subpopulations. It is still unclear, however, whether this counterintuitive phenomenon appears only in very specific cases which are interesting but not relevant in the empirical work of a demographer or whether this phenomenon is frequently encountered. These questions cannot be answered by analytical tools but only by simulation. In the paragraphs below we will describe several larger-scale simulations to arrive at estimates of the empirical relevance of situations where joint period life expectancies of two populations are greater or smaller than those of both populations taken separately.

We have to make several assumptions for the set of 100,000 pairs of population to be simulated below. We first must define the possible range of mortality patterns to be considered. We chose to do this by using the Brass logit life-table approach and expressing the complete range of regional model life-tables at different mortality levels in terms of the two parameters α and β in the Brass model (Brass and others, 1968).

Figure II presents the distribution in the space defined by α and β of the four regional types of model life-tables defined by Coale-Demeny (1966) and the five regional tables defined by the United Nations (1982) with life expectancy levels ranging from 60 to 80 in steps of 2.5 years. Altogether, this results in 81 data points that spread over a range of β-

Figure II. Space defined by the Coale-Demeny and United Nations regional model life-tables with life expectancy ranging from 60 to 80 in steps of 2.5 years and expressed in terms of the parameters α and β from the Brass logit life-table



NOTE: Lines are drawn randomly between any two points to produce some shading.

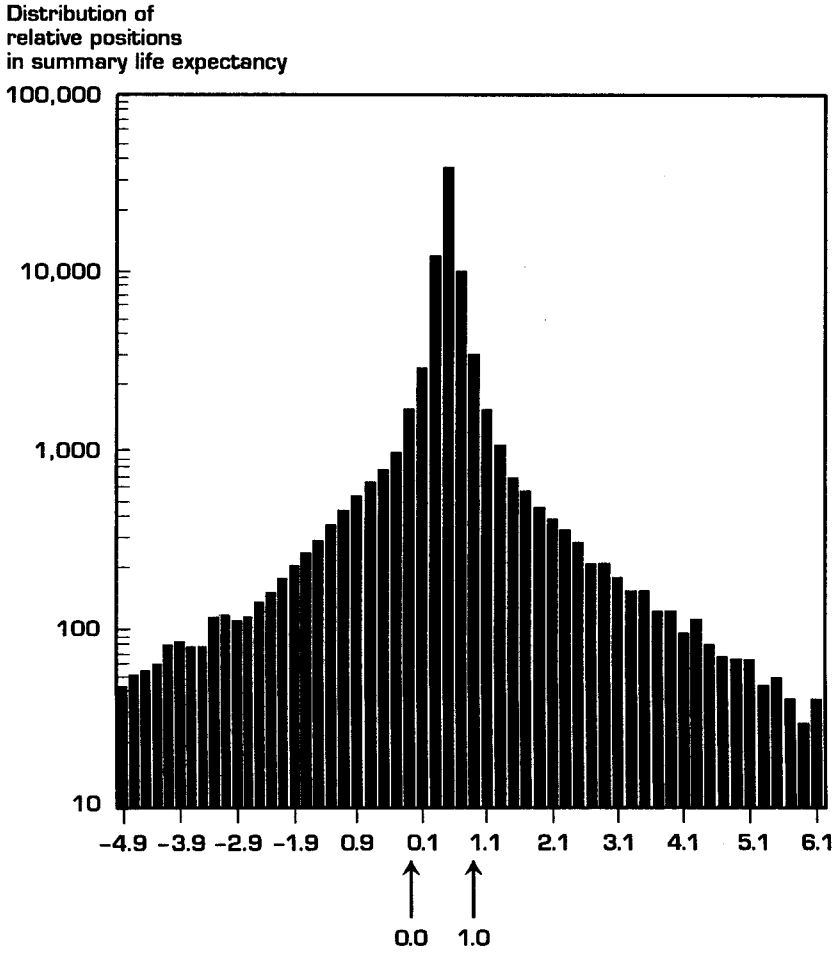
values of 0.85-1.4 and α-values of -1.5 to -0.5, with a concentration of points in the lower left corner.

Based on these 81 possible mortality patterns, couples of non-identical mortality schedules were randomly chosen, and if the difference in life expectancy did not exceed 3.0 years, they were combined with a set of two intrinsic growth rates randomly chosen from within the range -0.005-0.02 to give the age structures to which the rates will be applied. The three-year limit and the range of intrinsic growth rates were chosen in order to avoid cases unlikely to occur in reality. One hundred thousand

such pairs were randomly generated. Their distribution with respect to the relative position of the aggregate life expectancy is plotted in figure III. There, the x-axis gives the difference between the aggregate life expectancy (e_{tot}) and the lower life expectancy of the two given populations (e_{low}) divided by the difference between the two ($e_{high} - e_{low}$) (this indicator is denoted in the figure as e). If $e_{tot} = e_{low}$, the value will be 0. If $e_{tot} = e_{high}$, the value will be 1.0. Hence, all values below 0.0 and above 1.0 indicate that the aggregate life expectancy is outside the range of e_{high} and e_{low}.

On the y-axis the distribution of the 100,000 pairs is given on a logarithmic scale. The distribution is heavily concentrated around the

Figure III. Distribution of the mean life expectancy of 100,000 randomly chosen pairs based on the 81 points shown in figure II with a maximum difference in e_0 of three years



unweighted arithmetic mean of the two life expectancies. In other words, in the majority of likely empirical cases, the aggregate life expectancy lies close to the centre of the distance between the two life expectancies. In 80.5 per cent of all simulated cases, the aggregate mean lies within the range given by the two life expectancies. However, in almost one fifth of all cases it lies outside the range.

This numerical estimate gives only a very rough indication of the empirical likelihood of the phenomenon that the aggregate life expectancy may lie outside the range of the life expectancies in the populations that constitute the total. It is subject to the above-stated model assumptions and limitations. But the simulations show that the phenomenon initially described for the Soviet Union is not a marginal event that might be neglected empirically.

Sensitivity to the difference between life expectancies

A further point of interest is the question to what extent the difference between life expectancies within a given couple of populations determines the likelihood of the mean to lie outside the range. For this purpose different sets of 10,000 simulations were carried out for each of the 12 differences between life expectancies (0.25, 0.5, 1.0-10.0). Several populations were drawn from the same set of mortality schedules and age structures defined above. Only the difference in life expectancies was fixed. The results reveal a very smooth and monotonically declining association between the differences in life expectancies and the frequency of the mean lying outside the range (see fig. IV).

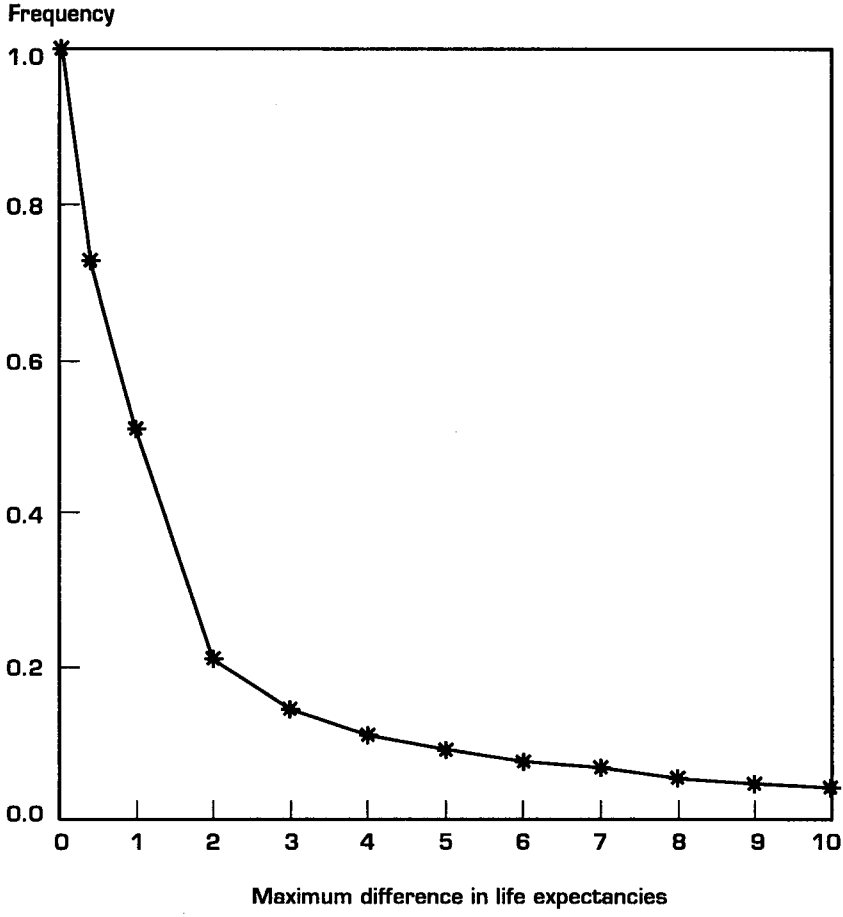
In the case of equal life expectancies (difference zero) it can be shown analytically[3] that the aggregate expectancy must be different (with probability 1) if the force of mortality functions is not identical. For a difference of one half year between the two life expectancies in about half of the 10,000 simulated cases, the aggregate life expectancy lies outside the range of that in the two subpopulations. For a difference of two years this happens only in 20 per cent of the cases. For greater differences this frequency seems to converge to a level of approximately 5 per cent.

From the above-described simulations, we may conclude that the probability for the aggregate life expectancy to lie outside the range of its components is clearly a function of the difference between life expectancies. Hence, in the case of rural and urban Ukraine with a difference in life expectancies of any percentage, it would have been the exception if the joint life expectancy would lie within the range. Of all cases considered empirically plausible here, with a difference in life expectancy of up to three years, the frequency of lying outside the range is about 20 per cent, as has also been shown in the previous simulation.

Sensitivity to the growth rate of the stable population

In the cases described above, the central point of interest was in the difference between underlying mortality schedules. Assumptions on the age

Figure IV. Dependence of the frequency of the mean lying outside the range on the difference between the two life expectancies considered



NOTE: Each point based on 10,000 simulated cases.

structures, as described by the intrinsic growth rates of the stable populations considered, had to be made but were not studied systematically. In this section, we want to study the sensitivity of the phenomenon with respect to changes in the intrinsic growth rates.

Tables 3a and 3b show matrices on the dependence of the aggregate life expectancy on the intrinsic growth rates r_1 and r_2 of stable subpopulations with randomly chosen mortality schedules for 3a: $e1 = 71.6$ and $e2 = 72.6$; for 3b: $e1 = 65.0$ and $e2 = 68.0$. As indicated in the previous section—that the probability of the mean to lie outside the range depends on the difference between the life expectancies considered—the matrices are given for differences of one year (table 3a) and three years (table 3b). Naturally the proportion of means lying outside the range is greater in the first case.

TABLE 3a. AGGREGATE LIFE EXPECTANCIES OF TWO STABLE POPULATIONS WITH RANDOMLY CHOSEN LIFE EXPECTANCIES, $e1$ AND $e2$,[a] AND INTRINSIC GROWTH RATES, $r1$ AND $r2$.

$r2 \times 1{,}000$ \ $r1 \times 1{,}000$	−5.0	−3.7	−2.5	−1.2	0.3	1.2	2.5	3.7	5.0	5.2	7.5	8.7	10.0	11.2	12.5	13.7	15.0	16.2	17.5	18.7	20.0
−5.0	72.06	71.92	71.79	71.66	71.52	71.39	71.25	71.13	71.00	70.87	70.74	70.61	70.48	70.36	70.24	70.12	70.00	59.88	69.77	69.66	69.55
−3.7	72.19	72.06	71.93	71.79	71.66	71.52	71.39	71.26	71.13	71.00	70.87	70.74	70.61	70.48	70.36	70.24	70.12	70.00	69.88	69.77	69.66
−2.5	72.33	72.19	72.06	71.93	71.79	71.66	71.52	71.39	71.26	71.13	70.99	70.86	70.74	70.51	70.48	70.36	70.24	70.11	70.00	69.88	69.77
−1.2	72.46	72.33	72.20	72.06	71.93	71.79	71.56	71.52	71.39	71.26	71.13	70.99	70.86	70.73	70.51	70.48	70.36	70.23	70.11	69.99	69.88
0.3	72.60	72.47	72.33	72.20	72.06	71.93	71.79	71.66	71.52	71.39	71.26	71.12	70.99	70.86	70.73	70.61	70.48	70.35	70.23	70.11	69.99
1.2	72.73	72.60	72.47	72.33	72.20	72.06	71.93	71.79	71.66	71.52	71.39	71.26	71.12	70.99	70.86	70.73	70.60	70.48	70.35	70.23	70.11
2.5	72.87	72.74	72.60	72.47	72.33	72.20	72.06	71.93	71.79	71.56	71.52	71.39	71.26	71.12	70.99	70.86	70.73	70.50	70.48	70.35	70.23
3.7	73.00	72.87	72.74	72.60	72.47	72.34	72.20	72.07	71.93	71.80	71.56	71.52	71.39	71.26	71.12	70.99	70.86	70.73	70.60	70.48	70.35
5.0	73.13	73.00	72.87	72.74	72.61	72.47	72.34	72.20	72.07	71.93	71.80	71.66	71.52	71.39	71.26	71.12	70.99	70.85	70.73	70.60	70.47
6.2	73.27	73.14	73.01	72.87	72.74	72.61	72.47	72.34	72.20	72.07	71.93	71.80	71.66	71.52	71.39	71.26	71.12	70.99	70.86	70.73	70.60
7.5	73.40	73.27	73.14	73.01	72.88	72.74	72.61	72.48	72.34	72.20	72.07	71.93	71.80	71.66	71.52	71.39	71.26	71.12	70.99	70.86	70.73
8.7	73.53	73.40	73.27	73.14	73.01	72.88	72.75	72.61	72.48	72.34	72.21	72.07	71.93	71.80	71.66	71.52	71.39	71.25	71.12	70.99	70.86
10.0	73.65	73.53	73.40	73.27	73.14	73.01	72.88	72.75	72.61	72.48	72.34	72.21	72.07	71.93	71.80	71.66	71.52	71.39	71.25	71.12	70.99
11.2	73.78	73.66	73.53	73.40	73.28	73.15	73.02	72.88	72.75	72.61	72.48	72.34	72.21	72.07	71.93	71.80	71.66	71.52	71.39	71.25	71.12
12.5	73.91	73.78	73.66	73.53	73.41	73.28	73.15	73.02	72.88	72.75	72.62	72.48	72.34	72.21	72.07	71.93	71.80	71.66	71.52	71.39	71.25
13.7	74.03	73.91	73.79	73.66	73.54	73.41	73.28	73.15	73.02	72.89	72.75	72.62	72.48	72.35	72.21	72.07	71.94	71.80	71.66	71.52	71.39
15.0	74.15	74.03	73.91	73.79	73.67	73.54	73.41	73.28	73.15	73.02	72.89	72.75	72.62	72.48	72.35	72.21	72.07	71.94	71.80	71.66	71.52
16.2	74.27	74.16	74.04	73.92	73.79	73.67	73.54	73.41	73.29	73.15	73.02	72.89	72.76	72.62	72.48	72.35	72.21	72.07	71.94	71.80	71.66
17.5	74.39	74.27	74.16	74.04	73.92	73.80	73.67	73.54	73.42	73.29	73.16	73.02	72.89	72.76	72.62	72.49	72.35	72.21	72.07	71.94	71.80
18.7	74.51	74.39	74.28	74.16	74.04	73.92	73.80	73.67	73.55	73.42	73.29	73.16	73.03	72.89	72.76	72.62	72.49	72.35	72.21	72.07	71.94
20.0	74.62	74.51	74.40	74.28	74.16	74.04	73.92	73.80	73.68	73.55	73.42	73.29	73.16	73.03	72.89	72.76	72.62	72.49	72.35	72.21	72.07

[a] $e1 = 71.6$; $e2 = 72.6$

TABLE 3b. AGGREGATE LIFE EXPECTANCIES OF TWO STABLE POPULATIONS WITH RANDOMLY CHOSEN LIFE EXPECTANCIES, $e1$ AND $e2$,[a] AND INTRINSIC GROWTH RATES, $r1$ AND $r2$.

