Supplement 2 to Health Transition Review 7, 1997

Preface

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Introduction

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By the end of 1996 the estimated number of people living with HIV/AIDS in the world was 22.6 million. Of that number, 14 million, accounting for nearly two-thirds of the total, were in sub-Saharan Africa. Nearly two decades into the outbreak, the epidemic has spread throughout the subregion with complex spatio-temporal variability within and between countries. While the epidemic has apparently peaked in parts of Uganda and the Democratic Republic of Congo (formerly Zaire), it is still rising in parts of Kenya and Southern Africa and is yet to become a major problem in most countries in West Africa. Secondly, the epidemic has become more diffused and has spread from among the initial 'high-risk groups' to the general population. Thirdly, evidence points to a possible increase in intensity in the years ahead since the co-factors (e.g. STDs, lack of male circumcision) and the forces (e. g. mobility and instability) which impel the spread of the epidemic still persist (Caldwell and Caldwell 1993; National Research Council 1996).

There is no doubt that AIDS is having a tremendous impact on the demographic, social, political and economic aspects of life in some parts of sub-Saharan Africa. However, there are uncertainties surrounding some of the consequences mainly because of the lack of adequate and reliable data (National Research Council 1996). Sub-Saharan Africa has worse demographic and epidemiological data than other major world areas, making it difficult to track the progress of the epidemic (Mann and Tarantola 1996). As a result, discussions on the socio-demographic impact of the epidemic on the sub-continent have mainly relied on estimates and models. For this reason, the Committee on AIDS of the International Union for the Scientific Study of Population (IUSSP) decided to organize a conference to bring together the current evidence of the socio-demographic impact of AIDS in Africa. Data bases for the measurement of the spread and consequences of the epidemic are modest, in both geographical coverage and length of study period. Yet, as is demonstrated in this volume, the small number of longitudinal studies of communities affected by HIV/AIDS provide a wealth of data on actual impact in their study populations and some important clues as to the possible impact elsewhere in Africa.

There is the *primary* effect of HIV/AIDS on demographic variables such as mortality, fertility, rate of population growth and age structure, and its *secondary* effect on the spread of other infectious diseases such as tuberculosis, on social structures such as orphanhood, fostering and marriage and the economy and welfare. Ten of the papers in this volume focus on the impact of the epidemic on adult mortality, fertility and household structure. The concluding contribution by John Caldwell puts the results of these papers into a broader perspective.

Adult mortality and morbidity

Data on adult mortality are scarce for most of Africa. In the absence of sound vital registration systems the principal data sources are censuses, surveys and longitudinal studies. George Bicego's paper on the use of national survey data to estimate adult mortality shows that the potential of household surveys is greater than perhaps was anticipated. He uses data from seven African countries to show that the sisterhood method used by the Demographic and Health Surveys (DHS) is a reliable indirect method of estimating adult mortality. To some extent DHS and other surveys can thus be used to estimate national trends in adult mortality; and thereby to estimate the effect of AIDS on adult mortality.

Simply ascertaining levels of adult mortality with any degree of accuracy has long proved a challenge for demographers in sub-Saharan African countries. Ian Timaeus and Andrew Nunn present an evaluation of one of the most popular indirect methods of estimating adult mortality, the orphanhood method, in the context of stable levels of HIV in a rural population. They found that the method is fairly robust provided fairly simple adjustments are made to compensate for the likelihood that seropositive mothers will transmit HIV to any future children and that these children will therefore not survive to report orphanhood.

HIV prevalence in Africa is determined by its incidence, the survival time between infection and death and the current population growth rate, as cure rates are insignificantly low. John Blacker and Basia Zaba begin from this premise to try and model the likelihood that an individual will die from HIV-related causes, given a certain level of prevalence. Using data from Kenya, they estimated that, where prevalence is under eight per cent (which implies that one person in 13 is seropositive), about 30 per cent will die of HIV-related causes. Such a figure may convince policy makers of the severity of the epidemic.

The most detailed data on the effect of the AIDS epidemic on adult mortality are derived from longitudinal studies. In a rural cohort of 20,000 in northwestern Tanzania, Ties Boerma and colleagues show that although HIV prevalence among adults between 15 and 44 years was under seven per cent, over a third of all adult deaths were classified as HIV/AIDS related. The next most prominent killer, diarrhoea, trailed far behind, causing just over six per cent of deaths. At these levels of HIV prevalence, Boerma and colleagues calculated the current probability of dying between the ages of 15 and 60 as 42 per cent for men and 43 per cent for women: about five times the likelihood commonly recorded in industrialized countries. It is argued that Africa, which already lagged behind in the epidemiological transition, will be thrown back even further compared to the other continents of the developing world.

The interaction of HIV with other diseases further complicates the mortality picture. Tuberculosis, traditionally an important killer of young adults in Africa, has been found to be the most important opportunistic infection associated with HIV infection in Africa. The case control study conducted in Malawi by Judith Glynn and colleagues shows that when under three per cent of the adult population was infected with HIV, some 17 per cent of tuberculosis was associated with the virus. But the proportion of tuberculosis associated with HIV rises dramatically with HIV prevalence. As HIV prevalence rose towards 11 per cent, nearly four tuberculosis cases in ten were related to the virus. The impact of HIV on tuberculosis is greater still, since a rise in the number of tuberculosis infections (and a lower likelihood that cases will be cured) increases the spread of the disease in the population at large, regardless of HIV infection. A better understanding of how HIV relates to tuberculosis, malaria and other communicable diseases is essential if policy-makers and service providers are to use precious resources to best effect.

Fertility

Most projections of the impact of the demographic epidemic have assumed that there will be no significant difference in the 'with and without AIDS' fertility in sub-Saharan Africa in the immediate future (United Nations 1991). However, the precise relationship between HIV infection and childbearing is important, not least because much of what we know about levels and trends of infection in different areas comes from testing pregnant women at antenatal clinics. The more we know about how HIV infection relates to pregnancy, the better idea we have of how our sentinel surveillance data relate to women in the general population. In addition, estimates of fertility among HIV infected women have a profound effect on projections of the numbers of orphans and, to a lesser extent, on child mortality rates.

In a paper that seeks to clarify the interaction between HIV and fertility, Simon Gregson and colleagues consider the possibility of a direct physical effect of the virus on the probability of conception. They also posit that changes in behaviour that result from a growing awareness of HIV and a desire to avoid infection or vertical transmission might change childbearing patterns. Behaviour change might work in both directions. Where post-partum abstinence and long periods of breastfeeding are common, women may feel these practices push their husbands into multiple partnerships and therefore increase their risk of infection. Curtailing abstinence and breastfeeding may increase fertility. Deliberately seeking to increase fertility in response to the high-mortality environment created by HIV would also increase the average number of children per woman. Where the most common response to the fear of infection is decreased sexual activity or increased condom use, behaviour change is, on the other hand, likely to limit childbearing. In high-HIV-prevalence situations, death and disabling illness will also cut down the time spent in sexual unions. Overall, Gregson and his colleagues from the Blair Institute in Harare consider that the forces reducing fertility are likely to outweigh those promoting fertility.

In the following paper Lucy Carpenter and colleagues examine in greater detail the physical relationship between seropositivity and fertility. Using data for a rural Ugandan population, they look at fertility in the seropositive over a six-year period. After adjusting for age, HIV-infected women were 23 per cent less likely to bear children than the seronegative. By contrast, they found no significant relationship between fertility and markers of previous syphilis infection. It may be that the reduction in fertility differs at different stages of the epidemic, and with different mixes of behavioural and physiological factors. These papers show that fertility is diminished even when controlling for behavioural differences. But they also reminded us that behavioural variables such as age at first sex, rates of partner exchange and coital frequency, some of which may alter in response to perception of risk of infection, can be important factors in reducing fertility in their own right.

Household composition and structure

Associated with the increase in adult mortality are likely increases in the level of orphanhood, fostering and the emergence of child- and adolescent-headed households. Given the already high adult mortality in sub-Saharan Africa, orphanhood is not a new phenomenon. Fostering has been one of the adaptive mechanisms to deal with the disruptions created by high adult mortality and other uncertainties in life. It has been common to bring up children other than one's own in most African societies. However, HIV-associated deaths have changed the dynamics and the magnitude of problems associated with child rearing. But the appearance of child- and adolescent-headed households does not necessarily mean that the extended family system has abandoned its responsibility towards the care of the children of relations. Rather, it may represent another dimension in societal dynamics as populations go through traumatic experiences. It is important to establish whether such adaptations are going to be transient. We

need long-term studies in order to put into perspective the possible life courses of the processes taking place.

Experience of HIV in the household is likely to affect people's perception of risk, so it was interesting to see quantified the difference between people affected and households affected. In a study of rural villages in Uganda's Rakai district, Joseph Konde-Lule showed that where adult HIV prevalence was 19 per cent, over 31 per cent of households were affected by HIV. Prevalence was higher among household heads than in the general population and HIV-associated deaths have more adverse consequences for the household than adult deaths not related to HIV: a greater decline in dependency ratio and more loss of household possessions were registered in Rakai.

In several rural societies child fostering was shown to be common regardless of orphanhood. Mark Urassa and colleagues use data from rural Tanzania to show that over a third of children whose parents were alive did not live with both their biological parents, and over one in ten lived with neither surviving parent. Two households in five were home to children who were neither indigenous to the household nor orphans, nearly three times as many as those housing orphans. In the population studied, orphans and foster-children seemed to fare as well in terms of health and schooling as biological children, at least up to the teen years when children not living with their birth parents were more likely to drop out of school.

In a study which examines the point at which traditional extended family mechanisms become saturated, Geoff Foster and colleagues document the appearance of child-headed households in Zimbabwe. They note that the problem is likely to be more acute in urbanized societies and those where migration is common. And it will become more acute still in the next generation, because the orphans of people who lost their own parents in the early years of the HIV epidemic will have no grandparent generation to care for them. When older children are forced to drop out of school or sell assets or sex to support younger children, they may be increasing their own vulnerability to HIV infection. Institutionalization is not the answer, but policy-makers should encourage social support for and beyond the extended family system.

Conclusion

When considering the impact of the epidemic one needs to be careful not to over-generalize the findings from small-scale studies to cover the whole of sub-Saharan Africa, which is a region of great diversity. Nonetheless the results reported here, the data collection techniques and the models used are among those that are likely to generate new challenges for demographers and epidemiologists in addressing issues of measurement and they will inform policy-makers about the structures needed for addressing some of the emerging problems. There is also a need to understand community responses as well as the survival strategies adopted by individuals and families in the face of HIV/AIDS morbidity and mortality. These will have to be obtained from localized detailed studies such as those reported in this volume.

The largest HIV/AIDS epidemic has occurred in a continent which is least able to deal with its ramifications. Sub-Saharan African countries constitute the bulk of the countries in the bottom third of the UNDP human development index. For instance, seventeen of the last 20 of the 174 countries in the index are in sub-Saharan Africa. Unfortunately these countries, which can least afford the cost of prevention and treatment, are the most affected. But as pointed out by Caldwell, African countries have shown a tremendous ability to shoulder adversities. Some of the changes in household structure are likely to be transient. Only through studying the dynamics of traditional and new coping mechanisms can we help the development of appropriate responses to mitigate the severe consequences of the AIDS epidemic in sub-Saharan Africa.

References

Caldwell, J. C. and P. Caldwell. 1993. The nature and limits of the sub-Saharan Africa AIDS epidemic: evidence from geographic and other patterns. *Population and Development Review* 19: 817-848.

Mann, J. and D. Tarantola (eds.). 1996. *AIDS in the World II*. York: Oxford University Press.

National Research Council. 1996. *Preventing and Mitigating AIDS in sub-Saharan Africa: Research and Data Priorities for the Social and Behavior Sciences*. Washington DC: National Academy Press.

United Nations. 1991. *The AIDS Epidemic and its Demographic Consequences.* New York: United Nations.

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Estimating adult mortality rates in the context of the AIDS epidemic in sub-Saharan Africa: analysis of DHS sibling histories*

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Abstract

Recent efforts to model the demographic effect of the AIDS pandemic in sub-Saharan Africa have far outnumbered empirical studies of adult mortality levels and patterns in AIDS-affected countries of the region. There is still a paucity of population-based data on adult mortality for nearly all countries in the region. Using data from recent Demographic and Health Surveys (DHS) of six countries and one in-depth DHS, this paper examines the use of sibling histories to directly estimate rates of adult mortality. The countries studied include Uganda, Zambia, Central African Republic, Côte d'Ivoire, Zimbabwe, Malawi, and Tanzania.

Rates of adult male and female mortality are presented at the national level in comparison to estimates obtained from other published sources, where available, and for subnational areas where cohort and other mortality studies have been recently conducted. The results indicate surprising consistency with external data and, on the whole, underscore the expected but hitherto only sparsely documented association between residence in high HIV-prevalence areas and sharply elevated mortality risk during the relevant adult ages. The cases of Zambia and Uganda in particular provide clear evidence of very high adult mortality levels among both men and women. In general, the findings of the study demonstrate that DHS-type sibling histories represent a promising, relatively untapped source of data that will add to our understanding of adult mortality dynamics in Africa. The paper discusses some of the advantages and potential limitations of the data and derived mortality estimates.

Since the late 1970s, the estimation of childhood mortality levels in developing countries has been routinely accomplished through the use of data from large-scale national surveys and decennial censuses. In sub-Saharan Africa especially, in the 1980s and 1990s there has been an increasing reliance on data from the Demographic and Health Surveys (DHS) program for population-based assessment of changes in rates of infant and child mortality (Sullivan, Rutstein and Bicego 1994; Bicego and Ahmad 1996). Estimation of adult mortality in sub-Saharan Africa has received much less attention.