$r2 \times 1{,}000$ \ $r1 \times 1{,}000$	−5.0	−3.7	−2.5	−1.2	0.0	1.2	2.5	3.7	5.0	6.2	7.5	8.7	10.0	11.2	12.5	13.7	15.0	16.2	17.5	18.7	20.0
−5.0	66.50	66.60	66.70	66.80	66.89	66.99	67.08	67.18	67.27	67.36	67.45	67.54	67.63	67.71	67.80	67.88	67.96	68.04	68.12	68.20	68.27
−3.7	66.40	66.50	66.60	66.70	66.79	66.89	66.98	67.08	67.17	67.26	67.35	67.44	67.53	67.62	67.71	67.79	67.87	67.95	68.03	68.11	68.18
−2.5	66.30	66.40	66.50	66.60	66.69	66.79	66.88	66.98	67.06	67.17	67.26	67.35	67.44	67.52	67.61	67.70	67.78	67.86	67.94	68.02	68.10
−1.2	66.20	66.30	66.40	66.50	66.59	66.69	66.79	66.88	66.97	67.07	67.16	67.25	67.34	67.43	67.52	67.60	67.69	67.77	67.85	67.93	68.01
0.0	66.11	66.20	66.30	66.40	66.49	66.59	66.69	66.78	66.88	66.97	67.06	67.15	67.24	67.33	67.42	67.51	67.59	67.68	67.76	67.84	67.92
1.2	66.01	66.11	66.20	66.30	66.40	66.49	66.59	66.68	66.78	66.87	66.97	67.06	67.15	67.24	67.33	67.41	67.50	67.59	67.67	67.75	67.83
2.5	66.91	66.01	66.11	66.20	66.30	66.40	66.49	66.59	66.68	66.78	66.87	66.96	67.05	67.14	67.23	67.32	67.41	67.49	67.58	67.66	67.74
3.7	65.82	65.92	66.01	66.11	66.20	66.30	66.39	66.49	66.58	66.68	66.77	66.86	66.96	67.05	67.14	67.23	67.31	67.40	67.49	67.57	67.65
5.0	65.73	65.82	65.92	66.01	66.11	66.20	66.30	66.39	66.49	66.58	66.68	66.77	66.86	66.95	67.04	67.13	67.22	67.31	67.39	67.48	67.56
6.2	65.64	65.73	65.83	65.92	66.01	66.11	66.20	66.30	66.39	66.49	66.58	66.67	66.77	66.86	66.95	67.04	67.13	67.21	67.30	67.39	67.47
7.5	65.55	65.64	65.73	65.83	65.92	66.02	66.11	66.20	66.30	66.39	66.49	66.58	66.67	66.76	66.85	66.94	67.03	67.12	67.21	67.29	67.38
8.7	65.46	65.55	65.64	65.74	65.83	65.92	66.02	66.11	66.21	66.30	66.39	66.48	66.58	66.67	66.76	66.85	66.94	67.03	67.12	67.20	67.29
10.0	65.37	65.46	65.56	65.65	65.74	65.83	65.93	66.02	66.11	66.21	66.30	66.39	66.48	66.58	66.67	66.76	66.85	66.94	67.02	67.11	67.20
11.2	65.29	65.38	65.47	65.56	65.65	65.74	65.84	65.93	66.02	66.11	66.21	66.30	66.39	66.48	66.57	66.66	66.75	66.84	66.93	67.02	67.11
12.5	65.21	65.30	65.38	65.47	65.57	65.66	65.75	65.84	65.93	66.02	66.12	66.21	66.30	66.39	66.48	66.57	66.66	66.75	66.84	66.93	67.02
13.7	65.13	65.21	65.30	65.39	65.48	65.57	65.66	65.75	65.84	65.93	66.03	66.12	66.21	66.30	66.39	66.48	66.57	66.66	66.75	66.84	66.93
15.0	65.05	65.13	65.22	65.31	65.40	65.49	65.57	65.66	65.75	65.85	65.94	66.03	66.12	66.21	66.30	66.39	66.48	66.57	66.66	66.75	68.83
16.2	64.97	65.05	65.14	65.23	65.31	65.40	65.49	65.58	65.67	65.76	65.85	65.94	66.03	66.12	66.21	66.30	66.39	66.48	66.57	66.66	66.74
17.5	64.90	64.98	65.06	65.15	65.23	65.32	65.41	65.50	65.58	65.67	65.76	65.85	65.94	66.03	66.12	66.21	66.30	66.39	66.48	66.57	68.66
18.7	64.82	64.90	64.99	65.07	65.15	65.24	65.33	65.41	65.50	65.59	65.68	65.77	65.85	65.94	66.03	66.12	66.21	66.30	66.39	66.48	66.57
20.0	64.75	64.83	64.91	65.00	65.08	65.16	65.25	65.33	65.42	65.51	65.59	65.88	65.77	65.86	65.95	66.03	66.12	66.21	66.30	66.39	66.48

[a] $e1 = 65.0$; $e2 = 68.0$

The intrinsic rates considered range from −0.005 to +0.020. The table clearly indicates that only in the cases when the growth rates are similar to each other does the aggregate life expectancy lie within the range of the expectancies of the two subpopulations (marked in the table). For the one-year difference in life expectancy, the difference between intrinsic growth rates may be up to 0.005; for the three-year difference, up to 0.020. In all other cases the aggregate expectancy lies outside the range.

Discussion: alternative approaches to period life expectancy

Some authors consider period life expectancy as a cumulative indicator of the mortality level in a given calendar year; other authors see it as an analogy to and a proxy of cohort life expectancy. If the first view is taken, merging the populations and calculating the joint life expectancy seems to be the appropriate procedure. Under the second approach, it seems more natural to weight the life expectancies by the number of births. This controversy is partly a matter of philosophy, but it also has significant consequences, as demonstrated above.

There is one argument in favour of merging populations which has not yet been mentioned and has to do with migration. So far we have looked only at closed populations. But in reality few populations are closed, and if we take adjacent regions into account, such as urban and rural areas, migration may be of great importance. In the urban/rural case, migration may even be the main factor that causes different age structures in the two regions.

If a certain proportion of the population born in region 1 (rural) moves to region 2 (urban) at some point, and we assume that people are exposed to the mortality risks of the region they live in (with life expectancy being higher in 2 than in 1), then the following situation appears. The group of persons born in region 1 moving to 2 will have a higher life expectancy than those staying in 1. Hence, even under stationary conditions the average cohort life expectancy for all born in region 1 will be higher than the period life expectancy in region 1. In region 2, period and cohort life expectancies should be identical. Hence, in the case of migration between regions with different life expectancies, it would be wrong even under a cohort perspective to calculate an arithmetic mean of period life expectancies weighted by births. In the stationary case, it may even be shown that merging the populations yields the correct life expectancy under a real cohort perspective (see Andreev, Lutz and Scherbov, 1989).

We may conclude that merging populations and calculating a joint life expectancy is the more appropriate procedure if we assume migration between the subpopulations. For closed populations, the case is less clear. Especially in the case of calculating a joint life expectancy for both sexes, the question becomes very difficult. Here we come back to the difference in philosophies mentioned above. However, under changing mortality conditions, a phenomenon currently observed in virtually every country in the world, it will be hard to justify the interpretation of period life expectancy as a proxy of the life expectancy of a baby born in that year. We know

that for the past decades, this would have been grossly misleading. The prospect of further mortality improvements that might even be different for different populations is also an argument against weighting life expectancies by the number of births.

The practices in national statistical offices differ. While in the United States of America male and female life expectancies are weighted according to the sex ratio at birth, in other countries, such as Austria, they are averaged 1:1, and in a third group of countries to which the Soviet Union belongs, the male and female populations are merged according to age groups in the way we discussed above.

In conclusion, we may say that the choice of an appropriate averaging procedure remains difficult and controversial in the case of averaging male and female life expectancy. When averaging regions or other subpopulations which are open to migration, a merging of populations and the calculation of a joint life expectancy seems to be clearly preferable. The fact that under this procedure, in some cases the seemingly paradoxical situation arises that the mean is higher or lower than life expectancies in the constituent populations is an explainable phenomenon due to the variation of mortality by age and not an argument against the procedure.

Notes

[1] The weighted harmonic mean $\bar{x}_H$ for j observed values x_j with frequencies $n_j (n = \Sigma nj)$ is defined by

$$\bar{x}_H = \frac{n}{\sum_{j=1}^{k} nj/xj}$$

[2] The mathematics of this phenomenon has recently been described in Andreev, Lutz and Scherbov (1989) and in Haunsperger (1991).

[3] This is a theorem proven in Andreev and others (1989).

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ESTIMATION OF ADULT MORTALITY FROM PATERNAL ORPHANHOOD: A REASSESSMENT AND A NEW APPROACH

*Ian M. Timæus**

SUMMARY

This article proposes a new procedure for estimating men's mortality from paternal orphanhood which generally yields more accurate results than the existing approach. A procedure for estimating mortality from maternal orphanhood data based on consistent assumptions is also presented. The theory underlying these methods is outlined, focusing on aspects of it that have not been explained fully in the existing literature and that influence the specification and robustness of the models used for estimation. The article also points out an error made in the tabulation of the weighting factors used until now to estimate mortality from paternal orphanhood. Investigations using simulated data are presented which support the theoretical arguments that suggest that the paternal orphanhood method is more robust than has often been assumed and which confirm that the new approach usually produces more accurate estimates than the weighting factors.

The development of reliable and affordable methods for measuring adult mortality in countries that lack adequate vital statistics systems has proved a major challenge. While considerable ingenuity has been deployed to good effect to devise ways of rendering incomplete reports of recent deaths usable, such methods can be applied only when the majority of events are reported (Brass, 1975; Preston and others, 1980; Preston, 1984; Timæus and Graham, 1989). Thus, indirect techniques remain important sources of mortality estimates. This article proposes an improved procedure for making such estimates from data on the survival of fathers.

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The first simple, robust method for estimating mortality from orphanhood was proposed by Brass and Hill (1973). It relates the proportions of respondents with living mothers or fathers in two adjoining age groups to measures of life-table survivorship by means of a system of weighting factors whose values depend on the mean age of child-bearing. Subsequent to the derivation of these weighting factors, several regression-based approaches to the estimation of women's mortality from data on maternal orphanhood have been proposed (Hill and Trussell, 1977; Palloni and Heligman, 1985; United Nations, 1983). Equivalent methods for the estimation of men's mortality from paternal orphanhood have not been developed because of the lack of a satisfactory, flexible model of male fertility and persistent scepticism about the robustness of the method (Brass, 1975; Hill and Trussell, 1977; Hill, 1984; Palloni and Heligman, 1985).

Despite the concern about the sensitivity of the paternal orphanhood method, little effort has been devoted to its evaluation. Research into the maternal orphanhood method has suggested that the weighting factors perform well for women's mortality at young ages but that a regression-based estimation procedure produces better results from data on older respondents (United Nations, 1983). Why this is so and whether it would also be true for paternal orphanhood remain unclear. Moreover, this analysis gives little clue as to which of the assumptions made in the derivation of the estimation procedures are crucial and whether there are particular conditions in which the accuracy of the estimation procedures is unacceptably low.

It is these issues, rather than reporting errors (Blacker and Mukiza-Gapere, 1988; Palloni, Massagli and Marcotte, 1984; Timæus, 1990 and 1991), that are considered here. The article outlines several theoretical considerations that affect the specification and robustness of the models used to estimate mortality from orphanhood. Using simulated data, it assesses the impact of breaches in the assumptions made by the estimation procedures on the results. On the basis of the preceding discussion, it proposes an improved method for analysis of paternal orphanhood data, which combines some of the advantages of both the weighting factors and the existing regression methods for analysing data on maternal orphanhood. A procedure for estimating women's mortality based on consistent assumptions is also presented. Finally, simulated data are used to assess the performance of the new paternal orphanhood method. To provide the basis for the discussion that follows, the rationale of Brass and Hill's approach is outlined briefly in the section below. The relationship between life-table survivorship and the survival of mothers is considered first.