This is in part because the appropriate data are lacking or of very poor quality, which makes the derived estimates difficult to interpret consistently. Still, some notable efforts at interpretation using indirect techniques and adjustments to survey and census data have been made. Timaeus (1993) has shown that adult survivorship, in many countries in the region, had been improving steadily between the 1950s and 1970s. His results indicate a highly variable and changing relationship between early childhood and adult mortality and that, in many areas of sub-Saharan Africa, gains in adult survival have not been matched by improvements in

* Helpful comments on an earlier version of this paper provided by Anne Cross and Jerry Sullivan are gratefully acknowledged.

child survival. More easily obtained information on the level of childhood mortality in a particular setting is thus not a good guide to the adult mortality level or pattern. This makes problematical the use of model mortality schedules, which posit a relationship between adult and child mortality.

The expanding AIDS epidemic means that an understanding of adult mortality levels and trends in the region will become at once increasingly important and more difficult to achieve over the next two decades. First, even assuming accurate, population-based measurement of mortality, attribution of trends at the national level will be confounded by concomitant and interacting effects of HIV/AIDS, variously paced social and economic changes, and the expansion, and sometimes the breakdown, of health systems. Indeed, for the foreseeable future, it should be acknowledged that causal interpretations of mortality trends at the national or large-area level will be limited to speculation. This situation could be improved should representative information on the changing cause-of-death structure of populations become available.

The immediate and practical issue regarding the effect of AIDS is one of reliable measurement of adult mortality at a geographic level and with time-specificity useful for policy analysis. At present, the existing data collection and analysis methodologies do not address this basic measurement issue. In this paper, a relatively untapped source of adult mortality data derived from DHS surveys is offered, and estimates are compared with data from other external sources where available. The base data sets used for estimation are all from recent DHS surveys in sub-Saharan countries experiencing intense HIV/AIDS epidemics.

Estimating adult mortality from sibling histories

To date, the assessment of adult mortality levels in sub-Saharan Africa has been an arduous and sometimes frustrating undertaking for a number of reasons, which have been discussed by Timaeus (1991). An essential problem in producing population-based estimates of adult survival has been the nature of the events under consideration (adult deaths) in relation to persons who would potentially provide information or histories of the events. In collecting data for estimating childhood mortality, a sample of mothers represents a convenient point of contact and communication from which to develop first-hand histories of recent events. The complete birth history first identifies all recent births, the units of exposure to risk (denominator data); collection of survival status and age-at-death information (numerator data) then allows a synthetic cohort of individuals to be followed through the age and calendar periods of exposure to risk. In sub-Saharan Africa, this approach is not viable for adult mortality estimation, since the relevant exposure to risk (ages 15+) occurs long after most respondent mothers have either died or have aged beyond reproductive years, severely truncating exposure data. Nevertheless, the first aim of demographers should still be to establish a genuine history of exposure events and deaths delineated on time and age dimensions, and to calculate death rates directly.

The work of Graham, Brass and Snow (1989) showed the advantages of collecting and analysing information from women on the survival of their siblings[1]. The original approach of the Sisterhood Method was a data-collection protocol combined with an analytical technique: collect aggregate-level information on adult sisters dying from maternal causes and analyse

[1] Hill and Trussell (1977) were the first to undertake careful comparative evaluation of the indirect methods of adult mortality estimation: widowhood, orphanhood, and sibling survivorship methods. Their results indicate that, of the three, the sibling survivorship method should be regarded as the most suitable method for non-cause-specific mortality in young adults.

the data by age group of the respondent, to produce a lifetime maternal mortality risk through use of adjustment factors based on model fertility and mortality schedules. Lifetime risk can be converted to a maternal mortality ratio using information on the prevailing fertility level and expressed in deaths per 100,000 live births. The DHS program expanded on this work by moving from aggregate data collection to collection of complete sibling histories, with ages and dates information elicited. Essentially, these are complete live birth histories of respondents' mothers. While these data continue to be used for the indirect estimation of maternal mortality, recently the emphasis has shifted to producing maternal mortality rates directly. Direct estimates are preferable since they allow rate calculation based on more recent events, which is superior on both substantive and data-quality grounds, and because models of demographic patterns are not required.

The DHS sibling histories also present a unique opportunity to evaluate overall male and female adult mortality rates. One of the key constraints in producing reliable maternal mortality rates has been the need for large sample sizes. Graham (1991) suggests that reports from 3000-6000 respondents are required to produce a sufficiently stable (single) rate. Since the number of overall female deaths is typically three to five times larger than the subset of maternity-related deaths, the relative standard error on an overall adult mortality rate is much smaller allowing, in many DHS surveys, sex-specific breakdowns by age or subnational area. As always, the ability to produce age-specific estimates of mortality is an important advantage of direct over indirect estimation techniques.

However, because this type of information has been widely collected only for the past few years, critical examination is still necessary to place the same level of confidence in derived adult mortality estimates as is placed in child mortality estimates from own birth histories. An underlying concern associated with use of sibling histories has been the implicit assumption of no relationship between sibship size and sibling survivorship[2]; but by limiting the exposure under observation to that upon reaching adulthood (15 years), the assumption is not an unreasonable one. Another potential problem is that information on brothers (births and deaths) may be less completely reported by the female respondents than information on sisters, since brothers will typically travel farther from their natal home and keep less in touch with their sisters. It should also be expected that fundamental data-quality problems of age and age-at-death reporting will be more pronounced than in 'own' birth histories, since the events are more removed from the observer. For example, in the typical high-fertility setting, large age differences between siblings may make even knowledge of an older brother's or sister's survival status questionable.

Data and methods

The following illustrative analyses use the sibling history data from DHS surveys recently conducted in six countries: Uganda, Zambia, Zimbabwe, Central African Republic, Côte d'Ivoire, and Malawi. The DHS sibling history, in all significant aspects, is identical to a conventional 'own' birth history, except that certain probes are used in the data collection process to ensure that only maternal siblings are included.

The estimation procedure is straightforward, and roughly follows the direct life table approach used to calculate childhood period mortality rates from survey-based birth histories (see Bicego and Ahmad 1996). However, mortality rates ($_nm_x$) rather than life table probabilities ($_nq_x$), are presented to allow easier comparisons with external estimates typically expressed as deaths per person-year. The rate estimates are produced for the time period 0-6

[2] Brothers and sisters from large sibships have a greater chance of being included in the sample of sibling histories, and therefore are overrepresented.

years before the survey date, which keeps sampling error at an acceptable level and avoids potential distortions in the estimates caused by heaping at five-year increments of time and age. Currently, DHS is looking at age structural effects on the use of data for the more distant time periods; meanwhile, estimates of trend from the data of a single survey are not recommended.

Where feasible, estimates are produced for subnational regions. For these, it is best to regard the estimates for the overall 15-49-years age range and the broad age-pattern of mortality, rather than any single age-specific estimate which will have a large relative sampling error.

Adult mortality levels

Table 1 and Figure 1 show directly calculated rates of adult mortality by sex for the six DHS countries; for comparison, rates for the United Kingdom in 1990 are also presented (OPCS 1992). For females aged 15-49, the estimates range from 11 per 1000 (Zambia) to 4 per 1000 (Zimbabwe); for males aged 15-49, the estimates tend to be higher and range from 11 per 1000 (Zambia) to 5 per 1000 (Zimbabwe). Compared to the UK, females experience 8 to 21 times and males 5 to 11 times higher rates of dying during these ages. In the Central African Republic and Malawi, overall female mortality is as high as male mortality, which may reflect unusually high female mortality conditions in those countries, an underreporting of male relative to female mortality, or a combination of the two. Male mortality exceeds female mortality by 7, 13, 19, and 28 per cent in Zambia, Uganda, Côte d'Ivoire and Zimbabwe, respectively.

The estimated age patterns of mortality are plausible, with fairly smooth, incremental rises in risk occurring with increases in age. Although departures from this pattern may be the result of age-selective underreporting, as with mortality in the 45-49-years age group in Côte d'Ivoire, it is incautious to jump to this broad-based conclusion. For instance, in the case of Zambia lower female mortality at ages 40-49 may simply be the result of genuine rises in mortality during ages 25-39 (see Figure 1). There is evidence of heaping of ages at death at 30 and 40 in a few countries, which, if associated only with age-at-death misreporting, and not omission of deaths, will not significantly affect the estimates for the overall 15-49 age range.

Uganda

Table 2 looks specifically at the Uganda DHS estimates in comparison to published mortality rates from two population-based cohort HIV/AIDS studies conducted in Rakai district (Sewankambo et al. 1994) and neighbouring Masaka district (Mulder et al. 1993), both located in the high HIV-prevalence area of the Central region. Three points emerge from the comparisons.

Table 1
Age and sex-specific mortality rates 15-49 years (deaths per 1000 person-years) for six countries of sub-Saharan Africa, and the United Kingdom for 0-6 years before the survey.

Age	Zambia 1996	Uganda 1995	Central African Rep. 1994-95	Malawi 1992	Côte d' Ivoire 1994	Zimbabwe 1994	United Kingdom 1990
Females							
15-19	4.3	3.9	4.4	5.3	2.0	2.1	0.3
20-24	7.7	7.5	6.8	3.6	3.6	3.0	0.3
25-29	13.9	9.0	7.8	6.8	4.5	4.5	0.4
30-34	15.3	12.7	9.2	7.2	5.9	4.8	0.5
35-39	16.5	11.5	9.7	9.0	6.1	5.7	0.9
40-44	14.7	11.1	13.8	8.9	11.1	5.4	1.3
45-49	14.5	17.6	17.1	9.6	7.3	5.4	2.2
15-49[a]	10.6	9.0	8.4	6.6	4.8	3.9	0.5
Males							
15-19	3.4	3.0	3.5	3.8	2.5	1.7	0.7
20-24	5.4	5.1	5.5	4.1	3.4	3.1	0.9
25-29	11.7	10.8	7.6	6.8	5.3	4.3	0.9
30-34	17.5	16.4	9.3	8.4	9.1	6.4	1.0
35-39	22.2	14.5	12.1	7.6	7.8	6.0	1.4
40-44	22.5	19.7	14.2	10.1	11.0	10.1	2.0
45-49	21.3	21.2	17.2	9.7	8.5	13.6	3.4
15-49[a]	11.3	10.1	8.3	6.5	5.7	5.0	1.0

[a] age adjusted

First, the DHS data describe a gradient in mortality rates (sexes combined, age 15-49) from very high mortality of over 14 deaths per 1000 in the four high HIV-prevalence districts that include Rakai and Masaka[3], and about 13 per 1000 for the whole of the Central region, to 8 per 1000 (still high) in the remaining North, West, and Eastern regions where the HIV/AIDS problem is generally less severe. Second, the DHS estimate for the four high HIV-prevalence districts combined (14 per 1000) is much lower than the rate calculated from the entire Rakai study population (32 per 1000), where adult HIV prevalence was estimated at 20 per cent. The DHS estimate, however, is much higher than in the Masaka study population (10 per 1000) where adult prevalence was estimated to be eight per cent. Third, the mortality rate calculated from the adult HIV-negative population of Rakai (8 per 1000) is comparable to the DHS rate for the non-high HIV-prevalence areas. But the observed Masaka rate for the HIV-

[3] This includes an area comprising the contiguous districts of Rakai, Masaka, Mpigi, and Kampala (STD/AIDS Control Programme 1994).

negative population (1-2 per 1000) indicates a problem with underreporting of deaths, at least in this segment of their study population.

Figure 1
Male and female adult mortality by age group, 0-6 years before survey, 6 DHS countries and United Kingdom

Table 2
Comparison of age-specific mortality rates (deaths per 1000 person-years) of 1995 Uganda DHS data 0-6 years before the survey with cohort studies in Uganda, sexes combined.

	Small area cohort studies (Uganda)				DHS sibling history data		
Age Group	Rakai study	Rakai-HIV only	Masaka study[a]	Masaka HIV only[a]	4 High-risk Districts in Central Region[b]	Central Region-Uganda	Rest of Uganda (WNE regions)
15-19	4.8	2.7	7.3	1.9	3.6	3.4	3.4
20-24	35.5	5.0	↓	↓	12.0	10.2	4.8
25-29	↓	↓	14.6	1.6	15.3	14.6	8.0
30-34	45.5	10.4	↓	↓	22.4	19.9	12.4
35-39	↓	↓	7.9	0.0	23.5	19.9	10.3
40-44	46.6	22.2	↓	↓	25.2	21.1	13.0
45-49	↓	↓	8.4	1.6	24.0	22.8	18.1
15-49 (age adj.)	32.1	8.1	10.4	1.5	14.4	12.9	8.1

Sources: Rakai data, Sewankambo et al. (1994); Masaka data, Mulder et al. (1994)

[a] Age groups are 13-24, 25-34, 35-44, and 45-49 years. The 45-49 group was estimated as the interpolated value of the mortality rates during age groups 35-44 and 45-54.

[b] Rakai, Masaka, Mpigi, Kampala districts.

Table 3 shows the sex-specific DHS-based estimates of adult mortality for the high HIV-prevalence districts relative to the estimates for the rest of Uganda. Overall residence in the high HIV-prevalence areas is associated with about 71 per cent excess risk for females and 86 per cent excess risk for males. Figure 2 shows that for both men and women, mortality during age 15-19 does not differ between high HIV-prevalence districts and other districts, but that from age 20-24 onwards, mortality is considerably higher in the high HIV-risk districts. Taken together these results indicate that the Uganda DHS sibling histories do capture differential mortality associated with the geographic spread of HIV/AIDS.

Côte d'Ivoire

Table 4 looks at DHS subnational estimates for Côte d'Ivoire and estimates reported by Garenne, Madison and Tarantola (1994) for the capital city of Abidjan based on vital registration data. Abidjan, like many cities of sub-Saharan Africa, has experienced a rapid spread of HIV infection. The authors of the Abidjan study carefully examined changes in the cause-of death structure and concluded that the upward trend in overall adult mortality between 1983-87 and 1988-92 (40%) was due primarily to increases in AIDS-related deaths. They also suggest that this is probably an underestimate of AIDS impact, because of changes in underreporting and residence shifts.

Table 3
Excess mortality risk associated with residence in high HIV-prevalence area: age-specific mortality rates (15-49 yrs) by sex for 0-6 year period before the survey, 1995 Uganda DHS.

	Females			Males		
Age	High-HIV area [a]	All other areas	Excess mortality (/1000)	High-HIV area [a]	All other areas	Excess mortality (/1000)
15-19	4.4	3.9	0.5	2.9	2.9	0.0
20-24	14.5	5.7	8.8	9.4	3.8	5.6
25-29	12.7	7.7	5.0	18.1	8.4	9.7
30-34	16.3	12.0	4.3	29.9	12.9	17.0
35-39	20.4	9.7	10.7	26.8	11.0	15.8
40-44	20.6	8.1	12.5	29.8	18.3	11.5
45-49	20.8	15.4	5.4	27.4	20.9	6.5
15-49 (age adj.)	13.2	7.7	5.5	16.0	8.6	7.4

[a] Rakai, Masaka, Mpigi, and Kampala Districts

The DHS estimates indicate relatively small geographic differentials in adult survival chances. The DHS estimate for Abidjan (which is centred in time a bit later than the most recent vital registration estimate) is in line with the rising mortality documented in the Abidjan study . A rise in AIDS-related urban mortality in recent years has apparently erased urban-rural differentials. These results need to be qualified by the fact that retrospective survey data, but especially sibling histories, can potentially suffer from residential misclassification. There is no way to correct for this in these data, but it is probably true generally that the estimation of an urban-rural differential will be biased towards zero when internal migration flows are significant, for example when the place of occurrence of a large proportion of sibling events—exposure and deaths—differs from the geographic domain of sampling and interview.

Zambia

The HIV/AIDS situation in Zambia is one of the most dire in the region and thus in the world, as was reflected in the enormously high national-level adult mortality rates shown in Figure 1. In Table 5, subnational DHS estimates are presented along with urban-rural estimates published from the 1990 national census. The census data show the expected (pre-AIDS) rural excess in mortality risk. The DHS estimates covering the 1990-1996 period on the other hand show higher mortality in urban areas, especially in the large urban areas in the capital of Lusaka and the Copperbelt where HIV prevalence estimates among antenatal clinic attenders typically exceeded 25 per cent in 1994 (Fylkesnes 1995). While the census estimates are probably not directly comparable because of different methodologies, if taken at face value, urban adult mortality has roughly doubled during the 1990s while rural mortality has risen by about 25 per cent. Again, residential misclassification may operate to understate the urban-rural differential. It should be noted that, unusually by regional standards, Zambia has a large urban population, 40 per cent (Central Statistical Office, Zambia 1995).

Figure 2
Male and female adult mortality by age group, high HIV-prevalence districts risk and other districts, 1995 Uganda DHS

Table 4
Comparison of age-specific mortality rates (deaths per 1000 person-years) of 1994 Côte d'Ivoire DHS data 0-6 years before the survey with vital registration estimates, Côte d'Ivoire, sexes combined.

				DHS sibling history data (1988-94)			
Age	Abidjan Vital Registr. 1983-87	Abidjan Vital Registr. 1988-92	Abidjan DHS 1988-94	Other Urban South	Rural South	Urban North	Rural North
15-19	1.7	1.9	2.9	1.3	2.0	1.6	2.9
20-24	2.0	2.7	4.2	2.9	3.6	3.2	2.9
25-29	2.6	3.9	5.4	5.3	4.4	5.7	4.6
30-34	3.4	5.1	7.2	7.2	7.7	6.9	7.5
35-39	5.1	6.9	6.9	5.7	7.8	6.5	6.0
40-44	6.6	9.1	9.5	13.9	14.0	5.6	7.1
45-49	8.4	9.5	4.2	9.1	9.7	8.4	7.3
15-49 (age adj)	3.3	4.6	5.1	4.7	5.7	4.3	5.0

Source vital registration-based estimates: Garenne et al. (1994)

Table 5
Comparison of age-specific mortality rates (deaths per 1000 person-years) of 1996 Zambia DHS data 0-6 years before the survey with estimates from the Zambia 1990 National Census, sexes combined.

	DHS Sibling History Data (1990-96)			1990 Census	
Age	Large urban[a]	Other urban	Rural	Urban	Rural
15-19	4.0	3.4	3.8	4.4	5.4
20-24	8.3	7.0	5.3	5.8	7.1
25-29	15.9	15.2	10.2	6.4	7.8
30-34	20.5	14.8	14.2	7.1	8.6
35-39	25.8	18.7	15.7	8.0	9.8
40-44	20.0	21.0	15.9	9.4	11.4
45-49	23.1	21.0	15.7	11.0	13.2
15-49 (age adj)	13.1	11.5	9.5	6.2	7.5

[a] Includes urban areas of Lusaka and Copperbelt Provinces.

Malawi

Table 6 shows geographic differentials in adult mortality in Malawi based on the 1992 DHS sibling histories. On the basis of these estimates, adults (age 15-49) in the Northern region have much better survival prospects (4 per 1000) than do adults in the Central and Southern regions (7 per 1000), which is consistent with the patterns of social development shown by education and employment levels, and differentials in childhood mortality (NSO 1994). Adult mortality in the urban areas, mainly Blantyre, Lilongwe, Mzuzu, and Zomba, was estimated to be higher by some 30 per cent than mortality in all rural areas combined. Given the vast

difference in access to tertiary health-care services between urban and rural Malawi, this suggests either significant urban-rural differences in underreporting in the sibling histories, or higher morbidity in adults of this age in urban centres. Malawi is thought to be facing one the of worst AIDS epidemics in the world, with sharp differences in HIV prevalence between the major urban centres of Blantyre and Lilongwe on one hand and rural areas on the other (US Bureau of Census 1996).

Table 6
Comparison of age-specific mortality rates (deaths per 1000 person-years) of 1992 Malawi DHS data 0-6 years before the survey with estimates from the Malawi 1987 Family Formation Survey, sexes combined.

	DHS Sibling History Data						
Age	Urban	Rural	South rural	Centre rural	North rural	Malawi 1986-92 DHS	Malawi 1983-84 FFS[a]
15-19	3.9	4.6	4.1	6.3	1.7	4.5	2.4
20-24	6.5	3.4	2.6	4.4	3.3	3.8	2.9
25-29	8.5	6.5	6.7	7.1	4.1	6.8	3.7
30-34	8.9	7.7	7.4	9.1	4.2	7.8	5.2
35-39	9.0	8.2	8.1	8.8	5.9	8.3	6.4
40-44	14.5	8.9	11.0	7.0	5.3	9.5	8.8
45-49	18.7	8.6	10.5	6.3	8.9	9.6	10.2
15-49 (age adj)	8.1	6.3	6.5	6.8	3.9	6.5	4.7

[a] Based on deaths reported in the last 12 months

The Malawi DHS took place in 1992, and consequently would not capture most of the expected AIDS-related rise in adult mortality during the 1990s. This may explain the lower than expected national-level mortality rate for Malawi seen in Figure 1.

Central African Republic

Table 7 shows variation in adult mortality across designated health regions (HR) in the Central African Republic. Overall mortality (age 15-49) ranges from 7 per 1000 in the East (HRs IV and V), to 10 per 1000 in HR I which surrounds the capital city of Bangui. In Bangui itself, mortality is 8 per 1000. This pattern is consistent with geographic variation in under-five mortality except that Bangui's adult mortality level is substantially higher than expected on the basis of childhood mortality reported by Ndamobissi, Mboup and Nguelebe (1995). Little is known about the geographic distribution of HIV/AIDS in the Central African Republic, except for evidence of high and rising HIV prevalence rates in the capital city (US Bureau of Census 1996).

Zimbabwe

Estimates of adult mortality for subnational areas in Zimbabwe are given in Table 8. Mortality is lowest in Matabeleland (3 per 1000) and highest in Mashonaland and urban areas outside the major centres of Harare and Bulawayo (5 per 1000). This pattern is roughly consistent with provincial variation in childhood mortality (CSO, Zimbabwe 1995). In the major cities of Harare and Bulawayo, the adult mortality rate is 4 per 1000. Given the very high level of

employment-related internal and external migration flows in Zimbabwe, the potential for residential misclassification is significant (see above regarding Côte d'Ivoire), making the interpretation of urban-rural differences problematical. Compared with the model-based estimate from the 1987 Intercensal Demographic Survey, national-level estimates suggest an increase of about 30 per cent in the rate of mortality during ages 15-49 since the mid to late 1980s.

Table 7
Comparison of age-specific mortality rates (deaths per 1000 person-years) of 1994-95 Central African Republic (CAR) DHS data 0-6 years before the survey with estimates from the 1988 CAR national census, sexes combined.

	DHS Sibling History Data (1989-95)						
Age	Bangui	Health Region 1 (except Bangui)	Health Region 2 (West)	Health Region 3 (N.west)	Health Regions 4 & 5 (East)	CAR DHS 1989-95	CAR Census 1988
15-19	3.9	4.1	4.1	4.5	3.8	3.9	4.9
20-24	5.8	7.9	4.1	8.6	4 .6	6.1	6.4
25-29	6.5	10.4	6.2	8.4	7.1	7.7	8.5
30-34	7.8	11.6	10.7	8.2	8.3	9.3	9.1
35-39	14.9	8.5	11.5	12.5	7.5	10.9	10.5
40-44	15.1	18.3	12.2	14.4	9.5	14.0	13.0
45-49	15.2	20.7	15.8	20.7	13.2	17.1	19.3
15-49 (age adj)	7.7	10.1	8.0	9.5	6.8	8.4	8.7

Table 8
Comparison of age-specific mortality rates (deaths per 1000 person-years) of 1994 Zimbabwe DHS data 0-6 years before the survey with estimates from the 1987 Zimbabwe Intercensal Demographic Survey (ICDS), sexes combined.

	DHS Sibling History Data						
Age	Harare/ Bulawayo	Other urban	Mashona -land rural	Matabele- land rural	Other rural	Zimbabwe 1989-94 DHS	Zimbabwe 1987 ICDS[a]
15-19	1.6	2.1	2.6	1.6	1.5	1.9	2.1
20-24	2.4	3.5	3.7	2.1	3.2	3.0	3.0
25-29	4.1	6.8	5.4	1.8	4.1	4.4	3.3
30-34	6.0	4.4	5.8	3.5	6.0	5.6	3.8
35-39	4.3	6.3	6.8	5.8	5.8	5.8	4.7
40-44	11.5	12.1	8.6	4.8	4.3	7.7	5.9
45-49	6.3	9.0	9.0	9.1	12.6	9.5	8.0
15-49 (age adj)	4.1	5.1	5.2	3.3	4.4	4.5	3.5

[a] Intercensal Demographic Survey, model-based using estimated under-five mortality

Mwanza Region, Tanzania

An in-depth study of mortality estimation was conducted during 1995 in Kwimba district of the Mwanza region in northwestern Tanzania (Bicego et al. 1997). The Sumve Survey on Adult and Childhood Mortality (SACM), undertaken with the principal aim of assessing the feasibility of collecting reliable birth histories by proxy (from sisters), also produced DHS-type sibling history data from which adult mortality rates were estimated for the period 1989-95. No HIV prevalence estimate is available for the study area, but it was likely to be 1-3 per cent during this reference period.[4] The mortality estimates obtained from these data are compared to mortality rates estimated from two cohort studies also undertaken in the Mwanza region. One of these, based in 12 scattered rural villages in various districts, was a population-based longitudinal study focusing on STD treatment as a means of reducing the spread of HIV (Todd et al. 1997). HIV prevalence was estimated to be four per cent in 1991-92.

The second study is continuing and aims to assess the causes and consequences of the HIV/AIDS epidemic in Kisesa Ward, Magu District (Boerma et al. 1997). The mortality data come from a two-year demographic surveillance of 20,000 individuals. HIV prevalence was found to be over ten per cent in the 40 per cent of the study population living in a trading centre along the main road leading to southern Kenya, and four per cent in the remaining rural population.

Table 9
Comparison of age-specific mortality rates (deaths per 1000 person-years) from three studies in Mwanza region,Tanzania.

Age	Coale-Demeny North Model: at q(5) of 134/1000	Sumve, Kwimba district 1989-95 All	Mwanza Region (rural) 1992-94 HIV only	Mwanza Region (rural) 1992-94 All	Kisesa, Magu District 1994-96 All
15-19	3.3	1.3	3.0	3.1	4.7
20-24	4.5	3.1	3.0	5.9	↓
25-29	4.9	4.1	3.8	8.4	9.9
30-34	5.5	7.2	6.2	10.2	↓
35-39	6.2	6.5	↓	↓	14.3
40-44	7.5	8.7	6.1	9.5	↓
45-49	8.9	8.2	8.4[a]	10.4[a]	15.2[a]
15-49[b]	5.1	4.6	4.5	7.2	8.7

Sources: Todd et al. (1997); Boerma et al. (1997). [a] Weighted interpolation of published rates for the age groups 35-44 and 45-54. [b] Standardized on the Sumve age structure

Figure 3
Adult mortality (age 15-49) in Mwanza Region Tanzania.

[4] Two studies undertaken in rural parts of Mwanza region estimated HIV prevalence of about 4 per cent. The Sumve area, where the SACM study was conducted, is on the whole more remote than the other two study areas. It thus seems unlikely that prevalence was as high as 4 per cent, and it was likely to have been much less for the 1989-95 reference period.

Table 9 and Figure 3 show the age-specific rates of death from these three studies against the Coale-Demeny North Model rates (Coale and Demeny 1983) implied by an under-five mortality rate of 134 per 1000 measured in the 1995 SACM study for the 1991-95 period. The rates from the SACM sibling history data are surprisingly consistent with rates produced from data of the Mwanza Region study (rural, HIV-negative population) and with levels implied by the model rates. The Kisesa rates are higher than the rates from the Sumve and Mwanza studies, with the greatest percentage differences occurring between ages 25 and 44. The overall adult mortality rate of 7 per 1000 for the entire Mwanza region study population, i.e., including HIV-positive individuals (4% HIV prevalence), reflects the intermediate position of this population with regard to the AIDS epidemic in the region.