Methodological background

If we consider respondents born a years before a demographic enquiry to women aged y and assume a stable age structure and that the mortality of orphans and children with living parents is the same (Blacker, 1984; Blacker and Mukiza-Gapere, 1988; Palloni, Massagli and Marcotte,

1984; Timæus, 1990), the proportion of respondents in a five-year age group with living mothers is (Brass and Hill, 1973):

$$ {}_5S_x = \frac{\int_x^{x+5} e^{-ra} l(a) \int_s^w e^{-ry} l(y+a) f(y)\, dy\, da}{\int_x^{x+5} e^{-ra} l(a) \int_s^w e^{-ry} l(y) f(y)\, dy\, da} \qquad (1) $$

where integration by y is over all ages at child-bearing, s to w; r is the rate of natural increase; and $f(y)$ is the fertility rate at age y.

There is no straightforward way of integrating either the numerator or the denominator of equation (1); thus it has to be evaluated numerically. Brass and Hill (1973) evaluate $e^{-ry} l(y+a) f(y)$ for five-year age groups and add the values together to approximate the integral. They take r, the rate of growth, as 2 per cent; the $l(a)$ values from Brass's (1971) General Standard; and represent fertility by a cubic function of the form $f(y) = (y - s)(s + 33 - y)^2$, where s is the age at which child-bearing starts.

Every value of ${}_5S_x$, for an age group with mid-point N, is equal to a survivorship ratio $l(B + N)/l(B)$ in the standard life-table used to generate the estimate of ${}_5S_x$. The value of B is close to $\bar{M}$, the unstandardized mean age at child-bearing, but is dependent on N. Instead of working with B and N, however, it is more convenient to adjust the proportions slightly and make estimates for b and n, where b is a fixed round numbered age near $\bar{M}$ and n is a multiple of five years. For the estimation of mortality from maternal orphanhood, Brass and Hill set b to 25 years and calculate the life-table survivorship ratios as weighted averages of the proportions of respondents with living mothers in the two age groups surrounding age n:

$$ {}_nP_{25} = W_n \cdot {}_5S_{n-5} + (1 - W_n) {}_5S_n $$

where ${}_nP_{25}$ is equivalent to $l(25 + n)/l(25)$. They present a table of these weights, W_n, for mean ages at child-bearing from 22 to 30 years.

Estimation of adult men's mortality from the proportion of respondents with living fathers is based on the same principles. It is possible for fathers to die between the time when a child is conceived and when it is born. This affects the relationships in equation (1) in several ways.

First, the age of the respondents becomes 0.75 years less than a, the duration of exposure of the fathers. Brass and Hill allow for this when they calculate the proportions of fathers surviving for five-year age groups of respondents from point estimates for a years of exposure.

Secondly, for paternal orphanhood the values of ${}_5S_x$ for an age group with a mid-point N are equivalent to a survivorship ratio $l(B + N + 0.75)/l(B)$ where B is, in this case, close to the mean age of fathers at the conception of their children. As for maternal orphanhood, it is convenient to calculate weighting factors for a fixed round age b and ages n that are multiples of 5 years. However, both because fathers' survival is being measured over an interval nine months longer than the respondents' ages and because of the high mortality of the men brought in by the long tail of the male fertility distribution, the amount of extrapola-

tion from the observed proportions is reduced if survivorship is measured to the end of the upper age group. Therefore, Brass and Hill estimate life-table survivorship from data on the survival of fathers using:

$$l(b + n + 2.5)/l(b) = W_n \cdot {}_5S_{n-5} + (1 - W_n){}_5S_n$$

They present two tables of weighting factors with age b set to 32.5 years and 37.5 years, depending on the mean age of fathers at the birth of their children.

Thirdly, equation (1) only holds for men if the $f(y)$ distribution is treated as the distribution of fathers' ages at the conception of their live-born children, rather than as a fertility distribution. As a result, the mean age, $\overline{M}$, calculated from this distribution and the age structure in equation (1), represents the mean age of fathers at the conception of their live-born children. It is approximately three quarters of a year less than the mean age of child-bearing.

By recalculating the weighting factors using the original methodology, one can establish that no allowance was made for this difference in the interpretation of $\overline{M}$ in the tables of weighting factors for paternal orphanhood presented by Brass and Hill. Thus, each of the columns of weighting factors tabulated for integer values of the mean age of child-bearing in fact applies to a mean age of child-bearing 0.75 years greater.[1] For example, the weights tabulated for $\overline{M} = 33$ years are the correct weights for a mean age of child-bearing of 33.75 years. Almost all estimates of male survivorship in adulthood made from orphanhood data during the past 20 years have been calculated using these weighting factors. They are all biased systematically upward. Fortunately, the size of the bias is reasonably small, being equivalent, on average, to the overestimation of life expectancy at age 15 or 20 by about half a year.

Robustness of the estimation procedures

Certain aspects of the rationale of the orphanhood method are not discussed in detail by either Brass and Hill or those who have contributed subsequently to this method's development. First, Brass and Hill do not explain why they adopt a series of weighting factors for estimating adult mortality from orphanhood, rather than the system of multiplying factors that Brass had proposed originally (Brass, 1975). Secondly, although it is widely assumed that the paternal orphanhood method is less robust than the maternal orphanhood method, neither the reasons why this is so nor the accuracy of the results have been investigated in detail (Brass, 1975; Hill and Trussell, 1977; Hill, 1984, Palloni and Heligman, 1985). Thirdly, Brass and Hill (1973) do not demonstrate that the relationship between the ${}_nP_b$ ratios and ${}_5S_x$ proportions, which they estimate using Brass's General Standard, holds at other levels of mortality. These issues are considered in this section of the article. The findings reveal that a better approach exists to the estimation of men's mortality from orphanhood than either the weighting factors or a regression model of the form proposed for women's mortality.

The advantage of a system of weighting factors, rather than one of multipliers, is that it reduces the sensitivity of the estimates to variation in age patterns of mortality and child-bearing from the standard patterns used to derive the weights. If the rate of decrease in parental survival around age n differs from that assumed, the relationships between ${}_np_b$ and the proportions of respondents with living parents in each of the two age groups adjoining n are shifted in opposite directions. The effect of these biases on the results tends to cancel out. By evaluating equation (1) with a range of fertility and mortality models, one can establish that, at young ages (i.e., approximately when ${}_5S_x > 0.5$), the impact of a different age pattern of mortality or child-bearing on the measures of interest is almost exactly proportional to the probability of *dying* between ages b and $b + n$ or its equivalent, the probability of being *orphaned* by age n. To the extent that this finding holds, such breaches in the assumptions introduce no error into the weighting factors. To express this point more formally, if the incidence of orphanhood and mortality differ from those in the standard population by a constant factor, k, so that ${}_np_b = {}_np_b^s + k(1 - {}_np_b^s)$ then the correct weight, W_n^*, equals W_n:

$$W_n^* = \frac{\{{}_np_b^s + k(1 - {}_np_b^s)\} - \{{}_5S_n^s + k(1 - {}_5S_n^s)\}}{\{{}_5S_{n-5}^s + k(1 - {}_5S_{n-5}^s)\} - \{{}_5S_n^s + k(1 - {}_5S_n^s)\}}$$

$$= \frac{(1-k){}_np_b^s - (1-k){}_5S_n^s}{(1-k){}_5S_{n-5}^s - (1-k){}_5S_n^s} = W_n \qquad (2)$$

In contrast, given these conditions, if a series of multiplying factors, m_n, is used, then the life-table estimates will all be in error by $k(m_n - 1)$.

To some extent, the derivation of regression-based approaches to the estimation of mortality from maternal orphanhood has provided justification for procedures that estimate life-table survivorship from parental survival data, controlling for the mean age at child-bearing. The goodness of fit (R^2) of models of this form is high, although the average relative error in the estimates increases with the age of the respondents and is rather large for those that are middle-aged (Palloni and Heligman, 1985).

Mammo's research has clarified the reasons why the sensitivity of the estimates increases with the age of the respondents (Mammo, 1988). He shows mathematically that, after controlling for the mean age at child-bearing, the relationship between parental survival and life-table survivorship is determined largely by the variance of ages at child-bearing multiplied by ${}_aP''_{\bar{M}}/{}_aP_{\bar{M}}$, where ${}_aP''_{\bar{M}}$ is the second differential of the probability of surviving from the mean age of child-bearing to that age plus the age of the respondents. This factor reflects the age pattern of mortality. Mammo further demonstrates that ${}_aP''_{\bar{M}}/{}_aP_{\bar{M}}$ increases with $\mu(\bar{M} + a)$ and, therefore, with a. Thus, at young ages the proportion of respondents with living parents is closely related to the parents' probability of surviving from $\bar{M}$ to $\bar{M} + a$, limiting the impact that mis-specification of either the pattern of mortality or the variance of ages at child-bearing (which is multiplied by the former factor) can have on the estimates of survivorship. As the age of

the respondents increases, ${}_aP''_{\bar{M}}/{}_aP_{\bar{M}}$ becomes much larger. The mortality estimates become increasingly sensitive to errors in the assumptions about age patterns of fertility and mortality which are incorporated in the models used to derive the estimation procedure.

Mammo's findings also explain why the paternal orphanhood method is less robust than the maternal orphanhood method. The variance of the distribution of ages at child-bearing for men is larger than that for women. Moreover, partly because of the prevalence of polygyny in some parts of the world, variability in both the timing and the dispersion of male fertility distributions is far greater than that in female fertility distributions. In addition, in almost all populations, people's fathers are several years older, on average, than their mothers. Therefore, ${}_aP''_{\bar{M}}/{}_aP_{\bar{M}}$ is larger. Thus, both the likely differences between the actual characteristics of the fertility and mortality distributions and those assumed and the impact of such errors on the results are larger for men than for women. It therefore seems important to assess the sensitivity of paternal orphanhood estimates to variation in age patterns of child-bearing and mortality.

To assess the size of the biases in the mortality estimates that could arise from inappropriate assumptions, one can calculate the proportions of respondents with living fathers from known fertility and mortality schedules by evaluating equation (1) numerically. Then one can estimate mortality from these proportions using the weighting factors and compare these estimates with the schedule used to generate the parental survival data.

To determine the sensitivity of the estimates to each of the assumptions, the effects of variation in the level (α) and age pattern (β) of mortality in the relational logit model life-table system based on the General Standard (Brass, 1971), age structure (as determined by r) and the width of the fertility distribution are examined in turn, holding the other parameters constant at the values used to derive the weights. The effect of different age patterns of fertility is examined using a relational Gompertz model and a standard developed recently by "stretching" the female standard to age 80 (Paget and Timæus, 1990). The model fits male fertility distributions remarkably well. The standard fertility schedule is broadly similar in shape to the polynomial used by Brass and Hill to represent male fertility and is taken as the central pattern, although it exhibits somewhat higher fertility at late ages. A β_f of 0.675 produces a broad fertility distribution, such as those characteristic of polygynous societies, and a β_f of 1.75 a narrow fertility distribution, such as those characteristic of countries with fairly low fertility.

Table 1 shows the absolute errors in the level of mortality, as indexed by α, which result when men's mortality is estimated from the proportion of respondents with living fathers under different conditions from those assumed by Brass and Hill (1973) to derive the weighting factors. Positive errors signify that the level of mortality is overestimated and vice versa. A bias in the estimated value of α of 0.1 represents about a 1.5 year error,

TABLE 1. ERRORS IN ESTIMATES OF THE LEVEL OF MORTALITY (α) FROM PATERNAL ORPHANHOOD IN POPULATIONS WITH DIFFERENT CHARACTERISTICS, SELECTED MEAN AGES AT CHILD-BEARING ($\bar{M}$) AND CENTRAL AGES OF RESPONDENTS (n)

	$\bar{M}$ = 29.75			$\bar{M}$ = 34.75			$\bar{M}$ = 39.75		
	n = 10	n = 25	n = 40	n = 10	n = 25	n = 40	n = 10	n = 25	n = 40
Mortality									
α = 0.4.....	−0.016	−0.032	0.022	−0.057	−0.067	−0.039	−0.075	−0.075	0.007
−0.4.....	0.019	0.043	0.029	0.043	0.064	0.061	0.059	0.074	0.032
−0.8.....	0.036	0.096	0.101	0.078	0.132	0.152	0.111	0.160	0.105
β = 0.6.....	0.015	0.050	0.154	−0.011	0.037	0.180	−0.003	0.074	0.250
1.4.....	−0.014	−0.056	−0.139	0.009	−0.043	−0.164	−0.002	−0.077	−0.202
Growth rate									
r = 0.5%....	0.008	0.011	−0.004	0.013	0.012	−0.006	0.016	0.011	−0.028
3.5%....	−0.010	−0.012	0.006	−0.014	−0.013	0.007	−0.016	−0.011	0.029
Fertility									
β_f = 0.675.....	0.079	0.052	−0.076	0.087	0.047	−0.068	0.083	0.019	−0.124
1.375.....	−0.040	−0.042	0.035	−0.051	−0.043	0.040	−0.055	−0.032	0.136
1.75.....	−0.065	−0.066	0.062	−0.078	−0.065	0.069	−0.085	−0.047	0.232
1.....	0.004	−0.003	−0.008	0.000	−0.006	−0.006	−0.002	−0.008	0.011

NOTE: The errors are estimated from the proportions of fathers surviving in populations in which $\alpha = 0$, $\beta = 1$, $r = 2\%$, $\alpha_f = 0$ and $\beta_f = 1$, except as explicitly varied. As the final row of the table indicates, the reference population has very similar characteristics to that used by Brass and Hill (1973) to calculate the weighting factors.

The mean ages of child-bearing given in the table are the correct ones, and the errors have been estimated using the weighting factors tabulated for $\bar{M} - 0.75$ years.

on average, in the corresponding estimate of the expectation of life at age 15.

If the actual demographic characteristics of a population differ from those assumed to calculate the weighting factors, the resulting errors in estimates of men's mortality are smaller than one might expect, given the reservations expressed about this method in the literature. As for maternal orphanhood (Palloni and Heligman, 1985), the errors increase in size with the age of the respondents, but are acceptable for the age groups used for estimation.[2] As a very approximate guide, for central ages of respondents, n, of 35 or less, deviations from the assumptions made in the derivation of the paternal orphanhood weights are unlikely to introduce errors into estimates of male life expectancy at age 15 of much over 1 year.[3]

Differences in the rate of natural increase of 1.5 per cent have a large impact on population age structure but an insignificant impact on the estimated level of mortality, confirming Brass and Hill's argument (1973) that the orphanhood method is very robust to errors in the assumptions made about the age distribution of the population. As the earlier discussion suggests, however, when the width of the male fertility distribution is very different from that assumed, appreciable biases are introduced into the estimates of mortality. If men's ages at child-bearing are very dispersed, the

level of mortality estimated from data on young respondents will be too high and the level estimated from data on older respondents too low. Thus, the decline in mortality over time inferred from the estimates will be underestimated slightly. In contrast, if child-bearing is very concentrated, the extent of mortality decline will be exaggerated. Except among older respondents in populations with extreme mean ages at child-bearing, the errors remain fairly small.

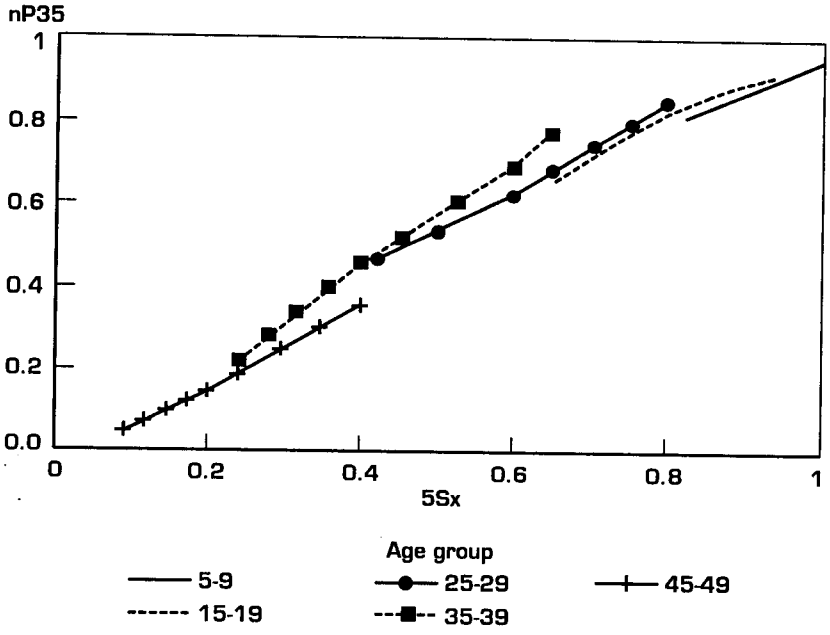
As earlier work on maternal orphanhood has suggested (Palloni and Heligman, 1985), the estimates are most sensitive to differences between the level and pattern of mortality and the standard schedule. The direction of the errors follows a systematic pattern. If mortality is higher overall than was assumed in the estimation of the weights or increases rapidly with age, the survivorship ratios estimated from parental survival are biased upwards. Mortality will be underestimated and, as the errors increase with age, the decline in mortality over time will be understated. Conversely, if mortality is lower than was assumed when deriving the weights or increases slowly with age, it will be overestimated and the decline in mortality exaggerated.

The sensitivity of orphanhood estimates obtained using the weighting factors to variation in the level and pattern of mortality is somewhat perturbing, especially as the weights represent the only method proposed hitherto for estimating men's mortality from the survival of fathers. The expectation of life at birth in the General Standard is about 43 years, which represents rather heavier mortality than is commonly found in the developing world today. In general, therefore, if reporting is accurate, the weighting factors produce slight overestimates of men's mortality. The bias is large enough to be of concern when mortality is either very heavy or very light. The fact that the size of the bias is systematically related to the level of mortality, and therefore orphanhood, suggests that it should be possible to improve the accuracy of the estimates.

The source of the bias becomes clear when life-table measures of survivorship at a range of levels of mortality are plotted against the corresponding simulated proportions of respondents with living fathers. This relationship is illustrated for several age groups in figure I using mortality schedules generated from the General Standard by varying α, which have expectations of life at birth that range from 30 to 74 years. The errors do not arise from non-linearities in the relationship between orphanhood and mortality. Except at extremely high and low mortality and in old age, the relationship is close to linear.

In fact, the bias in orphanhood estimates obtained using the weighting factors stems from the lack of an intercept term. The relationship *is* non-linear at the extremes, which largely fall outside the range of human experience. Thus, as mortality declines, the proportion of respondents with living fathers increases more slowly than a line fitted to a single mortality schedule and passing through the origin would suggest. The problem becomes more serious as the age of respondents increases because the proportion of them with living fathers varies more with the level of mortality.

Figure I. Relationship between paternal orphanhood and life-table survivorship at different levels of mortality in selected age groups ($\bar{M}$ = 35 years)



Thus, mortality is underestimated systematically, when it is high, and overestimated, when it is low.

This problem would not arise with an estimation procedure including an intercept term, which could model relationships such as those shown in figure I correctly. Further minor increases in the overall precision of the method would result from replacing the function used to represent fertility in the original derivation of the weighting factors with more sophisticated model fertility distributions.

Revised estimation procedures

New regression coefficients for the estimation of adult mortality from orphanhood are presented in tables 2 and 3. The coefficients have been calculated for maternal as well as paternal orphanhood to provide a consistent basis for estimation for the two sexes. They were estimated from simulated data created from relational logit model life-tables, based on the General Standard, and fertility distributions generated, using the relational Gompertz model, from Booth's standard (1984) for females and the Paget and Timæus' standard (1990) for males. These standards were developed for use in populations with high fertility. As in the original calculations, the age distribution of the parents is represented by a stable population with a rate of increase of 2 per cent.

TABLE 2. COEFFICIENTS FOR THE ESTIMATION OF FEMALE SURVIVORSHIP FROM THE PROPORTION OF RESPONDENTS WITH LIVING MOTHERS

n	$\beta_0(n)$	$\beta_1(n)$	$\beta_2(n)$	R^2	CV^a
10	−0.2894	0.00125	1.2559	0.997	0.0015
15	−0.1718	0.00222	1.1123	0.996	0.0031
20	−0.1513	0.00372	1.0525	0.995	0.0058
25	−0.1808	0.00586	1.0267	0.993	0.0088
30	−0.2511	0.00885	1.0219	0.992	0.0126
35	−0.3644	0.01287	1.0380	0.992	0.0172
40	−0.5181	0.01795	1.0753	0.992	0.0222
45	−0.6880	0.02342	1.1276	0.993	0.0271
50	−0.8054	0.02721	1.1678	0.992	0.0400

Estimation equation: $l(25 + n)/l(25) = \beta_0(n) + \beta_1(n)\bar{M} + \beta_2(n)_5S_{n-5}$

[a] Coefficient of variation = root mean squared error divided by the mean of ${}_nP_{25}$.

TABLE 3. COEFFICIENTS FOR THE ESTIMATION OF MALE SURVIVORSHIP FROM THE PROPORTION OF RESPONDENTS WITH LIVING FATHERS

n	$\beta_0(n)$	$\beta_1(n)$	$\beta_2(n)$	β_3	R^2	CV^a
10	−0.8251	0.00261	2.7269	−0.9953	0.978	0.0066
15	−0.4013	0.00576	1.5602	−0.3522	0.974	0.0117
20	−0.3329	0.01031	0.6656	0.3419	0.976	0.0172
25	−0.4726	0.01559	0.2161	0.7896	0.981	0.0216
30	−0.7056	0.02076	0.1997	0.9066	0.988	0.0239
35	−0.9153	0.02493	0.3484	0.8631	0.992	0.0261
40	−0.9950	0.02635	0.4269	0.8263	0.987	0.0466

Estimation equation: $l(35 + n)/l(35) = \beta_0(n) + \beta_1(n)\bar{M} + \beta_2(n)_5S_{n-5} + \beta_3(n)_5S_n$

[a] Coefficient of variation = root mean squared error divided by the mean of ${}_nP_{35}$.

The proportion of respondents with living mothers by five-year age group in each simulated population was calculated by evaluating equation (1) by summation over single years of age, y and a, taking the growth and survivorship functions in y at the midpoint of each year. For paternal orphanhood, the polynomial used by Brass and Hill to represent $f(y)$ has a fixed shape, implying that it can equally well be regarded as representing the distribution of men's ages at the conception of their live-born children and a fertility distribution. In contrast, the shapes of the $f(y)$ distributions produced by the relational Gompertz model vary with their mean. Therefore, it is preferable to retain the distributions' interpretation as representing fertility. Thus, the coefficients for paternal orphanhood were calculated by evaluating not equation (1) but a corresponding expression in which a and y retain identical definitions as for maternal orphanhood:

$$ {}_5S_x = \frac{\int_x^{x+5} e^{-ra} l(a) \int_s^w e^{-ry} l(y) l(y+a)/l(y-0.75) f(y)\, dy\, da}{\int_x^{x+5} e^{-ra} l(a) \int_s^w e^{-ry} l(y) f(y)\, dy\, da} \qquad (3) $$

The parameters used to generate the model populations are shown below. Inspection of the residuals and of the bivariate relationships between the mean age at child-bearing and the proportions of surviving parents, on the one hand, and the predicted life-table measures, on the other, confirms that the relationships are linear and that the variance of the independent variables is unrelated to the dependent measures for both males and females.[4] Coefficients can be estimated reliably, therefore, from a fairly small set of data. The β parameter of the mortality models, which affects the age pattern of mortality, and the β_f parameter of the fertility models, which largely determines the variance of the distributions, were set to two and three different values, respectively, to check that this did not affect the relationships of central concern. It did not seem worthwhile, however, to introduce a large number of intermediary populations, when it was not proposed to model this variation. The resulting populations have life expectancies at birth that range from 36 to 73 years, averaging 55 years, and life expectancies at age 15 that vary between 39 and 62 years, averaging 50.6 years.

Parameters that define the simulated populations for estimation of the relationship between parental survival and life-table measures

Mortality
$\alpha = 0.2, -0.2, -0.6, -1.0$
$\beta = 0.8, 1.1$
Brass (1971) General Standard

Fertility—females
$\alpha_f = -0.5, -0.2, 0.1, 0.4$
$\beta_f = 0.7, 1.0, 1.3$
Booth (1984) standard

Fertility—males
$\alpha_f = -0.4, -0.1, 0.2, 0.6$
$\beta_f = 0.7\ (\alpha < 0.6), 1.0, 1.3\ (\alpha > -0.4)$
Paget and Timæus (1990) standard

Age structure
$r = 2\%$

Although in the course of fertility decline the ages of women bearing children usually change from being distributed widely with a late mean to an earlier and narrower schedule, exceptions to this pattern exist. Therefore, the coefficients for maternal orphanhood were estimated using the full set of fertility distributions, resulting in a sample of 96 populations. The mean age at child-bearing of mothers falls between 23.5 and 31.5 years and averages 27.3 years. In contrast, the male fertility schedules assembled by Paget and Timæus (1990) suggest a strong relationship between the α_f and β_f parameters of the relational Gompertz model. Late ages at child-bearing are found mainly in polygynous societies that also have wide male fertility distributions. Therefore, no populations with early (but very wide) or late (but very narrow) fertility distributions were included in the set used to model the relationship between paternal orphanhood and men's mortality. A sample of 80 populations results, with mean ages at child-bearing that range from 29.3 to 40.3 years, with an average of 34.5 years.

The model used to predict women's mortality is the same as that proposed previously (Hill and Trussell, 1977; Palloni and Heligman, 1986; United Nations, 1983):

$$_nP_{25} = \beta_0(n) + \beta_1(n)M + \beta_2(n)_5S_{n-5}.$$

New coefficients for this model are presented so that estimates can be made for ages, n, of 10 and 15 years and can be obtained for men and women using the same assumptions. There is no reason to suppose that the coefficients will perform any better than those published before. Indeed, it is reassuring to note that, although they are based on a different set of simulated populations, the coefficients are very similar to those published in *Manual X: Indirect Techniques for Demographic Estimation* (United Nations, 1983).

The equivalent regression equation for men sometimes produces poor estimates of mortality. Better results can be obtained, especially for populations with light mortality and late ages at child-bearing, by including information on a second age group in the model. It is argued earlier that use of weighting factors, rather than multipliers, increases the robustness of the orphanhood method to variation in the age patterns of mortality and child-bearing. The model proposed here for men combines this advantage of the weights with those of a regression-based approach.[5] While the proportion of respondents with surviving parents in two adjoining age groups is highly correlated, the additional parameter captures the effect of variation in the slope of their relationship with mortality, reducing the mean errors in the estimates for the older age groups across the set of populations used here by about 17 per cent.

The mean age at child-bearing of men in the simulated data is 34.5 years, which represents a reasonable central value for the developing countries as a whole. This suggests that measures of life-table survivorship from a base age, b, of 35 years should be related closely to the proportions of fathers surviving. Experimentation with different regression models confirmed that this statistic could be predicted as precisely as any other dependent variable and also indicated that the gains from fitting different models to populations with early and late mean ages of child-bearing would be modest. Thus, the model chosen to estimate men's mortality is:

$$_nP_{35} = \beta_0(n) + \beta_1(n)M + \beta_2(n)_5S_{n-5} + \beta_3(n)_5S_n.$$

The relative errors in the estimated survivorship ratios, shown in tables 2 and 3, give some indication of the performance of the coefficients for paternal orphanhood, compared with the now well-established model for estimating women's mortality.[6] They are encouraging. For example, the precision of the estimates of men's mortality obtained from respondents aged 15-24 years is similar to that of the estimates of women's mortality obtained from respondents aged 30-34 years. The relative errors in the estimates increase rapidly at older ages. For this reason, no coefficients are presented for ages, n, of over 50 years for women's mortality or of over 40 years for men's mortality.