These results should be interpreted in context. The Sumve and Mwanza region populations are located in more remote areas, and are probably on average less exposed to high HIV-risk, than individuals in the Kisesa study area. Further, the Kisesa reference period (1994-96) is later than the Mwanza region reference period (1992-94), and considerably later than the Sumve reference period (1989-95). If this region is on an upward trajectory regarding the spread of HIV, then the Kisesa estimates, *ceteris paribus*, are expected to be higher.

Discussion

It is important to emphasize what this study of survey-based adult mortality estimation was and was not intended to accomplish. It was not meant to quantify trends in adult mortality. Confidence assessment of demographic trends should generally involve more than one data source, especially given the relatively rapid deterioration in survival chances predicted for adults in much of the region. Unfortunately, to date, no country has held more than one national survey that included sibling-history data.

Nor was the study intended to suggest that direct estimates of adult mortality from survey-based sibling histories should supplant or substitute for continuing population-based indirect estimation, longitudinal studies, or demographic surveillance systems. The DHS sibling histories are a new source of information that should be viewed critically. The second rounds of surveys will allow more in-depth assessment of the quality of sibling history-derived mortality estimates. At this time, it is clear that these data suffer from some imprecision in age and age-at-death reporting which makes age patterns of mortality difficult to establish with confidence. It is however equally clear that, especially in the absence of cause-of-death information, age-specific estimates of adult mortality are very helpful in

interpreting mortality level estimates in the context of AIDS and should be viewed as a priority.

This paper was intended to demonstrate that direct mortality estimates from sibling histories provide an additional tool to complement estimates derived from longitudinal and other cross-sectional mortality data in the evaluation of AIDS impact. Their particular advantages include the following.

1. As typically implemented, DHS-type survey data are population-based and the estimates refer to geographic domains of practical interest to national planners; as opposed to cohort study and sentinel surveillance data, from which it is often difficult to extrapolate to larger geographically-representative domains.

2. Although not emphasized in this paper, direct analysis of full sibling histories allows estimates for a more recent, and therefore relevant, reference period than is now possible with indirect estimation techniques. A national estimate of adult mortality by sex for a reasonably recent two-to-three-year period immediately before the survey is feasible from a sample of about 5000 women aged 15-49; the average DHS sample in sub-Saharan Africa now exceeds 6000 respondents. Subnational estimates are possible at the same sample size, but at the cost of extending the retrospective reference period, and with the potential of producing misleading results due to residential misclassification of reference events. The latter problem should be carefully examined; it should be feasible to include current residence information in the sibling histories and use post-stratification procedures to re-weight the estimates.

3. Upcoming second rounds of DHS surveys will permit estimates of trends. The standardized nature of DHS data collection instruments, processing, and analysis facilitates comparability of estimates over time and between countries. With the experience gained from validation studies of verbal autopsies, it may also be feasible to incorporate additional questions on causes of deaths. This may allow a classification of deaths into broad categories useful for drawing inferences regarding the composition of changes in mortality levels.

The results of this study indicate a good deal of consistency with externally derived estimates of adult mortality and on the whole underscore the expected, but hitherto sparsely documented, association between residence in high HIV-prevalence areas and very high mortality risk during the relevant adult ages. In general, these findings indicate that DHS sibling histories represent a promising, relatively untapped source of data that will add to our understanding of adult mortality dynamics in Africa during the 1990s.

References

Bicego, G. and O. Ahmad. 1996. *Infant and Child Mortality.* Demographic and Health Surveys, Comparative Studies No. 20. Calverton MD: Macro International.

Bicego, G., S. Curtis, H. Raggers, S. Kapiga and S. Ngallaba. 1997. *The Sumve Survey on Adult and Childhood Mortality, Tanzania 1995: An In-depth Study on Estimating Adult and Childhood Mortality in Settings of High Adult Mortality.* Calverton MD: Macro International.

Boerma, J.T., J. Ngalula, R. Isingo, M. Urassa, K. P. Senkoro, R. Gabone and E. N. Mkumbo. 1997. Levels and causes of adult mortality in rural Tanzania with special reference to HIV/AIDS. This volume.

Central Statistical Office, Zambia. 1995. *Zambia Analytical Report. 1990 Census of Population, Housing, and Agriculture.* Lusaka.

Central Statistical Office (CSO), Zimbabwe. 1995. *Demographic and Health Survey, 1994.* Calverton MD: Macro International, Inc.

Coale, A. and P. Demeny. 1983. *Regional Model Life Tables and Stable Populations,* 2nd edition. New York: Academic Press.

Fylkesnes, K. 1995. *Updates on Patterns, Trends and Demographic Implications of the HIV/AIDS Epidemic in Zambia.* Lusaka: National AIDS/STD/TB and Leprosy Programme.

Garenne, M., M. Madison and D. J. M. Tarantola. 1994. *Demographic Impact of HIV/AIDS in Three West African Cities; I Abidjan: Preliminary Report.* Data for Decision Making. Cambridge MA: Harvard University.

Graham, W. 1991. Maternal mortality: levels, trends, and data deficiencies. Pp. 101-116 in *Disease and Mortality in Sub-Saharan Africa,* ed. R. Feachem and D. Jamison. New York: Oxford University Press.

Graham, W., W. Brass and R. Snow. 1989. Estimating maternal mortality: the sisterhood method. *Studies in Family Planning* 20:125-133.

Hill, K. and J. Trussell. 1977. Further developments in indirect mortality estimation. *Population Studies* 31:313-334.

Mulder, D., A. Nunn, H. Wagner, A. Kamali and J. Kengeya-Kayondo. 1994. HIV-1 incidence and HIV-1 associated mortality in a rural Ugandan population cohort. *AIDS* 8:87-92.

Ndamobissi, R., G. Mboup and E. Nguelebe. 1995. *Enquête Démographique et de Santé, République Centrafricaine, 1994-95.* Calverton MD: Macro International, Inc.

National Statistical Office (NSO), Malawi. 1994. *Demographic and Health Survey, 1992.* Calverton MD: Macro International, Inc.

Office of Population Censuses and Surveys (OPCS). 1992. 1990 Mortality Statistics. *England and Wales Series DH 1,* No. 24. London.

Sewankambo, N., M. Wawer, R. Gray, D. Serwadda, C. Li, R.Y. Stallings, S.D. Musgrave and J. Konde-Lule. 1994. Demographic impact of HIV infection in rural Rakai District, Uganda: results of a population-based cohort study. *AIDS* 8:1707-1713.

STD/AIDS Control Programme (Uganda). 1994. *HIV/AIDS Surveillance Report: June 1994.* Entebbe: Ministry of Health.

Sullivan, J., S. Rutstein and G. Bicego. 1994. *Infant and Child Mortality.* DHS Comparative Reports No. 18. Calverton MD: Macro International, Inc.

Timaeus, I. 1991. Adult mortality: levels, trends, and data sources. Pp. 87-100 in *Disease and Mortality in Sub-Saharan Africa,* ed. R. Feachem and D. Jamison. New York: Oxford University Press.

Timaeus, I. 1993. Adult mortality. Pp. 218-255 in *Demographic Changes in Sub-Saharan Africa,* ed. K. Foote, K. Hill and L. Martin. Washington DC: National Academy Press.

Todd, J., H. R. Balira, H. Grosskurth, et al. 1997. HIV-associated mortality in a rural African population. *AIDS* 11:801-807.

US Bureau of the Census. 1996. Recent HIV seroprevalence levels by country: June 1996. Research Note, No. 21. Washington DC: Health Studies Branch, Center for International Research.

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Measurement of adult mortality in populations affected by AIDS: an assessment of the orphanhood method*

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Abstract

This paper demonstrates that orphanhood data can be used to estimate adult women's mortality in populations experiencing an epidemic of AIDS. It develops both a correction for selection bias in reports of orphanhood and a revised procedure for estimating life table survivorship for use in populations with significant AIDS mortality. These new methods yield mortality estimates for a Ugandan population that are consistent with those obtained by prospective surveillance. Countries that lack effective death registration systems should ask about the survival of mothers in the census and surveys in order to monitor the effect of the AIDS epidemic on mortality.

Most developing countries lack effective systems of death registration. Measuring adult mortality in such populations has always been difficult (Timæus 1991a). The large-scale AIDS epidemics that have developed in much of Africa and some other countries have emphasized the public health importance of monitoring adult mortality levels and trends in the developing world. They have also made this task even more of a challenge. This paper commences by reviewing briefly both the existing limitations of the methods used to estimate adult mortality in the developing world and the additional obstacles to the production of such estimates that arise in populations with substantial mortality from AIDS.

All mainland sub-Saharan African countries and most other developing countries lack complete and accurate civil registration systems. In addition, most deaths occur outside hospital. Thus, no possibility exists of compiling comprehensive data on deaths by age routinely and continuously. A straightforward and relatively cheap alternative way of generating information on mortality is to ask questions in a national census or single-round household survey about deaths in the past year. Unfortunately, such questions have proved unreliable and often yield incomplete data (Timæus 1991a). Respondents may find it difficult to recall exactly when a death occurred and often have little idea of the ages of those who have died. Moreover, only very large surveys can estimate adult death rates with reasonable precision. The deaths of adults who lived alone are unlikely to be reported. Furthermore, not everyone is attached clearly to a single household. Thus, both omission of deaths and double-counting occur. Such ambiguities in the scope of questions about recent deaths in the household are particularly severe when a household head dies, as this event is likely to stimulate the fission and reformation of households.

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It seems likely that the reliability of retrospective questions about deaths in the household will be particularly poor in populations suffering from an HIV epidemic. Little direct information on this issue yet exists. However, a stigma is attached to deaths from AIDS in many cultures: therefore, they may not get reported. AIDS tends to kill young adults and any spouse may respond by moving elsewhere. Thus, the tendency of households to disperse when someone dies is probably particularly strong when the death is from this disease. Moreover, because HIV is sexually transmitted it is common for both husbands and wives to become infected. As a result a significant proportion of the spouses of those who die may become seriously ill or die during the year following the earlier death. Neither death is likely to be picked up in a single-round enquiry.

In favourable circumstances, demographic analysis can be used to assess the completeness of reports of recent deaths (Preston 1984). Most of the techniques for this, such as the Growth Balance Equation, assume that trends in mortality have had little effect on the age distribution. They are unlikely to perform well in populations that are experiencing rapid increases in mortality concentrated in a fairly narrow range of ages. Techniques of evaluation that can cope with reversals in mortality trends do exist (e.g. Bennett and Horiuchi 1981). They can be applied only in populations where the age pattern of natural increase can be measured accurately. Thus, these methods have a poor record in regions such as Africa, where age reporting is poor and flows of international migrants are large (Timæus 1993).

Collecting information prospectively could prevent many of the reporting errors that afflict retrospectively collected direct data on adult deaths. Experience shows, however, that mounting a successful multiround survey of a representative sample of a national population is both expensive and difficult (Cleland 1996). This design multiplies the costs involved in interviewing a large sample. In addition, few multiround surveys have succeeded at maintaining high-quality field operations for the year or more needed to complete such a survey.

Studies involving the demographic surveillance of a geographically-localized population have proved more successful. Close supervision of the field operations by senior staff becomes possible. Such demographic surveillance systems have usually been set up as part of a program of research involving epidemiological and social as well as demographic studies. This greatly increases the benefits gained from demographic surveillance and makes it easier to justify the costs involved.

In response to the need for mortality data for health research, several prospective studies have been established in Africa that involve the demographic surveillance of a local population (e.g. Mulder et al. 1994; Sewankambo et al. 1994; Tollman, Herbst and Garenne 1995; Boerma et al. 1997). Those studies that collect serological data have generated valuable information on mortality and related aspects of the AIDS epidemic. The approach is not suited, however, to the measurement and monitoring of mortality in national populations. Indeed, complementary methods that are able to produce national estimates of mortality have to be deployed in order to gain the maximum benefits from intensive research studies. Without such data, it is impossible to gauge how far the health situation in areas subjected to intensive study is representative of wider populations, and thus what the import of the findings of such studies is for health policy and planning.

Several approaches to the measurement of adult mortality have been proposed that are intended to circumvent the problems that arise in collecting data on deaths directly (Timæus 1991a). One of the most important is the orphanhood method of estimating adult mortality (Brass and Hill 1973). This uses data collected by asking two simple questions, 'Is your mother alive?' and 'Is your father alive?'. These questions do not require respondents to recall the dates when deaths occurred or the ages at death of deceased individuals. They can be asked in single-round enquiries. Conventional life-table measures of adult mortality can be

derived from the answers using demographic models that relate the proportions of the population by age with a living mother (or father) back to the mortality conditions that produced them.

The idea of collecting information on parents to estimate adult mortality can be extended to other categories of relatives such as spouses and siblings. Asking about the deaths of first husbands and wives has seldom been a success. The widowed frequently report that they are single and the divorced that they are widowed. In particular, in much of Africa marrying someone is a lengthy process, which offers respondents opportunities to redefine their union history in socially acceptable ways. Moreover, co-infection of spouses with HIV, followed by the death of them both, will lead to substantial biases in adult mortality estimates from this source. Data about deaths of adult siblings would be less subject to this type of bias; however, methods for analysing them were only developed recently (Timæus, Zaba and Ali forthcoming) and few such data exist. A number of DHS surveys have collected full sibling histories that ask whether and when brothers and sisters died and about ages and ages at death. Such histories can be used to measure adult mortality without recourse to indirect estimation methods. Despite the difficulty of collecting this information accurately, the approach has yielded promising results (Rutenberg and Sullivan 1991; Timæus 1997).

Data on orphanhood have been collected far more frequently than data on the deaths of other adult relatives. Their quality has varied (Timæus 1991a): in East Africa, in particular, it is common for too few respondents to report that their parents are dead. It appears that a proportion of them instead answer the questions in terms of their foster-parents or step-parents. In many applications, however, comparison of successive sets of orphanhood data and of orphanhood-based and other estimates of adult mortality suggests that the method has worked well. Information on orphanhood has made a large contribution to our existing knowledge of adult mortality in Africa (Timæus 1993). If for no other reason than the inertia of data collection agencies, it also seems likely that it will continue to be collected. It therefore seems important to assess whether this source of mortality data continues to be useful in the face of the AIDS epidemic.

An inherent limitation of the orphanhood method is that data on parents' survival can only be collected from those of their offspring who themselves remain alive. In general, the selection bias that arises from this is small (Palloni, Massagli and Marcotte 1984). In populations affected by AIDS, however, the problem is likely to be more severe. HIV-positive women can transmit the virus to their children during pregnancy, at delivery, or shortly after birth. Thus, the children of women who are at highest risk of dying are also likely to suffer higher mortality than the population in general. Because women are likely to have been infected by or to infect their husbands, orphanhood-based estimates of adult men's mortality in populations affected by HIV may also be biased downwards, though to a lesser degree than those for women. A second major HIV-related bias in orphanhood estimates is that the data are usually converted into conventional life table indices of mortality using a set of coefficients based on very different age patterns of mortality in adulthood from those found in populations experiencing an AIDS epidemic.

A number of investigations have been conducted of the effect of the AIDS epidemic on orphanhood (e.g. Prebble 1990; Palloni and Lee 1992; Gregson, Garnett and Anderson 1994). The aim of this paper is to answer the inverse question: can information on orphanhood be used to measure adult mortality in populations affected by AIDS? Our investigation of this issue makes use of data from a longitudinal study of a population in rural Uganda that has collected information on serostatus, seroconversion, fertility, and mortality. This study is described briefly in the next section of the paper. The following three sections of the paper are theoretical. The first of them presents a mathematical model of the determinants of orphanhood in populations with significant AIDS mortality. Then, using values for the

parameters based on the Ugandan study, this model is deployed to quantify the main HIV-related biases in orphanhood estimates of adult women's mortality and to develop methods that correct for them. Finally, these methods are applied to orphanhood data collected in the Ugandan study. The mortality estimates that result are evaluated by comparing them with direct measures obtained prospectively by surveillance of the population.

The MRC study in Masaka district

The area of study is a rural subcounty of Masaka district in southwest Uganda. It is situated approximately 20 miles from Masaka town and ten miles from the trans-African highway. The study covers 15 neighbouring villages with a population of about 10,000. Most of the population are peasant farmers; they grow bananas as a subsistence crop and cultivate coffee for sale. The predominant tribal group, the Baganda, constitute about 70 per cent of the population. Substantial immigration of Rwandese occurred up to the 1970s.

Beginning in late 1989, the study villages were mapped and a *de jure* census and socio-economic questionnaire was administered to an adult member of each household, preferably its head. Within four weeks of these interviews, a medical team visited each household. All residents were invited to participate in a survey that included a brief medical history, a physical examination, and the collection of a blood sample. Absentees and refusals were revisited to encourage them to participate.

Blood specimens were transported at weekly intervals to the laboratory of the Uganda Virus Research Institute in Entebbe where they were tested for antibodies to HIV-1. All sera were tested using two EIA systems, Recombigen HIV-1 EIA (Cambridge Biotech Corporation, Worcester MA) and Wellcozyme HIV-1 Recombinant (Wellcome Diagnostics, Dartford, England) with Western blotting using Novopath HIV Immunoblot (Bio-Rad laboratories, Watford, England) when indicated (Nunn et al. 1993, 1994). None of the field workers was aware of the HIV status of study participants. Trained counsellors made results available to all those who requested them (Seeley et al. 1991).

At annual intervals (Rounds 2-6, 1990-95), the census team revisited each household to ascertain the vital status of those who were resident at the previous survey and to enumerate those who had joined the household through either birth or in-migration. As in Round 1, the medical team collected a blood sample from everyone willing to provide one. Children aged less than 13 years were not bled after Round 3.

At the beginning of Round 3, monthly registration of births and deaths was introduced to supplement the data obtained from the annual surveys. Questions were asked in Rounds 2 and 3 about whether the mothers and fathers of children aged less than 15 years were alive and the dates when deceased parents died (Kamali et al. 1996). The results presented here are based on the orphanhood data collected in Round 3. The information was obtained successfully for more than 98 per cent of children.

The mortality, seroprevalence and HIV incidence rates used here are based on person-years of observation from the time individuals were enrolled in the study (the date of their first seropositive or seronegative specimen) until the date of the fifth annual resurvey (Round 6), for those known to be alive; the date of death, for those known to be dead; or, for those migrating out of the study area, their date of departure. Individuals who seroconverted are counted as seronegative until the midpoint between their last negative and first positive antibody test; thereafter they are counted as seropositive. The seroprevalence rate among adults was about eight per cent in each round of the study (Mulder et al. 1995). Nearly 12 per cent of the women of childbearing age are infected with HIV. Death rates among infected adults are an order of magnitude higher than those among the seronegative population (Mulder et al. 1994; Nunn et al. 1997).

Methods of analysis

Let the number of children born *a* years before a demographic survey to women who were then aged *y* be *C(a,y)*. Integrating over all ages at childbearing *a* to *b*, the total number of mothers who are still alive is (Brass and Hill 1973):

$$N(a) = \int_a^b C(a,y)\,{}_ap_y\,dy$$

where ${}_ap_y = l(y+a)/l(y)$, that is the life table probability of surviving from age *y* to *y+a*.

To model the effect of the HIV epidemic on maternal orphanhood, we distinguish infected (seropositive) women, including those who have progressed to AIDS, from women who are uninfected (seronegative). Women who were seropositive when they gave birth *a* years ago have lower survivorship, ${}_ap_y^+$, than the equivalent seronegative women, whose survivorship is denoted ${}_ap_y^-$. In addition, seropositive women probably have lower fertility than other women (Gregson 1994). Thus, they have relatively few children to report on them. If $C^+(a,y)$ represents the seropositive women who would have given birth *a* years ago at age *y* if it was not for their reduced fertility and *F(a,y)* is the age-specific ratio of the fertility of seropositive to seronegative women at that time, the total number of seropositive women who gave birth *a* years ago and remain alive is:

$$N^*(a) = \int_a^b F(a,y)C^+(a,y)\,{}_ap_y^+\,dy$$

However, the number of living seropositive women who would have given birth *a* years ago if their fertility was the same as that of other women is:

$$N^+(a) = \int_a^b C^+(a,y)\,{}_ap_y^+\,dy$$

Those women who were not infected when they gave birth may become so later. If $C^-(a,y)$ is the number of uninfected women who gave birth *a* years ago at age *y*, the total number of mothers who gave birth at that time remaining alive and uninfected is:

$$N^-(a) = \int_a^b C^-(a,y)\,{}_ap_y^-\,{}_av_y\,dy$$

where ${}_av_y$ is the probability of remaining uninfected between ages *y* and *y+a*:

$${}_av_y = e^{-\int_y^{y+a} m^i(z)\,dz}$$

and $m^i(z)$ is the instantaneous rate of infection at age *z*. Mothers who become infected between ages *y* and *y+a* subsequently have an increased risk of dying. We assume that this risk is the same as that of other infected women of their age. Thus, the number of mothers who gave birth *a* years ago and have become infected since but remain alive is:

$$N^i(a) = \int_a^b \int_y^{y+a} N^-(z) m^i(z)_{y+a-z} p_z^+ dz dy$$

The proportion still alive of all mothers who were not infected when they gave birth is:

$$S^i(a) = \frac{N^-(a) + N^i(a)}{\int_a^b C^-(a,y) dy}$$

In total, the proportion still alive of women who would have given birth *a* years ago in the absence of any impact of HIV infection on fertility is:

$$S(a) = \frac{N^+(a) + N^-(a) + N^i(a)}{\int_a^b C^+(a,y) + C^-(a,y) dy} \quad (1)$$

Note that $C^+(a,y)+C^-(a,y) = C(a,y)$.

A proportion, *h*, of the children of seropositive mothers have the virus transmitted to them perinatally. In general, this study assumes that *h* = 25 per cent but it also explores the effect of a vertical transmission rate of 35 per cent (Ryder and Temmerman 1991; Lepage et al. 1993; Newell forthcoming). Few of these children live long (Newell forthcoming). If we make the somewhat pessimistic assumption that all of them die in the first five years of life, then *(1-h)l(a)* of them remain alive at age five and over.[1] As discussed in the previous section of the paper, we assume that, apart from vertical transmission, the mortality of orphans and children with living parents is the same. Thus, the proportion of respondents aged *a* who report that their mother is alive is:

$$S^*(a) = \frac{(1-h)N^*(a) + N^-(a) + N^i(a)}{\int_a^b (1-h)F(a,y)C^+(a,y) + C^-(a,y) dy} \quad \text{for } a \geq 5 \quad (2)$$

To assess the biases in orphanhood estimates of mortality, we compare estimates of *S(a)* and *S*(a)* calculated from Equations 1 and 2 with each other and with life table survivorship, using values for ${}_a p_y^+$, ${}_a p_y^-$ and *l(a)* obtained in the Masaka district study (Mulder et al. 1994; Nunn et al. 1997). The same study provides information on the serostatus and ages of women giving birth. Much of the initial impact of the HIV epidemic on age-specific mortality and age structure respectively has already been felt. Although further changes will occur, current demographic patterns can be used to model the approximate effect of the epidemic on patterns of orphanhood in the longer run.

Because the counts of births by age for seropositive women have large sampling errors, we assume that, although seropositive women have lower fertility than uninfected women, their age pattern of fertility is the same. Thus, $C^+(a,y)$ and $C^-(a,y)$ are calculated by

[1] This assumption will exaggerate rather than underestimate biases in the orphanhood data.

smoothing the age-specific fertility distribution for all women and applying it to the populations of seropositive and seronegative women respectively. The indirectly standardized fertility rate for seropositive women in the study in Masaka district is 27 per cent lower than the rate for seronegative women but has a wide confidence interval (Carpenter et al. 1997). Our main assumption is that seropositive women have 20 per cent lower fertility than seronegative women but we assess the sensitivity of our results to this assumption by also modelling the effect of a 40 per cent reduction in fertility (Ryder et al. 1991; Sewankambo et al. 1994; Serwadda et al. 1997). Thus, $F(a,y)$ simplifies to F=0.8 or F=0.6 for all a and y, $N^*(a)$ becomes $F.N^+(a)$, and Equation 2 simplifies to:

$$S^*(a) = \frac{(1-h)F.N^+(a) + N^-(a) + N^i(a)}{\int_a^b (1-h)F.C^+(a,y) + C^-(a,y)dy} \quad \text{for } a \geq 5 \qquad (3)$$

To model $N^-(a)$ and $N^i(a)$ one needs data on the incidence of HIV infection by age. Incidence rates are available for a subset of the cohort under study in Masaka district (Kengeya-Kayondo et al. 1996). Applied to a synthetic cohort, they yield a net lifetime risk of infection for women of 29 per cent. In contrast, the current mortality and seroprevalence data suggest that 37 per cent of adult women in the Masaka district study will die of AIDS. This discrepancy emphasizes that the demography and epidemiology of HIV in this area have yet to stabilize. It may reflect a recent decline in the incidence of HIV that has yet to be reflected in prevalence (Mulder et al. 1995). In order to match the incidence rates to the current prevalence of infection, therefore, they were scaled up to give a net lifetime risk of infection of 40 per cent.[2] This yields a prevalence-incidence mean survival time from infection of 6.7 years.

The integrals in Equations 1 and 3 are evaluated by summing data for five-year age group of mothers for respondents at five-year age intervals, a = 5, 10, ... 30, before interpolating to obtain results for age groups of respondents.[3] Most mothers of respondents

[2] The incidence rates have large sampling errors and were smoothed using a relational Gompertz model against a standard fertility schedule that was stretched to age 60 to allow for the infection of women aged 50-59 (Paget and Timæus 1994). The resulting single-year incidence schedule is used to calculate five-year cohort-period probabilities of HIV infection.

[3] To implement the discrete models, the probabilities that the members of a cohort become infected in each five-year age interval allowing for prior mortality from diseases other than AIDS, ${}_5u_z^g$, are calculated as:

$${}_5u_z^g = {}_5u_z\left(1 - \frac{{}_5u_z \cdot {}_5q_z^-}{{}_5u_z + {}_5q_z^-}\right)$$

where ${}_5u_z = 1 - {}_5v_z$ and ${}_5q_z^- = 1 - {}_5p_z^-$. Probabilities of dying before becoming infected are calculated using the equivalent equation. The proportion of respondents in each five-year age group with living mothers, ${}_5S_x$, is calculated by interpolating between the point estimates for five-yearly intervals assuming that births grew at a constant rate, r, of 2 per cent across each age group:

$${}_5S_x = \frac{l(x)S(x) + e^{-2.5r}\frac{l(x)+l(x+5)}{2}\sqrt{S(x)S(x+5)} + e^{-5r}l(x+5)S(x+5)}{l(x) + e^{-2.5r}\frac{l(x)+l(x+5)}{2} + e^{-5r}l(x+5)}$$

aged 30 are 50 to 65 years old. Few of them will die of AIDS in the future. Thus, the relationship between life table survivorship and orphanhood in the age group 25-29 years should indicate the nature of this relationship in older age groups.

There are problems with using data from the Masaka district study to implement our model of orphanhood. The epidemiology and natural history of HIV in this area may be atypical. The data are based on a fairly small number of person-years of observation for the measurement of demographic statistics. Moreover, while trends in the prevalence of HIV infection in the study population have been muted for the last few years, the age distribution of women giving birth has been changing and will continue to do so. Nevertheless, it seems useful to model biases in orphanhood data using demographic parameters from a real population rather than parameters derived from an epidemiological model of the HIV epidemic that itself cannot be validated, especially with respect to AIDS mortality.