The size of the errors in estimates of α for men which result when mortality is estimated from further simulated populations by means of the procedures presented here is illustrated for a range of populations in table 4. While these results can be compared with those shown in table 1, they are not exactly equivalent. First, other things being equal, the regression method should yield better estimates in low-mortality populations and worse ones when mortality is high, since it was derived from populations with an average life expectancy at birth 12 years greater than that in the General Standard. Thus, except when their impact is being assessed, the mortality parameters used to generate the data are set to $\alpha = -0.4$ and $\beta = 0.95$, rather than to 0 and 1, respectively. Secondly, while the fertility model used to derive the weighting factors has a fixed shape, the shape of the distributions generated by the Gompertz model varies systematically as the timing of fertility changes, affecting the results slightly. Thirdly, the coefficients for men sometimes yield poor results for populations with unusual age patterns of fertility or mortality in which male child-bearing is also unusually early or late. They perform well, though, for mean ages at child-bearing of 31-37 years, a range that encompasses most developing country populations.

Bearing these *caveats* in mind, the estimates of men's mortality produced by the new regression method and shown in table 4 are, as expected, significantly more accurate than those yielded by the weighting

TABLE 4. ERRORS IN REGRESSION-BASED ESTIMATES OF THE LEVEL OF MORTALITY (α) FROM PATERNAL ORPHANHOOD, SELECTED MEAN AGES AT CHILD-BEARING ($\bar{M}$) AND CENTRAL AGES OF RESPONDENTS (n)

	$\bar{M} = 31$			$\bar{M} = 34$			$\bar{M} = 37$		
	$n = 10$	$n = 25$	$n = 40$	$n = 10$	$n = 25$	$n = 40$	$n = 10$	$n = 25$	$n = 40$
Mortality									
$\alpha = 0$	−0.059	−0.038	0.061	−0.031	−0.019	0.009	0.024	0.006	−0.062
−0.8	0.065	0.002	−0.055	0.010	0.005	−0.008	−0.020	0.033	0.024
−1.2	0.218	−0.000	−0.035	0.070	−0.074	0.032	−0.095	−0.090	0.088
$\beta = 0.6$	0.067	−0.016	−0.016	0.009	−0.063	−0.000	−0.031	−0.087	0.009
1.4	−0.087	−0.044	−0.091	−0.035	0.010	−0.109	0.047	0.064	−0.151
Growth rate									
$r = 0.5\%$	−0.008	−0.002	−0.040	0.003	0.025	−0.030	0.040	0.059	−0.037
3.5%	−0.031	−0.028	−0.046	−0.038	−0.012	−0.026	−0.019	0.020	−0.022
Fertility									
$\beta_f = 0.675$				0.079	0.085	−0.051	0.087	0.085	−0.057
1	0.012	0.027	−0.041	−0.019	0.005	−0.027	−0.036	0.006	−0.010
1.75	−0.044	−0.054	−0.048	−0.095	−0.084	−0.007			

NOTE: The errors are estimated from the proportions of fathers surviving in populations in which $\alpha = -0.4$, $\beta = 0.95$ and $r = 2\%$, together with $\beta_f = 1$ for $\bar{M} = 34$ years, $\beta_f = 1.3$ for $\bar{M} = 31$ years and $\beta_f = 0.85$ for $\bar{M} = 37$ years, except as explicitly varied. The value of α_f is altered to produce the desired mean age at child-bearing.

factors at extreme levels of mortality. In particular, the errors in the estimates obtained from older respondents are much smaller. The new approach sometimes produces less accurate results than the weighting factors in populations with extreme age patterns of child-bearing, but the errors remain reasonably small.

The errors in estimates of women's mortality produced using the new regression coefficients follow a similar pattern to those in table 4 but are smaller. They are not shown here. As already reported in *Manual X* (United Nations, 1983), however, the regression-based estimates for the older age groups are significantly more accurate than those obtained from the weighting factors in populations subject to extreme levels and patterns of mortality.

When the new coefficients are applied to real data and the results are compared with those from other variants of the orphanhood method, the expected pattern of differences is found. The estimates of women's mortality are similar to those obtained using the weighting factors or other regression methods. When mortality is light, however, the majority of the estimates indicate lower mortality than those arrived at using the weighting factors. At the moderate levels of adult mortality now prevailing in many developing countries, the regression method of estimating mortality from paternal orphanhood usually yields estimates of life expectancy at age 15 that are about one half to one year higher than is indicated by estimates made using the weighting factors.

Conclusions

Concern has been expressed about the robustness of the method that exists for estimating adult men's mortality from orphanhood. This article assesses the procedure. It proposes a new method for estimating men's mortality from the survival of fathers, together with a consistent procedure for estimating women's mortality. Both theoretical considerations and analyses, using simulated data, suggest that the new method yields more accurate estimates than a system of weighting factors.

The analysis presented here indicates that there is an appreciable degree of uncertainty attached to paternal orphanhood estimates, even if the reports from which they are made are accurate. Such errors pale into insignificance compared with the uncertainty that exists about the level of adult mortality in countries where it has to be guessed on the basis of data concerning children (Blacker, Hill and Timæus, 1985). In particular, although it is less robust than the maternal orphanhood method, the paternal orphanhood method is less sensitive than the general tenor of the literature about the orphanhood method would suggest to variation in the distribution of men's ages at child-bearing.

Because of the way that the relationship between parental survival and life-table indices of survivorship changes with the level of mortality, a specification of the model used for estimation which incorporates an intercept term will eliminate a significant source of the bias that affects the sys-

tem of weighting factors. This is particularly important for men and for low-mortality populations. Therefore, a regression-based procedure for estimating adult men's mortality from paternal orphanhood is developed for the first time. A set of estimation coefficients for women's mortality, based on consistent assumptions, is also presented. Estimation of the coefficients for men takes advantage of the recent development of a male standard for use with a relational Gompertz model of fertility.

Because the fathers of respondents of any age tend to be older than their mothers and subject to higher mortality and because of the greater dispersion of male ages at child-bearing, a regression model of the form proposed previously for maternal orphanhood does not prove to be a robust way of estimating mortality from paternal orphanhood. Atypical age patterns of fertility and mortality produce offsetting changes in the proportions of respondents with living parents in two adjoining age groups, relative to the equivalent proportion at the age that divides them. As a result, using data on two age groups to estimate men's mortality from paternal orphanhood yields better results than a model using data from only a single age group.

The kinds of procedure presented here represent a way of estimating adult mortality from orphanhood data quickly and conveniently, with a minimum of data. Yet, appreciable errors in the results can be expected in particular populations where age patterns of child-bearing and mortality differ from those used to derive the coefficients. Given the increasing diversity in demographic terms of developing country populations, it is doubtful that the best way to improve orphanhood estimates further is to produce different sets of estimation coefficients for use in populations with particular characteristics or to attempt to introduce additional parameters that capture, for example, the impact of differences in the variance of ages at child-bearing. Instead, estimates can be calculated readily from equation (1), using fertility and mortality schedules and age distributions that are appropriate for the population under study. The exact methodology can be tailored to the scope, historical depth and accuracy of the data available.

NOTES

[1] The author has discussed this conclusion with Professor Brass, who agrees with this interpretation of the weighting factors for paternal orphanhood presented in the 1973 paper. Note that the weighting factors in *Manual X* (United Nations, 1983) were reproduced from the 1973 paper without modification and are affected the same way.

[2] The errors sometimes change direction as the age of the respondents increases because of the way that the parameters affect the distribution of ages at child-bearing. For example, if the variance of ages at child-bearing is high, more young respondents are orphaned than expected because there are more elderly fathers than anticipated. Mortality will be overestimated. By the same token, however, the rate at which orphanhood increases with the respondents' age then declines to less than is assumed, because most older fathers have died already and more fathers are relatively young than is assumed. Beyond some crossover age, mortality will be underestimated.

[3] Further simulations, not presented here, suggest that empirically common combinations of demographic characteristics are more likely to produce offsetting errors in mortality estimates from orphanhood than errors that compound one another. The largest errors occur in countries with "developing country" mortality and "developed country" fertility or with the opposite combination of characteristics.

[4] As one would expect, given the origins of the data to which the models are fitted, outliers are not a problem.

[5] If the weighting factors are thought of as embodying two stages—first, the application of a multiplying factor, appropriate for age n, to convert the measures of orphanhood into measures of life-table survival, and secondly, the averaging of two of these measures—then:

$$
\begin{aligned}
{}_nP_b &= W_n \cdot {}_5S_{n-5} + (1 - W_n){}_5S_n \\
&= w_n \cdot f_n \cdot {}_5S_{n-5} + (1 - w_n) f_n \cdot {}_5S_n.
\end{aligned}
$$

If an intercept term is added, one obtains:

$$
\begin{aligned}
{}_nP_b &= w_n(a_n + f_n \cdot {}_5S_{n-5}) + (1 - w_n)(a_n + f_n \cdot {}_5S_n) \\
&= a_n + w_n \cdot f_n \cdot {}_5S_{n-5} + (1 - w_n) f_n \cdot {}_5S_n \\
&= \beta_0(n) + \beta_1(n){}_5S_{n-5} + \beta_2(n){}_5S_n.
\end{aligned}
$$

Thus, a regression model using information on two adjoining age groups can be thought of as equivalent to the use of weighting factors with an intercept term.

[6] Note that the error statistics presented here seem relatively large (including those for the maternal orphanhood estimates compared with the error statistics presented by Palloni and Heligman, 1985), simply because a fairly high proportion of the set of populations to which the models are fitted have extreme characteristics. They are a pessimistic guide to the performance of the methods. When female mortality is estimated from these data, using Palloni and Heligman's coefficients, the errors are even greater.

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SOME ASPECTS OF THE SOCIAL CONTEXT OF HIV AND ITS EFFECTS ON WOMEN, CHILDREN AND FAMILIES

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SUMMARY

This article explores the effects of the human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) epidemic on women and children in African countries. We identify some of the relations between women's (and children's) social positions and roles and the impact of HIV/AIDS and argue that social conditions prevailing in Africa (and elsewhere, in parts of South and Central America) not only may increase the exposure to the risk of contracting the infection but also may magnify its deleterious effects on the health and social and economic welfare of women and children. We attempt to provide numerical illustrations of these effects but their validity depends entirely on the validity of assumptions that remain to be verified.

THE PROBLEM

It is well known that the natural history and epidemiological characteristics of HIV in societies where the epidemic is transmitted mainly through heterosexual contact (or a combination of heterosexual and bisex-

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ual contact and IV drug abuse) are quite distinct. Indeed, so sharp is the contrast that it justifies the classification of various patterns of HIV/AIDS world-wide (Chin, Lwanga and Mann, 1989). Equally important but much less studied is the fact that differences in the patterns of the disease are likely to result in differences in the consequences for individuals and social groups. The most salient effects of the epidemic must necessarily be different in societies where it is largely confined among gay men than in societies where it is reproduced among adult males and females alike. Where the HIV/AIDS epidemic is driven by heterosexual contact, women's multiple roles as sexual companions, wives, daughters and mothers are of paramount importance both because they have a non-trivial impact on the characteristic mode of spread of the virus and also because they mediate at least some of the ultimate social and economic effects of the epidemic on other members of the family. Since women's positions and roles are closely associated with the organization and functioning of the family, the study of the conditions and consequences of the HIV/AIDS epidemic requires researchers simultaneously to characterize the family and to discern the impact of HIV/AIDS on its organization and day-to-day functioning.

In this article a simple framework for the study of the effects of HIV/AIDS on women is suggested. The article starts with a brief discussion of the mechanisms through which women's positions and roles determine conditions that increase the likelihood of infection and worsen the deleterious impact of the disease. We then explore the nature of the effects on the family organization but stress those directly or indirectly related to the health status and survival of young children, parents and particularly mothers. To calculate estimates of the magnitude and direction of some important effects we use a combination of models and empirically based procedures. The formal basis of these procedures is explained elsewhere (Palloni and Lee, 1991). Throughout the article the focus is on the conditions generally found in Central Africa, although some of them are also common in the Caribbean and parts of South America. The estimation procedures that we use, however, are quite general and should be applicable to a wide variety of situations.

Faced with a direct threat to the health and survival of some or all of its members, families could select from among multiple adaptations. Some of these may turn out to be quite efficient to muffle the most damaging consequences of the epidemic without altering the main features of the family. Others, however, may do so only at the price of eroding the very foundations of familial relations. We emphasize that the article is not centrally concerned with the most feasible or even most likely adaptations to the new conditions but rather with the nature of the stress exerted by the HIV/AIDS epidemic on family organization. We should also note that there is no such thing as "general conditions" for the spread of HIV and that many generalizations can be accepted only at the price of some loss of realism. The spread of HIV/AIDS, for example, is almost surely sustained by behaviours that vary not just across nations but also across ethnic groups and social classes. To reduce some of the resulting complexity, we

attempt to identify general mechanisms but in the process we blur the actual conditions that make their operation feasible.

Families, women's positions and transmission of HIV

We begin with two simple observations. First, the probability that women become infected with HIV and the nature of the progression of the disease once it is contracted are partially dependent on women's positions and roles within the family. Secondly, the magnitude and direction of direct or indirect effects of HIV/AIDS on susceptible and infected women and children alike is contingent on the familial relations that define the boundaries of permissible behaviour for women and children. In this section we discuss the first observation, and in the succeeding section we explore the implications of the second one.

Exposure and risk

A woman's probability of contracting HIV through sexual transmission at exact age x can be expressed as the product of the probability of being exposed to contact with infected male(s), $G(x)$, and the conditional probability that if exposed to the contact at that age it will result in infection, $F(x)$:

$$I(x) = G(x) * F(x) \tag{1}$$

A woman's lifetime probability of becoming infected is simply the sum of $I(x)$ over all ages in the reproductive span. Although numerical integration of $I(x)$ is at least in principle quite simple, exact measurement is complicated by the fact that $G(x)$ and $F(x)$ are dependent not only on women's characteristics at age x but also on features of the male population that enters in contact with women at that age. The value of $G(x)$ is, in fact, the product of two somewhat distinct quantities which are considered jointly in this article for the sake of simplicity. The first is the probability of being exposed at age x to any sexual contact at all and the second is the conditional probability of being exposed to contacts with infected men. This calculus is unequivocal: the first condition required for women's infection is exposure to any sexual union and the second is that the probability of finding infected men among those eligible for sexual unions be non-zero. If the dominant mechanism of transmission is heterosexual contact, the conditions of women's exposure to the virus must be completely defined by the rules that regulate the timing, age pattern and frequency of contacts with men.