HIV-related selection bias

The first column of results in Table 1 shows estimates by age of respondent of the proportions alive of mothers who have never contracted HIV. These women are subject to the background mortality of the population and the proportions were calculated using data on the age structure and mortality of the seronegative population in Masaka district. The second column shows estimates of the proportions of people born to seronegative women who still have living mothers. The increase in orphanhood, compared with the previous column, reflects the mortality of women who became infected with HIV after they gave birth to the index child. The third column of simulation results refers to women who were already infected when they gave birth. Nearly half of them die while the index child is aged less than five years. The fourth column shows the overall proportions still alive of women who would have given birth if serostatus had no impact on fertility. These data equate to the proportions of living mothers that would be obtained in a survey if infected and uninfected women were equally likely to be reported on. In contrast, the last but one column shows the estimated proportions of mothers who would actually be reported to be alive in a survey, allowing for both the higher mortality of vertically infected children and the reduced fertility of infected women. Note that these estimates differ from those that would have been obtained by collecting data in Masaka district in the early 1990s. They represent the prevalence of orphanhood that would result from the current demography of the population: orphanhood in the early 1990s reflected demographic patterns during the previous 30 years.

Table 1
Expected proportions of respondents with living mothers.

Age group (x to $x+5$)	0-4	5-9	10-14	15-19	20-24	25-29
Mother subject to background mortality ($_5S_x^-$)	99.2	97.2	95.1	92.7	89.6	85.1
Mother not infected at birth ($_5S_x^i$)	98.6	94.7	89.5	83.9	78.0	71.4
Mother infected at birth ($_5S_x^+$)	81.6	44.2	23.2	11.6	5.6	2.6
Total if no process of selection[a] ($_5S_x$)	96.1	87.6	80.2	73.8	67.9	61.8

Total that would be reported[b] $({}_5S_x^*)$	97.0	90.2	83.6	77.5	71.6	65.3
Adjusted total reports $({}_5S_x')$	-	87.6	78.7	73.0	67.4	61.5

[a] Assuming that seropositive and seronegative women have identical fertility and that their children have identical mortality.

[b] Assuming that the fertility of women infected with HIV is reduced by 20 per cent and a 25 per cent vertical transmission rate.

The most important conclusion to be drawn from Table 1 is that one should expect fairly small biases in reports on the survival of parents in populations affected by AIDS. According to the Masaka district based simulation, the errors in the reported proportions of mothers alive rise from about one per cent for respondents aged less than five years to nearly four per cent in the three oldest age groups considered. Thus, the simulated reports are much closer to unbiased figures for the population than to what would be reported in the absence of the HIV epidemic. This should also be true of populations with epidemics that are more severe or less severe than that in Masaka. The reason why the selection bias is fairly small is that reduced fertility and vertical transmission are unlikely to select out more than half of the reports on infected women: the simulated results shown in Table 1 assume that 40 per cent of such reports are missing. From a lifetime perspective, our data on incidence and mortality imply that women who become infected with HIV bear about two-thirds of the children that they would have had otherwise. They have about 60 per cent of these births before becoming infected.

Simplification of Equations 1 and 2 yields a useful approximation for the selection biases in data on orphanhood. Assume that all the women who are seropositive when they give birth die in the following five years, so that $N^+(a)$=0 for respondents aged at least five years. This assumption makes the numerators of Equations 1 and 2 identical so that, dividing the former by the latter:

$$\frac{S(a)}{S^*(a)} = \frac{(1-h)\int_a^b F(a,y)C^+(a,y)dy + \int_a^b C^-(a,y)dy}{\int_a^b C^+(a,y)dy + \int_a^b C^-(a,y)dy} \qquad (4)$$

Assume also that the seroprevalence rate, *P*, among women seen in antenatal clinics is a measure of the proportion of all women giving birth who are infected, so that:

$$P = \frac{\int_a^b F(a,y)C^+(a,y)dy}{\int_a^b F(a,y)C^+(a,y)dy + \int_a^b C^-(a,y)dy} \qquad (5)$$

Dividing Equation 4 though by the denominator of Equation 5, one obtains:

$$\frac{S(a)}{S^*(a)} = \frac{(1-h)P + (1-P)}{\frac{P}{F} + (1-P)} = \frac{1-hP}{1+\frac{1-F}{F}P} \qquad (6)$$

Thus, the bias in the reported proportion of respondents with living mothers increases with prevalence at a rate determined by *F* and *h*. As one would expect, the higher vertical transmission, and the lower the fertility of infected women, the greater the bias in reports on the survival of mothers. In practice, estimates of seroprevalence are more widely available than estimates of vertical transmission or of the reduction in the fertility of the infected. Substituting our assumptions that the fertility of infected women is 20 per cent lower than that of uninfected women and that the vertical transmission rate is 25 per cent into Equation 6, produces a simple correction factor for selection bias:

$$ {}_5S'_x = \frac{1-0.25P}{1+0.25P} {}_5S^*_x \approx (1-0.5P)\,{}_5S^*_x \qquad (7) $$

To allow for the survival for more than five years of a significant proportion of mothers who were seropositive when they gave birth, the adjustment can be halved for respondents aged 5 to 9 so that ${}_5S'_5 = (1-0.25P)\,{}_5S^*_5$.[4] These correction factors can be revised using Equation 6 when more is known about *F* and *h*.

The final column of Table 1 shows the adjusted proportions of mothers alive, ${}_5S'_x$, that result from correction of the data in the previous column using Equation 7. They are close to ${}_5S_x$, the unbiased values calculated using Equation 1. Table 2 investigates whether the correction factor of 1-0.5*P* yields adequate results in populations where the reduction in the fertility of the seropositive and the vertical transmission rate is unknown. Even in the presence of HIV-related mortality, nearly all the mothers of young respondents are alive. Thus, both the absolute and the relative errors in the data on survival of mothers seem small even though they imply substantially different mortality from that actually prevailing. To convey the significance of the errors more accurately, therefore, they are presented in terms of the ratio of the estimated to actual odds of mothers surviving:

$$ \frac{{}_5S'_x}{1-{}_5S'_x} \cdot \frac{1-{}_5S_x}{{}_5S_x} $$

Table 2
Estimates of the bias in reported and adjusted odds that mothers are alive [a].

	Model reports	Adjusted model reports				
Age group	Baseline assumptions	Baseline assumptions	40% reduction in fertility of the seropositive	35% vertical transmission of HIV	Higher non-HIV mortality (e_{10}=46.9)	Twice the prevalence of HIV

[4] If a population-based estimate of seroprevalence among women of childbearing age, P^*, is available, rather than one based on antenatal data, one must assume that the proportion of births to seropositive women aged *y* can be estimated from P^*, so that $C^+(a,y) = P^*.C(a,y)$. Dividing Equation 1 by Equation 3, one obtains:

$$ \frac{S(a)}{S^*(a)} = 1-(1-(1-h)F)P^* $$

With *F*=0.8 and *h*=0.25, the correction factor is:

$$ {}_5S'_x = (1-0.4P^*)\,{}_5S^*_x $$

5-9	1.298	0.989[b]	1.164[b]	1.046[b]	0.990[b]	0.963[b]
10-14	1.257	0.914	1.063	0.953	0.924	0.871
15-19	1.223	0.960	1.092	0.998	0.965	0.932
20-24	1.192	0.980	1.095	1.014	0.982	0.961
25-29	1.164	0.989	1.088	1.019	0.990	0.976

[a] Baseline assumptions: 11.8% seroprevalence among women aged 15-49 years, 20% lower fertility among the seropositive, 25% vertical transmission, background life expectancy at age 10 (e_{10}) of 54.2 years.

[b] Adjusted using a correction factor of 1-0.25*P*.

The first two columns of ratios in Table 2 correspond to the last two columns of Table 1. According to our model, the odds that the mother of a living respondent is alive are 15 to 30 per cent higher than the odds for all women who would have given birth if serostatus did not affect fertility. The errors in the odds decrease slightly with age. Adjustment of reported data using the correction factor given in Equation 7, however, should produce nearly correct estimates. The next two columns of Table 2 shed light on how sensitive adjusted data are to the assumptions made about size of the reduction in fertility of seropositive women and the level of vertical transmission. The third column of ratios also uses data from Masaka district on the ages and mortality of infected and uninfected women to calculate ${}_5S_x^*$ and ${}_5S_x'$. However, it is assumed that the fertility of seropositive women is 40 per cent below that of seronegative women, rather than 20 per cent lower. Similarly, the next column illustrates the impact of a 35 per cent, as compared with a 25 per cent, vertical transmission rate. As one would expect, the survival of mothers tends to be overestimated even after correction by 1-0.5*P*. In both scenarios, however, the errors in the odds are moderate and the adjusted estimates much more accurate than the unadjusted ones.

The derivation of the correction factor suggests that it should be valid whatever the level of background mortality or seroprevalence. The last two columns of Table 2 confirm this. A correction factor of 1-0.5*P* operates equally well in a model population with much higher background mortality than the baseline model and nearly as well in a model population in which the net lifetime risk of infection is raised to 67 per cent in order to double the prevalence of infection among women of childbearing age.

The model data support the theoretical argument that Equation 6 provides a basis for correcting orphanhood data for selection bias. They also suggest that the further simplification presented in Equation 7 is a robust one: while information on the fertility of infected women and vertical transmission rate can be used to refine the adjustment, it is not essential. This method of adjustment would perform less well for young respondents in a population where infected women survived longer than in Masaka district. Too little is known about the natural history of HIV infection in developing countries to investigate the importance of such differences thoroughly. It seems unlikely, however, that survival times vary enough across the developing world to affect greatly the conclusions drawn from Table 2.

Bias in the estimation procedure

The second source of bias in orphanhood-based estimates of adult mortality for populations affected by AIDS lies in the regression equations used to convert proportions of respondents with living mothers into life table probabilities of surviving from age 25. These equations take the form:

$$ {}_np_{25} = b_0(n) + b_1(n)M + b_2(n){}_5S_{n-5} $$

where M is the mean age at childbearing and is a control for variation between populations in the ages over which the mothers are exposed to the risk of dying. Deaths from AIDS are concentrated among young adults. Therefore, the epidemic produces age patterns of mortality that differ from those in the mortality models used to estimate the coefficients of the estimation equations (Timæus 1997).

Table 3
Ratios of the odds of estimated to actual life table survivorship ($_np_{25}$).

	Existing coefficients	Revised coefficients			
Years of survivorship from age 25	Unbiased proportions	Unbiased proportions	Adjusted model reports	Unbiased proportions. (HIV prevalence 23.6%)	Unbiased proportions. (Background e_{10}=46.9)
10	1.283	1.002	0.997	1.011	1.023
15	1.174	1.038	0.933	1.041	0.958
20	1.037	1.035	0.990	1.042	0.978
25	1.037	1.039	1.017	1.054	0.985
30	1.007	1.008	0.996	1.025	1.029

The first column of results in Table 3 shows the biases in estimated life table survivorship produced by applying the existing orphanhood method of estimation to unbiased proportions of respondents with living mothers ($_5S_x$). The errors are again presented as odds ratios.[5] The estimates were made from the simulated data presented in Table 1 using the regression-based procedure proposed by Timæus (1992).[6] The results would be very similar if another of the existing estimation procedures was used. They are compared with the information on survivorship in Masaka district that was used to generate the simulated data in the first place. Thus, the ratios in the first column indicate the errors that result from the use of regression equations to estimate life table survivorship that do not take into account the unusual age pattern of mortality in populations experiencing AIDS mortality. The life table odds of surviving are overestimated substantially from data on the first two age groups. In contrast, the biases in estimates based on the reports of respondents aged more than 15 years are small.

The reason why the existing coefficients overestimate life table survivorship from 25 to 35 and 40 years is that seroprevalence among women in Masaka district peaks among women in their mid-twenties. A smaller proportion of all women giving birth are infected with HIV than of women aged 25. Therefore, the proportion of mothers who survive ten years is higher than life table survivorship from 25 years to 35 years. On the other hand, mothers as a whole are more likely to become infected in the years after they give birth than are women aged 25 as the younger mothers are still passing through the peak ages of infection. After about 15 years, the close relationship that exists in populations unaffected by HIV between the proportion of mothers alive and life table survivorship from age 25 is re-established. By

[5] Note that these odds ratios, R, define the error in a, the level parameter of any relational logit system of model life tables (Brass 1971), irrespective of the choice of a standard mortality schedule:

$$a^* - a = -0.5 \ln R$$

[6] We use a mean age of childbearing of 26.4 years calculated from the follow-up data on births by age of mother.

the time the respondents are aged 25 years or more, the bias in the existing coefficients can be ignored for all practical purposes.

The more severe the epidemic, the higher the ratio of mothers infected at birth to those infected subsequently and the greater the bias in the existing coefficients for data on 5-9 and 10-14 year old respondents. This is illustrated in Figure 1 for respondents aged 10-14 years and $_{15}p_{25}$. The data points are for prevalences of 2.9, 5.9, 11.8, 17.7, 23.6 and 35.3 per cent among women of childbearing age and two levels of background mortality, that prevailing in Masaka district and a higher mortality life table in which e_{10} is 46.9 not 54.2 years. In essence, AIDS mortality steepens the slope of the line linking the survival of mothers to the survivorship of adult women.

Figure 1
Relationship between the survival of the mothers of respondents aged 10-14 years ($_5S_{10}$) and life table survivorship from 25 to 40 years ($_{15}p_{25}$) for different prevalences of HIV infection, estimates based on data from Masaka district.

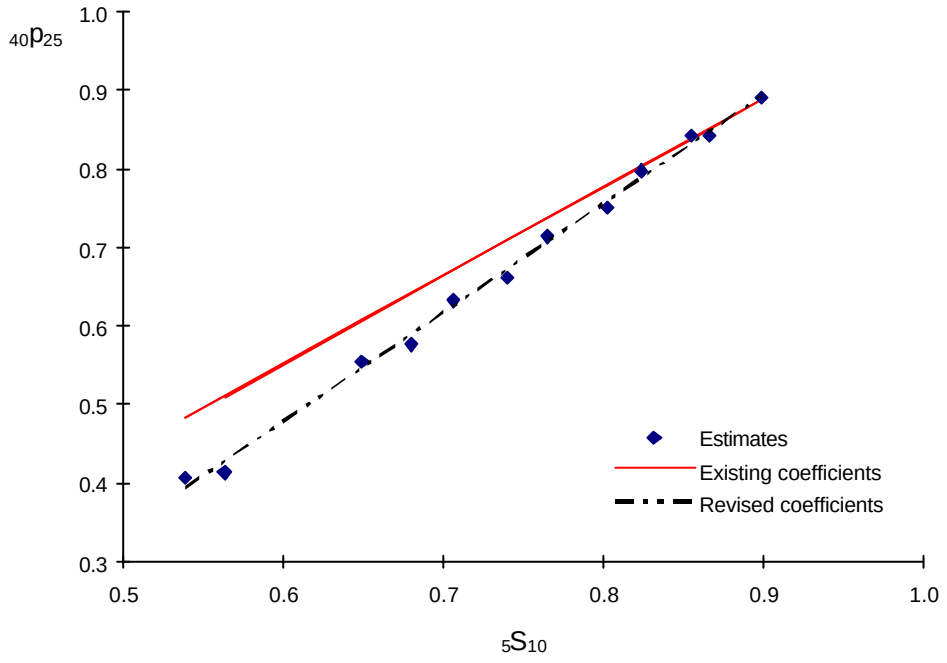

Figure 1 reveals that for respondents aged 10-14 years the relationship between survival of mothers and life table survivorship from 25 to 40 years remains close to linear. This is also true of all other age groups. This means that one can estimate revised coefficients for the estimation of survivorship from orphanhood data in populations subject to significant AIDS mortality. These are presented in Table 4. For respondents aged less than 25 years the regression lines have lower intercepts and steeper slopes than in existing estimation procedures. For the 25-29 year age group, however, the regression line is almost identical to that proposed by Timæus (1992).

Table 4
Provisional revised coefficients for estimating life table survivorship from 25 to 25+*n* years from the proportion of the mothers alive of respondents aged *n*-5 to *n*.[a]

Age (*n*)	$b_0(n)$	$b_1(n)$	$b_2(n)$
10	-0.3611	0.00125	1.2974
15	-0.4030	0.00222	1.3732
20	-0.2120	0.00372	1.1342
25	-0.2389	0.00586	1.1131
30	-0.2513	0.00885	1.0223

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

The regression coefficients in Table 4 are provisional. They are based entirely on data from Masaka district. In particular, the age pattern of incidence relative to that of childbearing and the distribution of survival times of the infected differ between populations. Few data exist with which to investigate how predicted survivorship varies with these characteristics of AIDS epidemics and we do not know whether the population in the Masaka district study is typical in these respects. The finding that the biases in the existing coefficients are negligible for older respondents should apply to all populations. The revised coefficients would not be ideal, however, for a population in which prevalence peaked among older women, not those in their twenties.[7] Nevertheless, as even methods that make no allowance for the distinctive age pattern of AIDS mortality yield moderate errors, as demonstrated in the first column of Table 3, we expect the revised coefficients to be robust to variation in those epidemiological parameters of AIDS epidemics that are not investigated here.

Returning to Table 3, the second column of results shows that, when survivorship is estimated using the revised regression coefficients, very accurate results are obtained. Minor errors persist because the points linking survival of mothers and survivorship at a prevalence of 11.9 per cent do not fall exactly on the regression lines. In the third column of results, life table survivorship has been estimated from the adjusted proportions of mother alive (${}_5S'_x$) produced by application of the correction factor given in Equation 7. As one would expect from the results in Table 2, the estimated odds of surviving still differ only slightly from the actual values for Masaka district. The remaining two columns present errors in the estimated odds for populations in which mortality is higher and orphanhood more prevalent than in the baseline model because the prevalence of infection and of background mortality respectively is higher. The errors are again small.

Orphanhood and adult mortality in Masaka district

This section of the paper uses the results obtained so far to analyse orphanhood reports collected in the Masaka district study. Such data have been collected only from children. Estimates of adult women's mortality based on them are compared with direct measures of the mortality of adult women calculated from data obtained prospectively. As the study collected

[7] Including the level of seroprevalence in the regression model does not improve the prediction of ${}_np_{25}$ from these data because seroprevalence is very closely associated with the level of orphanhood. While adding seroprevalence to the model might improve precision if a more diverse set of data was modelled, an index of the age pattern of infection would probably be of more value. Obtaining the data required first to fit and then to use such a model is probably impossible at present.

information on when parents died, it is possible to calculate not only the proportion of respondents with living mothers at the time of the survey but also the proportions of women who were alive five and ten years earlier. Using these three series of proportions, one can calculate the proportion of mothers alive in synthetic cohorts for the periods 0-4 and 5-9 years before the Round 3 enumeration. These synthetic data reflect the level of orphanhood that would result if a cohort went through childhood experiencing the rates of orphanhood of 1987-92 and 1982-87 respectively.

Table 5
Reported and adjusted proportions of mothers alive and estimated survivorship of women, Masaka district.

Age	1982	1987	1992	1982-87	1987-92
Reported proportions of mothers alive					
0-4	0.9946	0.9888	0.9837		
5-9		0.9755	0.9301		
10-14			0.9230		
Adjusted proportions of mothers alive					
0-4	0.9946	0.9888	0.9837	0.9888	0.9837
5-9		0.9473	0.9027	0.9417	0.8980
10-14			0.8695		0.8244
Survivorship of women					
${}_{10}p_{25}$		0.9009	0.8431	0.8937	0.8270
${}_{15}p_{25}$			0.8497		0.7876

Table 5 presents the reported data, together with proportions adjusted for selection bias using the correction factor given in Equation 7. The prevalence of orphanhood has been increasing rapidly in Masaka district. A higher proportion of children are orphaned in each successive cohort and more children were orphaned in 1987-92 than 1982-87. Table 5 also presents estimates of life table survivorship. These estimates were derived from data on the age groups 5-9 and 10-14 years using the coefficients presented in Table 4. They reveal that a rapid decrease in the survivorship of adult women has occurred between the two cohorts and two periods on which data are available.

Figure 2
Orphanhood and direct estimates of women's survivorship from 25 to 35 years, Masaka district.

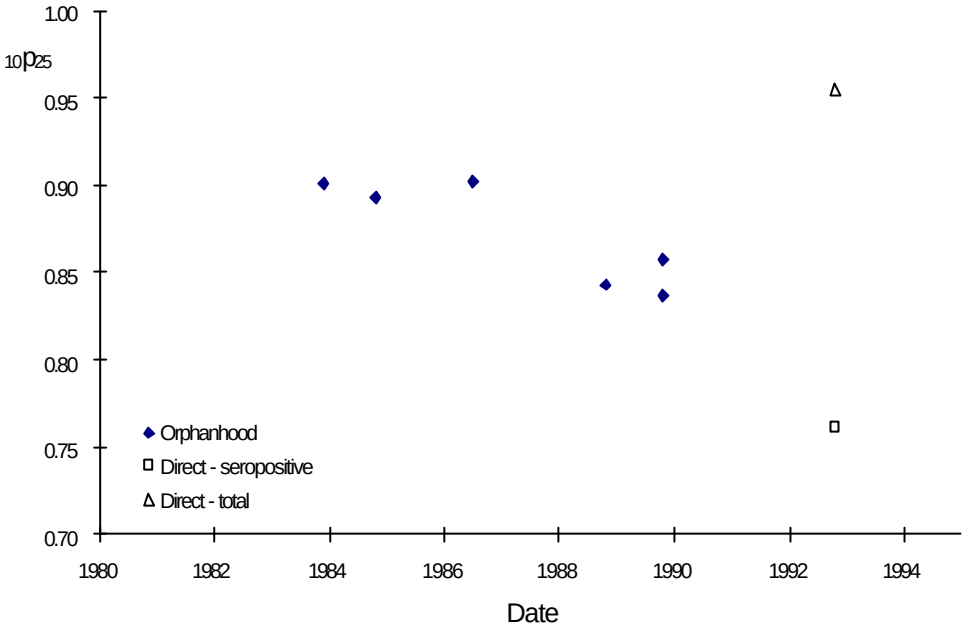

Figure 2 examines the time trend in the survivorship of women. It compares the orphanhood estimates with those calculated directly from the follow-up data. The time location of those orphanhood estimates based on cohort data was estimated using the standard procedure (Brass and Bamgboye 1981). The orphanhood estimates are somewhat erratic. This is to be expected. It reflects misreporting of orphanhood status, dates of death, and children's ages and also the approximations involved in adjustment of the proportions and estimation of survivorship. In addition, chaining the cohort proportions to produce proportions for synthetic cohorts tends to magnify any errors in the original data. Despite these potential problems, the estimates document a rapid increase in women's mortality between 1983 and 1989. They all indicate a level of survivorship intermediate to that in the total population and that in the seronegative population for 1990-95. Extrapolating the trend in the orphanhood estimates backward suggests that early in the 1980s the total population had the level of mortality now found in the seronegative population. Extrapolating forward yields an estimate of survivorship for 1993 that is only slightly higher than that calculated directly. Thus, the orphanhood method works well in this application. To a limited extent this is because the revised regression coefficients were developed using data from this population. On the other hand, the derivation of the procedure proposed for adjusting for selection bias was not tailored to Masaka district.

Discussion

This paper investigates the biases to be expected in orphanhood-based estimates of adult mortality in populations where HIV-related deaths occur in significant numbers. It presents a mathematical model of the relationship between mortality and the survival of mothers in populations affected by the AIDS epidemic. The model is operationalized using demographic data collected during five years of surveillance of a rural Ugandan population. The analysis reveals that the majority of the children of women who die from AIDS are born before their mothers become infected. As only about a quarter of these women's remaining children are infected vertically, selection biases originating in vertical transmission lead the orphanhood method to overestimate adult survivorship by only a few percentage points. Reductions in the fertility of seropositive women can produce a comparable additional error. Apart from among the youngest respondents, this bias is a function of seroprevalence among women of childbearing age, the vertical transmission rate and the amount by which infected women's fertility is reduced. A simple way of adjusting for the bias is proposed that can be used whenever an estimate is available of seroprevalence among either pregnant women or all women of childbearing age. Estimates based on antenatal samples are both widely available and ideal for this purpose.

A second source of bias anticipated in the orphanhood-based estimates of adult mortality is that the estimation method is based on models with different age patterns of mortality from those in populations with high AIDS mortality. Modelling based on the data from Masaka district suggests that, despite this, the existing regression equations continue to perform fairly well for data on respondents aged 15 years and over. For younger respondents, in particular, the prevalence of orphanhood increases more slowly as overall mortality rises in populations affected by AIDS than in those that are not. The Masaka district data suggest, however, that the relationship between the proportion of mothers alive and life table survivorship from 25 years remains linear. On this basis, a provisional set of revised estimation coefficients are proposed for use in populations with significant AIDS mortality. The coefficients are provisional because ideally they would be based on data from a wide range of populations with different age patterns of HIV infection. Unfortunately, few such data yet exist for the developing world. While a single set of coefficients should be adequate for use in a wide range of populations, it is unlikely that the AIDS epidemic in Masaka district is in all respects typical. Therefore, a more satisfactory revision of the coefficients will become possible as our understanding of the parameters of AIDS epidemics in developing countries improves.

Estimates of adult women's mortality made from the data on orphanhood collected for children in Masaka district are very consistent with those calculated directly from the prospective data. Orphanhood data not only indicate the approximate level of mortality in populations affected by AIDS but can document the upward trend in adult mortality over time. Equally, the orphanhood-based results confirm the high quality of the direct information on adult deaths collected by the longitudinal study in Masaka district and the value of the information on the mortality effect of the HIV epidemic that is emerging from this study.

Information on orphanhood has not been gathered from the study population for those aged 15 years and more. However, both the proposed adjustment for selection bias and the new regression coefficients are less robust for the data on 5-9 and 10-14-year-old respondents than for those on older age groups. As the quality of orphanhood data on children is also more suspect than that of those supplied by older individuals, it seems reasonable to infer that the latter data will continue to represent a useful source of estimates of adult mortality. At present, almost no populations exist in which many mothers of adult respondents were infected when they gave birth to the respondent. Selection bias is not yet a problem in

older age groups and mortality would be overestimated if one adjusted the reported proportions of mothers alive downward unnecessarily. Equally though, this means that extensions of the orphanhood method that allow recent mortality to be measured using data supplied by adult respondents (Timæus 1991b,c) should continue to provide representative estimates of the mortality of the middle-aged until at least early next century.

The extent of the biases in estimates of adult male mortality obtained from data on the survival of fathers has not been investigated in this paper. Selection biases linked to vertical transmission will be less severe for data on fathers than those on mothers as not all couples are both infected. However, it is possible that serious bias in the regression coefficients for male mortality may not be restricted to those for respondents aged less than 15 years. Men tend to be older than women both when they become fathers and when they contract HIV infection. They also tend to do both over a wider range of ages. Nevertheless, as the basic structure of the determinants of the prevalence of orphanhood is the same for men and women, it seems likely that data on the survival of fathers will also continue to be of use for the estimation of mortality.

The orphanhood method remains a valuable way of measuring adult mortality even in populations with a relatively high prevalence of HIV infection that are undergoing rapid increases in adult mortality. Analysed appropriately, orphanhood data yield estimates that are less subject to bias in populations affected by HIV than those from most other methods of measuring adult mortality that can be used in national enquiries. Countries that lack effective death registration systems should ask questions about the survival of mothers in the census and national household surveys to monitor the effect of the AIDS epidemic on mortality. Demographers and international advisers should promote the orphanhood method with renewed vigour.

References

Bennett, N. G. and S. Horiuchi. 1981. Estimating the completeness of death registration in a closed population. *Population Index* 47:202-221.

Boerma, T., J. Ngalula, R. Isingo, M. Urassa, K. P. Senkoro, R. Gabone and E. N. Mkumbo. 1997. Levels and causes of adult mortality in rural Tanzania with special reference to HIV/AIDS. This volume.

Brass, W. 1971. On the scale of mortality. Pp.69-110 in *Biological Aspects of Demography*, ed. W. Brass. London: Taylor and Francis.