The first factor influencing the magnitude of exposure is the timing of entrance into sexual unions or the onset of sexual activity. In societies where the sexuality of young unmarried women is rigidly controlled to preserve their chastity until marriage occurs, women are discouraged from entering into pre-marital casual relations of their choice. When, simultaneously, there is a significant difference between age at menarche and age at onset of sexual activity, women are spared the risks of exposure to HIV

for a non-trivial length of time. If, however, pre-marital sex is either actively encouraged or tacitly tolerated and the age at onset of sexual activity is indistinguishable from the age at menarche, young females will become exposed to HIV very early in their lives. For the most part societies in Central Africa (but much less so in the Caribbean and South America) have long emphasized, although never rigidly enforced, the chastity of young unmarried females and preserved a regime of early first marriage. However, there are indications that gradually, under the onslaught of westernization, the foundations of this regime are crumbling in many places and that pre-marital sex—if not explicitly prescribed—is at least widely practised (Larson, 1990; Caldwell, Caldwell and Quiggin, 1989; Oruboloye, Caldwell and Caldwell, 1990; Oruboloye, 1990). Although intensification of liberal teenage sexual activity and growing disregard for tradition undoubtedly account for part of the increased exposure, an equally important factor is the strong parental incentive for anticipating the timing of entrance of daughters into sexual unions. Engaging daughters (or granddaughters) in more or less steady sexual relations with older men could be a relatively efficient means to secure direct payments or deferred protection in risky environments (Reining, 1990; Swantz, 1985).

The lifetime reductions in the risk of contracting HIV attributable solely to postponement of the initiation of sexual contacts can be substantial. Using a very simple expression for the female age-specific force of infection, we calculated the potential reduction in the lifetime probability of becoming infected that can be achieved by postponing initiation of sexual activity from age 10 to age 20 in intervals of two or three years. Table 1 displays the estimated probabilities that a female will not become infected from the onset of initiation to about age 40 under alternative scenarios for infectivity (the probability of infection per sexual contact) and for five different ages of initiation of sexual contact. Not unexpectedly when infectivity is very low, the gains from delaying entrance into sexual unions are trivial. For example, if infectivity per contact is .001, the probability of avoiding infection is .976 when the age of initiation is 10 and

TABLE 1. FEMALE PROBABILITIES OF REMAINING UNINFECTED FROM THE AGE OF INITIATION OF SEXUAL UNIONS TO AGE 40 FOR ALTERNATIVE REGIMES OF HIV

	Age of initiation				
Infectivity[a]	*10*	*12*	*15*	*18*	*20*
.001	.976	.977	.979	.981	.983
.010	.787	.794	.810	.830	.847
.100	.091	.101	.122	.155	.190

Source: The procedures to calculate these figures appear in A. Palloni and Y. J. Lee, Methods for the estimation of demographic effects of HIV/AIDS in Africa. Unpublished manuscript (University of Wisconsin, Center for Demography and Ecology, Madison, 1991).

[a]Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.

.983 when the age of initiation is 20. However, when infectivity attains the highest value in the table (.10) a 10-year postponement virtually doubles the probability of escaping infection. For intermediate values of infectivity (.01), a 10-year postponement will increase the probability of remaining HIV-free until age 40 by about 8 per cent. The increases are even larger if compared with the maximum that can be gained in any particular regime. Thus, in a regime where infectivity is .01, the maximum gain can only be around .213 (1.0-.787). The contribution to this maximum that is attributable to a postponement of, say, five years (from age 10 to age 15) is about 11 per cent ((.810-.787)/.213). The values in the table were calculated assuming that the levels of male HIV-prevalence range from .01 at ages 15-19 to a maximum of .05 at age 30. Naturally, if the levels of seroprevalence are higher, the gains attributable to postponement could also increase, but this would depend on the details of the age pattern of increase of HIV prevalence (Palloni and Lee, 1991).

Exposure to sexual contact is relevant for HIV infection only if, once it begins, it leads to a non-zero probability of contacting infected male(s). The likelihood of sexual contact with infected men is a function of the group within which a male partner is selected.[1] An important characteristic determining the infective status of males is their age. By and large, male sexual activity increases with age. In Africa, where the epidemic has been growing steadily, a non-trivial part of this increase is due to the fact that opportunities are a function of power conferred by seniority, accumulated assets and freedom from restrictive village rules. As a result relatively older men are more likely to be infected than younger ones.[2] Thus, from the point of view of young women, the societal rules that result in a large gap between the ages of spouses or sexual partners will increase their lifetime probability of contracting infection. This is the case in polygynous societies where the age differences among spouses are maximized by the tendency of older males to constantly search for younger females as prospective spouses and gradually replace older ones (Locoh, 1988; Caldwell, Caldwell and Quiggin, 1989; Lesthaeghe and Surkin, 1988). But although polygyny is an efficient mechanism to maximize the age difference between spouses, it is by no means the only one. A rigid adherence to rules of bride wealth in monogamous patrilineal societies undergoing an economic crisis will invariably result in males delaying marriage until a time when sufficient assets can be accumulated (Larson, 1990). Simultaneously, a strong preference for virgin brides will encourage selection among the youngest and thus lead to an increase of the average age gap between spouses.

The effects of the age gap between sexual partners depend on the prevailing HIV regime. Table 2 displays estimates of the probability of *not* becoming infected between ages 15 and 25 for alternative values of the age gap in three HIV regimes. In all cases we assumed that the age of onset of sexual contacts for both sexes is 15. As before, the gains are proportionately larger in HIV regimes of higher infectivity. A reduction of 15 years in the age gap between partners in a regime with an intermediate infectivity leads to an increase of about 24 per cent in the probability of

TABLE 2. FEMALE PROBABILITY OF REMAINING UNINFECTED IN THE AGE INTERVAL 15-25 FOR ALTERNATIVE VALUES OF THE MEAN AGE GAP BETWEEN SPOUSES AND FOR ALTERNATIVE REGIMES OF HIV

	Mean age gap			
Infectivity[a]	*0*	*5*	*10*	*15*
.001	.989	.984	.975	.963
.010	.895	.846	.779	.668
.100	.330	.190	.083	.024

Source: A. Palloni and Y. J. Lee, Methods for the estimation of demographic effects of HIV/AIDS in Africa. Unpublished manuscript (University of Wisconsin, Center for Demography and Ecology, Madison, 1991).

[a]Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.

avoiding infection. In contrast, in the regime with highest infectivity, the gains multiply the probability of remaining uninfected more than tenfold. By and large, the gains attributable to a reduction of one year in the age gap are somewhat higher than the gains attributable to a one-year postponement of the age of initiation of sexual exposure (see table 1).

A third factor implicated in the female exposure to HIV is the level of age-specific prevalence among males. This is, in turn, dependent on several conditions. Among married males it is the frequency of extramarital relations that largely regulates the exposure to the infection. Adulterous relations established by wives are also a conduit for it, but, although not altogether uncommon, in most African and Latin American societies, female adultery is seen as morally wrong and generally punished. Male adultery, although seldom sanctioned (Davis and Whitten, 1987; Southwold, 1973; Hagan, 1983), is regarded as less offensive. In many African societies, for example, husbands' extramarital activities are, if not directly encouraged, at least widely tolerated, particularly during the long and rigidly enforced periods of post-partum abstinence (Caldwell, Caldwell and Quiggin, 1989; Larson, 1989; Oruboloye, 1990; Obbo, 1979 and 1987). Also, at least among new urbanites, the emergence of informal but regular attachments to a few younger women in lieu of formal but considerably more expensive polygynous arrangements is conducive to higher exposure for males and hence for their partners. Among younger unmarried males, early sexual contacts—particularly, but not solely, with prostitutes—are not greatly discouraged and appear to be on an increasing path (Oruboloye, 1990; Larson, 1989). The consequence is an increase in single males' risk of contracting infection prior to marriage. Finally, the frequency of males' extramarital relations is also a function of population movements and in particular of male voluntary migration, involving more or less extended periods of spouse separation. When men leave rural areas without their families, the chances of their entering into multiple casual contacts with infected (urban) females are considerably increased and, when circular or return migration is part of the population flows, so is their wives' exposure to the HIV infection.

The increase in women's risks of contracting HIV when husbands or partners have relatively frequent extramarital sexual contacts with high-risk women can be staggering. Table 3 displays the estimated probabilities that a susceptible woman will contract the infection over a five-year period following the onset of a union of a healthy male and female in the age group 20-25. We assume that only males engage in extramarital relations, that the frequency of sexual contacts between spouses is roughly equal to the average observed in empirical populations (about 48 per year), that male-to-female infectivity is three times the value of female-to-male infectivity and, finally, that no protection whatsoever is used during sexual intercourse. Note that even when infectivity is very low, the probability that a married woman will contract HIV from the husband could reach a level of about .07 in only five years. Under slightly higher infectivity, the spouses of the most sexually active males face sure infection in the short span of five years. It is difficult to find a better example of the consequences for women of a regime where males enjoy ample sexual liberties while women have little if any say over the regulation of sexual contacts in which they participate.

A fourth factor that influences female exposure to the infection in parts of Africa involves a more subtle but equally powerful relation between women's roles and socio-economic characteristics of the family. In areas where male migration is pervasive, women and children are left behind as anchors in an extended family network to secure property rights. However, oftentimes their economic support depends more on women's and children's labour than on migrants' remittances. Adulterous relations involving a migrant's wife could be a device of last resort to secure payments and economic support as insurance in environments where the odds are that there will be insufficient remittances or none at all. Married women's adulterous relations may at times be condoned since they represent a way to avoid deprivation of the right to reproduce in societies where women's reproductive role overwhelms her roles as a wife (Caldwell, Caldwell and Quiggin, 1989). Such conditions also offer strong incentives to anticipate the entrance of young daughters into a sexual market. Thus, the joint influence of economic conditions, family relations, cultural values attached to high fertility, and the family's management strat-

TABLE 3. FEMALE PROBABILITY OF CONTRACTING HIV IN A FIVE-YEAR PERIOD WHEN THE SPOUSE HAS EXTRAMARITAL SEXUAL CONTACTS

	Husband (partner)'s number of sexual contacts per year			
Infectivity[a]	*1*	*12*	*24*	*48*
.003	.0017	.0200	.0380	.0720
.030	.0920	.6720	.8820	.9800
.300	.7220	1.000	1.000	1.000

[a] Levels of infectivity from male to female (approximately three times the levels of infectivity from female to male).

egies in times of crisis facilitate younger and older women's exposure to infection (Zalduondo, Msmanga and Chen, 1988).[3]

Finally, the norms regulating remarriage and the sexual behaviour of widows are also an important mechanism of infection but, unlike others reviewed above, the effects may cut both ways and increase exposure of either males or females. Societies with extensive practice of levirate strongly encourage sexual contacts between a recently widowed woman and her brother(s)-in-law. The pervasive practice of levirate encourages the spread of HIV since it increases the number of partners with whom sexual contacts are established. When adult mortality is greatly influenced by AIDS, chances are that a recent widow would have been exposed to HIV (since the dead husband is more likely to have died of AIDS) and, as a consequence, that she will be exposing her brother-in-law rather than the other way around. But, the opposite situation is also possible. Thus, levirate is another practice that increases the risk of women, although not always more than it increases the risk of men. Paradoxically, reliance on this social practice, which serves to reinforce the authority of elders and to strengthen the lineage, contains the seeds of destruction of the family itself.

In summary, the function $G(x)$ is a joint outcome of factors that may inhibit or enhance entrance into sexual unions and of those that directly regulate the frequency of contacts with and the nature of the mixture of susceptible and infected male partners. These factors are at least in part dependent on the rules regulating the formal and informal exchange of women as sexual partners and companions, the degree of monopoly that older males may have over the exchange of women, family norms and practices to control the circulation of women among members of the most immediate family, and the unspoken or explicit rules that define the behaviour of females and males in their sexual practices.

Although HIV in Africa is predominantly transmitted through sexual contact, injections and transfusions play more than a trivial role. At least in some societies women (and children) more than adult males are exposed to these alternative sources of infection. First, radical (pharaonic) circumcision, which is widely practised in parts of East Africa, directly affects exposure by subjecting women to contact with potentially infected instruments. In the long run, however, the resulting trauma leads to tissue damage that produces tearing and bleeding during vaginal intercourse. This increases the likelihood of choosing sexual practices that entail higher risks of infection (Zalduondo, Msmanga and Chen, 1988). Secondly, a long child-bearing period punctuated by numerous pregnancies increases the chances that higher order deliveries will result in acute anaemia, particularly if women are already affected by other debilitating diseases, such as malaria, or suffer mild or chronic malnutrition. Since the treatment of anaemia frequently requires blood transfusions and since blood products may not be subjected to screening, child-bearing itself leads directly to increased exposure to HIV. Although the effects of these two mechanisms are difficult to document, it is at least possible that even non-sexual means of transmission affect females more than males and that the inequality is a

result of societal definitions of women's roles as mothers, producers and guardians of property and assets.

Just as the quantity $G(x)$ can be decomposed into two parts—timing of exposure to sexual contact and probability of encountering an infected male—so can the quantity $F(x)$, the conditional probability of becoming infected, be broken down into a small number of elementary components, each of which is at least in part affected by women's roles within the family.

The first of these components is the probability of contracting the infection while using protected sexual intercourse. The second is the probability of using protection and of being exposed to the enhancing factors.

In spite of the fact that the findings of empirical research on the matter are far from being definitive, there is some evidence supporting the idea that, *ceteris paribus*, the probability of male-to-female transmission is higher than the probability of female-to-male transmission. This inequality has been largely attributed to differences in the rate of exchange of fluids and to the sheer volume of the virus that can be hosted in semen in contrast to vaginal fluids. In the absence of offsetting factors, this difference alone could account for a heavier presence of HIV among females in some age groups (Quinn and others, 1986). If this disparity is not always empirically observed, it is only due to the compensating influence of sex differentials in sexual activity.

One conditon that exacerbates the differences in infectivity is the prevalence of sexually transmitted diseases (STD). Although the empirical evidence is murky at best and the direction of causality somewhat unclear, it is widely believed that sexually transmitted diseases enhance infectivity (Plummer and others, 1987; Kreiss, Carael and Meheus, 1988; Fleming, 1988; Mann and others, 1987; Van de Perre and others, 1987). Since the conditions of transmission of sexually transmitted diseases are similar to those regulating the sexual transmission of HIV, the remarks made above about factors influencing exposure to sexual contacts with HIV-infected men apply also for the sexually transmitted diseases. However, because some sexually transmitted diseases in females are asymptomatic and because there are differences in social acceptance and in the various stigmas attached to female and male sexually transmitted diseases, females are by and large less likely to seek adequate medical treatment and more likely to harbour the infections for longer periods of time. The net consequence of this is that an HIV-infected male is more likely than he would otherwise be to pass on the infection to a susceptible but STD-infected female. A symmetric relation holds if the female and not the male is an HIV-carrier. If others things are equal, we should expect that in areas with high STD prevalence, the female/male ratio of HIV prevalence exceed levels observed in areas that are STD-free.

Male/female status inequalities with a direct effect on the risks of contracting HIV involve disparities in the control over the initiation and regular establishment of sexual unions, their consummation and their termination. We have seen already the impact of changes in the age of initiation

and the age gap between partners. We will now show the effects of the inability to practise protected sexual intercourse. It is known, for example, that the use of condoms and spermicides may reduce the probabilities of transmission. However, although knowledge about this fact is quite widespread, the use of condoms remains dismally low in Africa, parts of the Caribbean and South America. These protective measures are viewed by males as being strongly associated with infertility as well as loss of pleasure and spontaneity. And as long as males are empowered to establish the boundaries of what is and what is not permissible in sexual contacts, the use of the protection paraphernalia will continue to be dangerously low.[4] As an illustration, in table 4 we have calculated the probabilities of infection under different scenarios. We have assumed that men have variable frequency of extramarital relations with prostitutes and that 50 per cent among the latter are sero-positive. In societies where the epidemic is driven by men who have extramarital relations with prostitutes, the frequency of extramarital contacts is directly associated with the levels of prevalence (Palloni and Lamas, 1991). Thus, changing levels of adult HIV prevalence is directly related with (and can be indexed by) the frequency of extramarital relations. The levels of efficiency refer to the conditional probability that a protective device (condom, spermicide) will block the transmission of the virus from the infected to the susceptible individual. Table 4 was constructed assuming that if protection was used, all sexual relations were protected at the given level of efficiency. The table displays the probabilities of becoming infected during a five-year period for females whose spouse engages in extramarital relations with variable frequency, and for several levels of efficiency. The figures in the three panels

TABLE 4. FEMALE PROBABILITY OF BECOMING INFECTED DURING A FIVE-YEAR PERIOD FOR ALTERNATIVE LEVELS OF EXTRAMARITAL ACTIVITY AMONG ADULT MALES AND FOR TWO LEVELS OF EFFICIENCY OF PROTECTION

	Frequency of extramarital relations per year			
Infectivity[a]	*1*	*12*	*24*	*48*
		Level of efficacy of protection is .00		
.003	.0017	.0200	.0380	.0720
.030	.0920	.6720	.8820	.9800
.300	.7220	1.0000	1.0000	1.0000
		Level of efficacy of protection is .50		
.003	.0004	.0052	.0104	.0200
.030	.0297	.2960	.4890	.7090
.300	.4690	.9999	1.0000	1.0000
		Level of efficacy of protection is .90		
.003	.0000	.0002	.0007	.0011
.030	.0017	.0197	.0385	.0736
.300	.0092	.6720	.8820	.9800

[a] Levels of infectivity from male to female (approximately three times the levels of infectivity from female to male).

correspond to levels of efficiency of the means of protection equal to .00 (no use of protection), .50 and .90. Note that the reduction induced by an efficient means (compare the first to the last panel) is quite considerable at *all* levels of infectivity. At intermediate levels of infectivity, for example, the probabilities of infection are reduced more than tenfold regardless of the frequency of extramarital contacts. But even an inefficient means of protection is better than none at all: a comparison of the first and second panels shows that at all except the highest levels of infectivity and extramarital activity, the probabilities are reduced by about half. This illustrates the changes that can be achieved through adoption of some protection even in the absence of behavioural changes regarding the selection of sexual partners.

The progression of infection

HIV has an insidious character, for its presence may remain unnoticed for very long periods of time before the gradual collapse of the immune system sets in. The median time elapsed from the onset of infection to the onset of full-blown AIDS is estimated to vary between five and 15 years. And as new studies consolidate information from longer follow-ups, the estimates of the median incubation time increases. Furthermore, while we know with some certainty that, once contracted, HIV will remain in the organism, there are considerable doubts about the ultimate probability of eventually developing AIDS or, equivalently, about the fraction of the infected population that will ever develop AIDS. The main consequence of a protracted period of incubation is that infected individuals can remain active and infective for a long period of time and will thus increase the reproductive value of the HIV virus.

Although the potential role of individual heterogeneity in the risk of developing AIDS has been recognized, less attention has been paid to the fact that females and males are affected by conditions that predispose them to potentially different incubation processes. First, higher order pregnancies among sero-positive women are suspected of initiating secondary processes that trigger a full-blown attack of HIV on the immune system (Pinching, 1987; Fleming, 1988; Badi and others, 1990). If this association were to be confirmed, fecund but sero-positive women could experience shorter incubation times than the average infected population. Since AIDS is always fatal, this amounts to saying that the expected lifetime for infected and fecund females is shorter than for infected males. This mechanism may have a non-negligible influence on the natural history of HIV in societies where there is a strong emphasis on high fertility and birth control is generally discouraged. In these cases, the prevention of a pregnancy is not just a way of avoiding perinatal infections and protecting unborn infants but also of prolonging an infected woman's life. Secondly, infected women in Africa and parts of the Caribbean and South America who deliver healthy babies breast-feed for relatively long periods. Under conditions of poor nutrition, breast-feeding is an added stress which, even in an HIV-free environment, may increase women's susceptibility to infec-

tious and parasitic diseases. That increased stress, which may be rather innocuous in the absence of HIV, has, however, potentially devastating consequences for women infected with the virus. It is suspected, although not decisively proven, that repeated attacks by other infectious and parasitic diseases can lead to shorter incubation times (Plummer and others, 1987; Curran and others, 1985; Fauci, 1988). Lactating sero-positive women could thus be at higher risks of experiencing a shortened disease progression. Overall then, the child-bearing experience among sero-positive women could induce shorter incubation and, hence, shorter survival times.

A third but admittedly more questionable male/female differential in the progression of HIV has to do with the role of reinfection. In the medical literature on the subject, some evidence suggests that repeated reinfection could accelerate the breakdown of the immune system (Fauci, 1988). As long as infected women remain in positions that force them to exchange sex for favours and remuneration or to acquiesce to kin's demands in order to reinforce men's authority, and as long as these norms, behaviours and practices keep women fully exposed to infected men, repeated reinfection will be more likely and so will be the consequent shortened female survival time.

In summary, constraints in the choices that women face produce conditions for a typical incubation process with shorter median incubation times and possibly lower variances. This is the result of a somewhat unexpected interaction between immune system dysfunction and societal rules and mores that increase the likelihood that women will engage in risky behaviour.

Children, females and families and the impact of HIV/AIDS

Social scientists and epidemiologists alike have already alerted us to some of the most visible consequences of the HIV/AIDS epidemic (Carballo and Carael, 1988; Muhondwa and Carballo, 1988; Miller and Rockwell, 1989; Fleming and others, 1988). A pervasive and prolonged presence of HIV will surely lead to mortality increases, particularly among adults and young children. A more insidious consequence is that such increases may not occur across the board but could have a differential impact on social strata or classes. The available evidence is simply too spotty to identify the social groups among which the impact will be harshest. It is likely, however, that they will vary across countries and ethnic groups.

The HIV/AIDS epidemic, however, can have potentially more subtle consequences for individuals and families than those implied by group-selective increases in mortality. First, in regimes where the HIV/AIDS epidemic is transmitted heterosexually, chances are that if one member of a relatively steady union or couple contracts the infection, the other also will. However, children who are already born and elderly people who are members of the same co-resident group as the HIV-parent(s) will not

necessarily be exposed to increased risks. There is a unique asymmetry in the spread of HIV/AIDS that spares some members of a family while threatening directly the health of others. We will show that this asymmetry has important consequences for residential arrangements. Secondly, unlike the catastrophic epidemics that devastated pre-industrial societies, HIV/AIDS leads to a gradual rather than sudden deterioration of the infected individual. As noted above, a long incubation period almost surely raises the potential for transmission of the virus. But it also increases the economic, social and psychic costs for those who are infected and for those who are members of their immediate families. Again, this is a unique feature of HIV/AIDS, one that may overshadow the increases in mortality.

Adult mortality excesses: orphanhood, widowhood and coresidence

When HIV is transmitted mainly through heterosexual contact, the maximum levels of sero-prevalence are generally found among young adults and very young children (Quinn and others, 1986). In the absence of countervailing influences, the consequent mortality increases will invariably affect these same groups but at slightly older ages. The magnitude and age patterns of increases in adult mortality will have three distinct consequences, explored below.

Orphanhood

The levels of paternal and maternal orphanhood at young ages (between 0 and 10 or 15) will rise, reflecting the increased mortality of young parents. The assessment of increases in the levels of orphanhood is at its initial stages (Preble, 1990; Hunter, 1989), and much remains to be done in the areas of both empirical estimation and forecasting. In this article we offer an alternative procedure that provides illustrative results.

To calculate expected levels of orphanhood we use a model-based procedure described elsewhere (Palloni and Lee, 1991). Table 5 displays

TABLE 5. PROBABILITIES OF BECOMING A MATERNAL ORPHAN (MO), A PATERNAL ORPHAN (PO), OR BOTH (OO) BY AGES 5 AND 10 IN SEVERAL HIV REGIMES, FOR CHILDREN BORN TO HEALTHY PARENTS

	Child of 5 years			*Child of 10 years*		
Infectivity[a]	*MO*	*PO*	*OO*	*MO*	*PO*	*OO*
.00[b]	.030	.042	.001	.059	.085	.005
.010	.038	.051	.003	.113	.134	.031
.100	.067	.083	.006	.199	.225	.090

[a] Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.

[b] No HIV.

estimated values of the probabilities that a child who is born healthy to healthy parents will survive to exact ages 5 and 10 and experience maternal orphanhood, paternal orphanhood or both. In all cases the estimates were calculated assuming that mortality of parents and children are independent but that the forces of mortality of parents are dependent on each other and that such dependency is completely explained by HIV/AIDS. Comparing the scenarios with infectivity equal to zero to the other two provides an idea of the magnitude of the impact of HIV/AIDS. Under normal mortality conditions, not more than 5 per cent of children will experience maternal orphanhood before age 10. But its prevalence is considerably increased (to 13 and 25 per cent) under two alternative scenarios for HIV.[5] Naturally, the seriousness of the problem multiplies if one uses as a benchmark the fraction of children whose mothers will be HIV-infected (rather than deceased) at ages 5 and 10. Elsewhere (Palloni and Lee, 1991) we have estimated that under conditions similar to those used in the calculation of table 5, over 45 per cent of the children surviving to age 10 whose mothers were already infected with HIV will continue to have HIV/AIDS mothers and about 30 per cent of those whose mothers were susceptible will have an HIV/AIDS-infected mother.

Although the growing incidence of orphanhood should in theory affect male and female children alike, the actual impact will depend on the strategies that families adopt to cope with excess adult mortality. Increased reliance on fosterage, for example, may be one of the many available resources to families directly affected by HIV/AIDS. But if children living under fosterage arrangements experience higher mortality (Bledsoe and Brandon, 1989) and if there are strong female preferences in the fosterage arrangement, as documented in some parts of Africa (Page, 1988), female children may end up experiencing the brunt of the crisis. Barring the deployment of massive social resources to absorb the growing burden of orphanhood, chances are that the rates of school drop-out, child-labour participation and outright abandonment will increase and, together with them, the vulnerability of the youngest generation. Whether or not women will disproportionately suffer the consequences will depend on the pervasiveness of male preferences.

Widowhood

Increases in adult mortality will also lead to a growing incidence of widow(er)hood. Depending on the patterns of spread of HIV and on the resulting sex differential in HIV prevalence, widowhood may increase more rapidly than widowerhood or vice versa. When, as appears to be the case in many parts of Africa, the HIV epidemic is driven by male contact with a core of sexually active females, a typical sequence of events could be as follows: the male becomes infected, develops AIDS and dies shortly thereafter. The spouse may or may not become infected but if she does, her expected survival time will be considerably shortened. Thus, the same forces that increase the incidence and anticipate the timing of widowhood will operate to offset the tendency towards longer duration of widowhood.

This is shown in the estimated duration of widowhood in table 6a. Table 6b displays the estimated (non-cumulated) probabilities of being widowed at various durations of marriage. The results in the table are based on the assumption that both partners were healthy at the onset of the union and that, on average, women initiated the union at age 25 and males at age 28. Since the prevalence of HIV is highest at these ages, the results in the table are upper bounds for the probabilities of widowing.

The future consequences of potentially large increases in widowhood could be staggering. How will societies cope with it? Will remarriage rates increase? Or will remarriage decrease as fear of contracting the diseases spreads, thus restricting the pool of potential partners? What effects will this have on widows from polygynous unions? And what will be the fate of widows in monogamous unions with potentially larger numbers of children to care for and less resources to do so? Will the lineage absorb the losses? Will the brunt of the burden fall on women's shoulders, since neither the extended family, the fosterage system, nor the State can accommodate quickly and efficiently to the sudden change?

Coresidence

The third consequence of the increase in adult mortality is a result of the other two: as young adult mortality tends to rise, a relatively powerful

TABLE 6A. EXPECTED DURATION OF WIDOWHOOD IN A 20-YEAR PERIOD FOR SEVERAL HIV SCENARIOS AND FOR FEMALES WHO START THEIR UNION AT AGES 15, 20 AND 25

	Age at initiation of union		
Infectivity[a]	*15*	*20*	*25*
.00[b]	9.2	8.91	8.70
.010	5.8	5.14	5.13
.100	4.3	4.80	4.75

[a] Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.
[b] No HIV.

TABLE 6B. PROBABILITIES OF BEING A WIDOW IN THE AGE INTERVAL 30-45 FOR HEALTHY WOMEN FOR SEVERAL HIV SCENARIOS

	Age of the woman			
Infectivity[a]	*30*	*35*	*40*	*45*
.00[b]	.04	.08	.13	.17
.010	.05	.13	.20	.22
.100	.08	.23	.26	.23

[a] Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.
[b] No HIV.

demographic constraint is imposed on family composition. When the preferred arrangement is the co-residence of three generations, an upward pressure in young adult mortality will increase the prevalence of co-resident arrangements where some members of the intermediate generation are missing. Barring drastic changes in remarriage rules, the dominance of the parental generation will be weakened and the relations between children and grandparents will become more influential. By the same token, however, the older generation stands to experience important economic setbacks. In fact, in societies where the joint residence of parents and grandparents is one of the adjustments that facilitates the support of the elderly, an increase in young adult mortality will lead to a deterioration in the efficacy of the residential arrangement. Since normal sex differentials in mortality at older ages favour females, it is likely that those who stand to lose most in the presence of HIV are elderly women: not only will they be burdened with the care of the very young but they will also experience a drastic deterioration in their control over the materials and social means to cope with it.

Quantitative assessment of this effect requires rather elaborate models. We have, however, utilized an approximate procedure that allows us to calculate the probability that a three-generation family arrangement will be reduced to only two generations, children and grandparents (with or without grandfather present). Table 7 displays the proportion of three-generation families that will keep at least some members of the youngest and oldest generations but none of the intermediate one. The calculations assume that one follows a "cohort" of coresident individuals, that at the onset the parental couple consisted of a healthy woman aged 25 and a healthy man aged 28 and, finally, that the mortality of children and grandparents is independent of the mortality of parents.

In the absence of infection, the proportion of households that will be reduced to two generations by the end of the 15th year after the onset of the process is about .014. When infectivity is higher, between 11 and 26

TABLE 7. NUMBER (OUT OF 1,000) OF CORESIDENTIAL UNITS WITH THREE GENERATIONS WHICH BECOME A CORESIDENT UNIT INVOLVING ONLY THE OLDEST AND YOUNGEST GENERATIONS

	Years from the start of the projection					
	5		*10*		*15*	
Infectivity[a]	*Both grand-parents*	*Only grand-mother*	*Both grand-parents*	*Only grand-mother*	*Both grand-parents*	*Only grand-mother*
.00[b]	.99	.010	4.38	.57	9.54	3.12
.010	1.97	.020	2.63	.34	75.65	26.77
.100	5.92	.050	2.88	1.02	79.93	63.66

[a] Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.
[b] No HIV.

per cent of the households will lose the intermediate generation. Thus, another consequence of the sharp deterioration in adult survival is an increase in the chances that coresidence of children and grandparents becomes an important household arrangement. And if so, the vulnerability of grandparents—particularly of grandmothers—will inevitably transfer to the children.

Increases in infant mortality may exacerbate women's health problems

In societies with a strong emphasis on descendants and a rigidly enforced norm of high fertility, a sudden upsurge in infant mortality may trigger an adaptive response towards even higher fertility. This can occur in two different ways. First, an increase in infant mortality will, on average, shorten the period of post-partum amenorrhea and, in the absence of offsetting changes, will result in an increase in the number of children ever born. The increase will be particularly visible where a variety of practices such as breast-feeding and post-partum abstinence ensure, on average, long interbirth intervals. The second mechanism is a volitional replacement response on the part of couples. If emphasis on a large number of descendants implies a high, though vaguely defined, fertility target, detectable increases in infant and child mortality in the community at large and for a couple in particular could lead to fertility increases in order to ensure that the target is met.

If it occurs at all, the increase in fertility is likely to be short-lived and fully offset by the increase in infant and child mortality and the reduction in the reproductive span precipitated by higher female mortality. However, even a transient fertility increase may have potentially serious consequences for women of reproductive ages. By and large, fertility increases in areas with already high fertility norms will occur at the price of a deterioration of maternal health in general. But perhaps more importantly, those who will pay a higher health and survival toll are women who are already infected with HIV and who are more likely to experience child losses directly.

Do these effects amount to much? First, in the absence of further exogenous improvements in child health, the potential increase in early child mortality could be quite substantial. Table 8 displays the estimated increase in child mortality (below age 5) that will occur under a variety of scenarios regarding the probability on perinatal transmission, excess mortality of sero-positive children and the levels of sero-prevalence among pregnant mothers. Conditions approximating those in some of the most seriously affected countries in Africa (Burundi, Rwanda, Uganda, United Republic of Tanzania) suggest that the levels of prevalence among pregnant mothers hover around 20-25 per cent. Under the most conservative assumptions, these sero-prevalence levels should result in an increase of about 8 per cent in child mortality. Under the worst scenario, the increase could be of the order of 50 per cent.

Secondly, the effects on the short-term fertility trends will involve increases, the magnitude of which will depend on the average reduction in

TABLE 8. PROPORTIONATE INCREASES IN CHILD MORTALITY BELOW AGE 5 FOR SEVERAL HIV SCENARIOS

	Level of sero-prevalence among pregnant women					
	.10		*.252*		*.50*	
Probability of perinatal transmission	20	50	20	50	20	50
Low HIV/AIDS mortality excess (1.50 times the normal force of mortality)	1.03	1.08	1.08	1.20	1.15	1.38
High HIV/AIDS mortality excess (5 times the normal force of mortality)	1.08	1.20	1.20	1.50	1.40	2.00

the post-partum amenorrhea period. An increase of child mortality of around 25 per cent (average of the worst and best scenarios) should decrease the post-partum period by about 30 per cent and the consequent increase in fertility should be of the order of 9 per cent (Jones and Palloni, 1990). Although this increase will not be high and sustained enough to bolster the long-run rates of natural increase, it will inflict severe setbacks on the health of the HIV-infected women.[6]

The bottom of the iceberg: distribution by health status

Excessive emphasis on the upward pressure on adult and child mortality diverts attention from a more devastating effect of the HIV epidemic. Relatively long incubation periods combined with environments that predispose the population to repeated viral, bacterial or protozoal infections could lead to health deterioration among the infected population in a measure that is not experienced in areas with different environments (WHO, 1987; Fleming, 1988). It is possible that the health impairment induced by the infection itself (prior to the onset of AIDS) will be powerful enough to prematurely disable productive individuals. Although the evidence is not fully convincing, some studies do report increased incidence of disorders among those who are asymptomatic at all durations of infection (Taelman and others, 1990; Riera and others, 1990; Melchior and others, 1990).

If the relation between HIV and health deterioration is indeed strong, a proper assessment of the economic losses induced by HIV and the evaluation of the impact on the family organization should take into account not just the larger prevalence of orphanhood and widowhood but also the stark shift in the distribution of members of the family by health status. Damage to the family economy and to the care systems for children and the elderly may begin much earlier than at the time of the death of one or both parents. In fact, it may start shortly after the onset of infection in any one of them. If so, the losses incurred by the family will be spread

over a longer period of time and their magnitude will be higher than those implied by the statistics on orphanhood and widowhood alone.

Using a projection procedure involving multiple states (Palloni and Lee, 1991) we calculate the distribution of parents by health status several years after the birth of a healthy child. Table 9 shows the estimated proportion of children by health status of parents for several HIV regimes. If contracting HIV implies quick deleterious health effects and harbours the potential for immediate economic losses, the figures displayed in this table are much more telling than those related to orphanhood alone: approximately 20 per cent of all children born to a healthy female aged 25 (roughly the average age at child-bearing) in an HIV regime with intermediate levels of infectivity will have at least one HIV-infected parent by age 5. By their tenth birthday, the figure will have doubled.

Even if these figures are reasonably accurate, their exact meaning is fuzzy. If the health impairment in a asymptomatic individual is trivial, table 9 adds nothing to what we knew already from a reading of table 5. If, on the other hand, the health impairment is significant, though possibly variable across individuals and social strata, then the figures in table 9 contain more information than those in table 5. This is because the families' ability to generate resources for the care of children and the elderly is jeopardized by health-induced disability as well as by death. Perhaps even more telling is the average duration of the health impairment—namely, the average length of time in the life of a child (or an elder) during which one or both of the parents will experience illness. Elsewhere we have shown that the analysis of durations (instead of prevalence) can reveal other important features of the problem (Palloni and Lee, 1991).

TABLE 9. PROPORTION OF CHILDREN WITH AT LEAST ONE PARENT ILL (I) AND WITH BOTH PARENTS ILL (II), FOR SEVERAL HIV SCENARIOS AND SELECTED MOTHER'S AGE AT BIRTH

		Age of mother at birth					
		15		*25*		*35*	
Years[a]	*Infectivity*[b]	*(I)*	*(II)*	*(I)*	*(II)*	*(I)*	*(II)*
5	.00[c]	.00	.00	.00	.00	.00	.00
	.010	.007	.005	.205	.170	.289	.216
	.100	.063	.050	.670	.589	.739	.613
10	.00[c]	.00	.00	.00	.00	.00	.00
	.010	.091	.071	.420	.363	.338	.295
	.100	.407	.333	.828	.772	.788	.710

[a] Years elapsed since the start of the projection.

[b] Infectivity refers to the conditional probability that an infected individual transmit the infection to a susceptible one in one single contact. The levels used here refer to female-to-male probabilities.

[c] No HIV.

Can the potential responses erode further women's positions?

It is likely that societies seriously affected by HIV will improvise alternative adaptive responses to accommodate to health deterioration, increased mortality and possibly increased fertility. Although it is difficult to predict in advance which one will occur where, some responses will surely involve the transformation of traditional practices and could end up favouring conditions for the further entrenchment of HIV.

Fosterage is a widespread institution in parts of Central and West Africa. It involves the circulation of young children from parent to foster parents who become responsible for providing subsistence, instruction and training (Page, 1988; Isiugo-Abanihe, 1985). This practice tends to sustain high fertility regimes by spreading the costs of child-rearing among several groups (Page, 1988) and could potentially absorb cost increases associated with economic or population crisis. But is it adaptable enough to respond to the disruption caused by HIV/AIDS? Increases in orphanhood may induce at least a temporary intensification of fosterage as urban mothers (or grandparents) seek to relieve the burden of child-caring. However, this type of response will become quickly inefficient for several reasons. First, the potential demand for transfers of children in an HIV epidemic could be considerably more massive than the system is designed to accommodate. Searching costs will increase, and the entire operation will become more cumbersome and taxing for the sending family. Secondly, since fosterage involves reciprocity, the partial or total disabling of the sending family by illness may render the exchange less attractive to the receiving end and may ultimately become impossible.

Yet another factor could play a more important role. Families affected by HIV, where one or both parents are ill and incapable of sustained productive activity or dead, will require support from young children. The children will be more likely to be withdrawn from school or else may become frequent absentees; their participation in informal markets will increase and, overall, the incentives to send some of the children into fosterage will be considerably reduced. Thus, although fosterage may provide temporary relief to the needs created by the presence of HIV, it is likely that new conditions will erode its very basis of existence.

Female children may share the bulk of the costs as households struggle to preserve entitlements for subsistence. Where females represent bride wealth, there will be strong incentives to reinforce early unions with older and wealthier males. This will reproduce conditions of high exposure to HIV and will make possible the persistence of at least one necessary condition for HIV to become endemic (age gap between sexual partners). If the sexual favours of younger girls can be exchanged for cash payments or other economic benefits by engaging them directly with protectors, "sugar daddies" and various other assorted types of more or less casual remunerative relations, the practice could become a court of last resort precisely among those families affected by HIV/AIDS. Thus, paradoxically, a consequence of HIV may be that, in the absence of massive external interventions and left to themselves, families will adapt to the new conditions by

clinging to traditional behaviours and by creating a social environment that sustains the continued reproduction and transmission of HIV.

Conclusion

This article has dealt with the conditions that heighten the exposure to and risk of HIV for women in societies of Africa. It has also identified some of the features of family organization, women's positions and roles that mediate the action of selected effects of HIV/AIDS on women and children.

Although our estimates hold only approximately, the evidence available indicates that there is substantial room to reduce the amount of exposure to and the risk of HIV infection that women face. Most important among these are the delay in the entrance into a sexual union and the reduction in the age gap between sexual partners. Interactions between the natural history of HIV and social conditions constraining the behaviour of females (but not of males) could lead to differentials in the incubation process that will result in more rapid health deterioration and reduced expected survival for infected females.

The increase in the incidence of orphanhood, widowhood and incomplete coresidence units will all be the outcome of increases in adult mortality. While rising child mortality may have an important impact on short-term fertility trends and deleterious effects on the health of fecund seropositive women, increases in adult mortality could potentially induce a complete overhaul of family arrangements. Families may be forced to alter rules of recruitment, to reshuffle members and, more importantly, to redefine completely their roles and positions. And although all members of the family unit stand to lose in the process, female children, mothers and grandmothers will probably bear the bulk of the personal, social and economic costs unless social interventions effectively reallocate the burden.

Notes

[1] This does not imply that women select men but rather that the coupling of males and females follows certain rules that restrict the number of possible partnerships that can be formed.

[2] Rates of sero-prevalence above age 10 increase smoothly from about age 14 to about age 40 and then decline. The profile of increase by age can be captured quite accurately by a simple exponential function.

[3] Intensification of the economic crisis that most of the African and Latin American countries are undergoing and the "belt-tightening" programmes to which their economies are strapped can only complicate the situation.

[4] It is, of course, possible that cost considerations and not just motivational factors play also an important role in the demand for condoms or spermicides.

[5] The expected values of orphanhood presented here are compatible, although not equivalent to estimates obtained by Preble (1991) and Hunter (1990).

[6] Some of these effects may be offset by the higher probability of pregnancy terminating in stillbirth, which has been verified in a variety of contexts (Guay and others, 1990; Lopita and others, 1990).

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