Brass, W. and E. Bamgboye. 1981. *The Time Location of Reports of Survivorship: Estimates for Maternal and Paternal Orphanhood and the Ever-Widowed.* Centre for Population Studies Working Paper 81-1. London: London School of Hygiene and Tropical Medicine.

Brass, W. and K. Hill. 1973. Estimating adult mortality from orphanhood. Pp. 111-123 in *International Population Conference, Liège, 1973*, Volume 3. Liège: International Union for the Scientific Study of Population.

Carpenter, L. M., J. S. Nakiyingi, A. Ruberantwari, S. S. Malamba, A. Kamali and J. A. G. Whitworth. 1997. Estimates of the impact of HIV infection on fertility in a rural Ugandan population cohort. This volume.

Cleland, J. 1996. Demographic data collection in less developed countries 1946-1996. *Population Studies* 50:433-450.

Gregson, S. 1994. Will HIV become a major determinant of fertility in sub-Saharan Africa? *Journal of Development Studies* 30:650-679.

Gregson, S., G. P. Garnett and R. M. Anderson. 1994. Assessing the potential impact of the HIV-1 epidemic on orphanhood and the demographic structure of populations in sub-Saharan Africa. *Population Studies* 48:435-448.

Kamali, A., J. A. Seeley, A. J. Nunn, J.F. Kengeya-Kayondo, A. Ruberantwari and D.W. Mulder. 1996. The orphan problem: experience of a sub-Saharan Africa rural population in the AIDS epidemic. *AIDS Care* 8:509-515.

Kengeya-Kayondo, J.F., A. Kamali, A. J. Nunn, A. Ruberantwari, H.-U. Wagner and D. W. Mulder. 1996. Incidence of HIV-1 infection in adults and socio-demographic characteristics of seroconvertors in a rural population in Uganda: 1990-1994. *International Journal of Epidemiology* 25:1077-1082.

Lepage, P., P. van de Perre, P. Msellati, et al. 1993. Mother-to-child transmission of HIV-1 and its determinants: a cohort study in Kigali, Rwanda. *American Journal of Epidemiology* 137:589-599.

Mulder, D. W., A. J. Nunn, A. Kamali, J. Nakiyingi, H. Wagner and J.F. Kengeya-Kayondo. 1994. Two year HIV-1 associated mortality in a Ugandan rural population. *Lancet* 343:1021-1023.

Mulder, D. W., A. J. Nunn, A. Kamali and J. F. Kengeya-Kayondo. 1995. Decreasing HIV-1 seroprevalence in young men in a rural Ugandan cohort. *British Medical Journal* 311:833-836.

Newell, M. Forthcoming. HIV infection in women and children. In *Brass Tacks*, ed. A. Basu and B. Zaba.

Nunn, A. J., B. Biryahwaho, R. G. Downing, G. van der Growen, A. Ojwiya and D.W. Mulder. 1993. Algorithms for detecting antibodies to HIV-1: results from a rural Ugandan cohort. *AIDS* 7:1057-1061.

Nunn, A. J., B. Biryahwaho, R. G. Downing, A. Ojwiya and D.W. Mulder. 1994. Computer-assisted quality assurance in a HIV serology laboratory. *Methods of Information in Medicine* 33:170-173.

Nunn, A. J., D. W. Mulder, A. Kamali, A. Ruberantwali, J. F. Kengeya-Kayondo and J. Whitworth. 1997. Five-year HIV-1 associated mortality in a rural Ugandan cohort. *British Medical Journal*. In press.

Paget, J. and I. M. Timæus. 1994. A relational Gompertz model of male fertility: development and assessment. *Population Studies* 48:333-340.

Palloni, A. and Y. J. Lee. 1992. Some aspects of the social context of HIV and its effects on women, children and families. *Population Bulletin of the United Nations* 33:64-87.

Palloni, A., M. Massagli and J. Marcotte. 1984. Estimating adult mortality with maternal orphanhood data: analysis of the sensitivity of the techniques. *Population Studies* 39:387-405.

Prebble, E. A. 1990. Impact of HIV/AIDS on African children. *Social Science and Medicine* 31:671-680.

Preston, S. H. 1984. Use of direct and indirect techniques for estimating the completeness of death registration systems. Pp. 66-76 in *Data Bases for Mortality Measurement*. New York: United Nations.

Rutenberg, N. and J. Sullivan. 1991. Direct and indirect estimates of maternal mortality from the sisterhood method. Pp. 1669-1696 in *Demographic and Health Surveys World Conference, 5-7 August 1991, Washington DC*.Columbia MD: IRD/Macro International.

Ryder, B., V. Batter, M. Nsuami, N. Badi, L. Mundele, B. Metela, M. Ntshudi and W. L. Heyward. 1991. Fertility rates in 238 HIV-1 seropositive women in Zaire followed for 3 years postpartum. *AIDS* 5:1521-1527.

Ryder, B., and M. Temmerman. 1991. The effect of HIV-1 infection during pregnancy and the perinatal period on maternal and child health in Africa. *AIDS* 5(supplement):S75-S85.

Seeley, J. A., H.-U. Wagner, J. Mulemwa, J.F. Kengeya-Kayondo and D.W. Mulder. 1991. The development of a community based HIV/AIDS counselling service in a rural area in Uganda. *AIDS Care* 3:207-217.

Serwadda, D. R.H. Gray, M. J. Wawer, et al. 1997. HIV, STDs and fertility: a population-based study in Rakai district, Uganda.

Sewankambo, N., M. Wawer, R. Gray, D. Serwadda, C. Li, R. Y. Stallings, S. D. Musgrave and J. Konde-Lule. 1994. Demographic impact of HIV infection in rural Rakai District, Uganda: results of a population-based cohort study. *AIDS* 8:1707-1713.

Timaeus, I. M. 1991a. Measurement of adult mortality in less developed countries: a comparative review. *Population Index* 57:552-568.

Timaeus, I. M. 1991b. Estimation of mortality from orphanhood in adulthood. *Demography* 28:213-227.

Timaeus, I. M. 1991c. Estimation of adult mortality from orphanhood before and since marriage. *Population Studies* 45:455-472.

Timaeus, I. M. 1992. Estimation of adult mortality from paternal orphanhood: a reassessment and a new approach. *Population Bulletin of the United Nations* 33:47-63.

Timaeus, I. M. 1993. Adult mortality. Pp.218-255 in *Demographic Change in Sub-Saharan Africa*, ed. K. A. Foote, K. Hill and L. G. Martin. Washington DC: National Academy Press.

Timaeus, I. M. 1997. Age patterns of mortality in sub-Saharan Africa: an initial study of the impact of AIDS. Paper presented to the annual meeting of the Population Association of America, Washington DC, 27-29 March.

Timaeus, I. M., B. Zaba and M. Ali. Forthcoming. Estimation of adult mortality from data on adult siblings. In *Brass Tacks*, ed. A. Basu and B. Zaba.

Tollman, S. M., K. Herbst and M. Garenne. 1995. *The Agincourt Demographic and Health Study*. Johannesburg: Health Systems Development Unit, University of Witswatersrand.

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HIV prevalence and lifetime risk of dying of AIDS*

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Abstract

The paper examines the relationship between the lifetime risk of dying from AIDS and HIV prevalence, using a female stable population model in which the epidemic has stabilized. In addition to prevalence, lifetime risk is determined by various other factors, notably the level of mortality from causes other than AIDS, age at infection, and survival time between infection and death. Typically, the lifetime risk of dying from AIDS is between three and five times the HIV prevalence. Regression equations are developed for estimating lifetime risk from the prevalence and other parameters. The methods are applied to data for Kenya, and it is shown that the 1995 prevalence estimate of 7.5 per cent for the population aged 15 and over would be equivalent to a lifetime risk of about 30 per cent.

HIV prevalence, or the proportion of the adult population which is HIV-positive, is the most commonly used index for measuring the scale of the epidemic. In African countries, national estimates of HIV prevalence are generally only available for females, as the main source of data is the anonymous testing of women who use antenatal services. More direct measures of the effect of HIV could be found from the annual death rate due to AIDS in the adult population, or the proportion of all adult deaths which are due to AIDS. These measures are rarely available in practice, as most African countries do not have death registration systems, and in any case, AIDS deaths are frequently perceived as due to other causes (Boerma et al. 1997). None of the above measures tells us about the overall risk of contracting HIV or dying from AIDS faced by an individual approaching adulthood and sexual maturity. But this last measure, which for a disease like AIDS is three or four times as large as the current prevalence measure, is one which would be very useful to planners and health educators alike. It would enable those who train skilled workers and professionals to judge what proportion of their cadres would be lost to this disease before the end of their working lives. It would allow insurance companies to set realistic premiums in countries affected by the epidemic. And it could provide a powerful advocacy tool for those trying to persuade young people not to follow the norm in terms of widely prevalent unsafe sexual behaviour.

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This paper investigates the relationship between HIV prevalence and lifetime risk, using models of age-specific incidence which have been tailored to reflect the type of sexual union formation patterns observed amongst females in a wide range of African populations. We focus on females because information on HIV prevalence, participation in sexual unions and fertility is widely available for females but not for males. The relationships which we discover

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in our model populations enable us to estimate lifetime risk statistics in populations which do not have the data required to calculate such risks directly.

Although HIV prevalence rates are usually quoted for 'the adult population' there is no generally accepted definition of 'adult', and different countries and different modellers adopt a variety of age ranges in their calculations. In Kenya, the National AIDS Control Programme defined prevalence relative to the population aged 15 and over, and the estimated figure for 1995 was 7.5 per cent, or about one person in 13. Confronted with this statistic, an uninformed reader might be tempted to infer that, if one person in 13 has the disease, the lifetime risk, or the proportion who could expect to contract it during the course of their adult lives, would also be about one in 13. In reality however the lifetime risk is very much higher than the prevalence, since the denominator for the prevalence rate consists of those currently alive, rather than all those who were ever at risk. On the other hand, detailed surveillance studies of small populations affected by HIV (e.g. Boerma et al. 1997; Nunn et al. 1997) have shown that at prevalence levels of between five and seven per cent, over 45 per cent of all adult deaths are due to AIDS. But it would be equally incorrect to assume that the lifetime risk of dying from AIDS for an adult in these populations was as high as 45 per cent, since the distribution of deaths by cause is affected by the age structure of the population, and in a growing population, younger adult ages, in which AIDS deaths predominate, would carry more weight.

Epidemiologists use a variety of measures to gauge the severity of a disease in a population. The most commonly used are prevalence: the proportion of the population suffering from the disease at a given point in time; and incidence risk: the annual number of new cases observed divided by the number of healthy individuals[1]. The relationship between prevalence and incidence is determined by the disease duration, which can be equated to survival time for diseases such as HIV/AIDS, which are generally fatal. In stationary and stable populations, simple relationships exist between these measures and the lifetime risk of dying of the disease (Preston 1987).

In a stationary population with a constant size and structure, and in which the number of births each year is exactly equal to the total number of deaths, these relationships are:

$$\frac{prevalence}{1-prevalence} = incidence \times mean\ survival\ time \qquad \ldots \mathbf{1}$$

$$lifetime\ risk = \frac{prevalence}{mean\ survival\ time \times birth\ rate} \qquad \ldots \mathbf{2}$$

For a stable epidemic in a stable population, with constant fertility, mortality, and growth rate, and a constant proportionate age structure, these equations are modified to give the following approximate relationships:

$$\frac{prevalence}{1-prevalence} \approx incidence \times mean\ survival\ time \times \exp(-rd) \quad \ldots \mathbf{3}$$

[1] Throughout this paper, all the incidence measures used are 'occurrences/exposure'-type measures, as is the convention in epidemiology, rather than 'occurrences/total population'-type measures which are occasionally referred to as demographic incidence rates, e.g. Hoem 1978.

$$lifetime\ risk \approx \frac{prevalence \times \exp(r \cdot [a+d])}{mean\ survival\ time \times birth\ rate} \qquad \ldots \mathbf{4}$$

where r is the growth rate of the population, a is the mean age at infection in the population, d is the mean length of time since infection for those people suffering from the disease, so that (a+d) is the mean age of persons currently infected[2]. When prevalence is defined as relating to a specified age group, then the 'birth rate' becomes the number of persons reaching the lower bound of the age group annually, divided by the total number in the age group. In populations which are growing in an irregular fashion, due to past changes in vital rates, the values *r* in equations 3 and 4 would be replaced by integral expressions of the type $r = \int_a^w r(x,t)dx$ appropriate for the time in question.

Data on all these variables are rarely available, but certain relationships are clear. Thus lifetime risk is higher when prevalence is higher; survival time is lower (this would also tend to shorten the mean time since infection); age at incidence is higher; mortality from other causes is lower (since growth rate will be higher). A low fertility rate might increase lifetime risk, as it would lower the crude birth rate in the denominator; but it would also reduce the growth rate in the numerator, so it could act in either direction.

Preston's equations allow us to appreciate the possible scale of the difference between prevalence and lifetime risk. Table 1 shows some hypothetical data conforming to the relationships shown in equation 4, for two different types of fatal disease. Both are assumed to have a prevalence of two per cent, a survival time of eight years, and a mean duration since infection of five years. But the cancer-type disease, which is shown occurring in a slow-growing population (growth rate 1%) has an older pattern of infection (mean age of incidence 50) than the AIDS-type disease (mean age of incidence 25) which is shown in a population growing at three per cent per annum. If it reached this prevalence level in spite of its old age pattern, the cancer-type disease would carry a lifetime risk of almost 20 per cent, nearly ten times its prevalence rate. A disease like AIDS which strikes at earlier ages, at this level of prevalence would be associated with a lifetime risk over five times its prevalence.

[2] Equations 3 and 4 would be exact if infection was concentrated at a single age, and if the illness was of fixed duration; a better approximation could be derived by using a series expansion of exp(r[a+d]) in terms of the means and variances of a and d.

Table 1
Theoretical relationship between disease prevalence and lifetime risk of dying (hypothetical data)

Disease	prevalence	Growth rate	Age at infection	Duration since infection	Survival time	Birth rate	Lifetime risk	Risk prevalence
Cancer	0.02	0.01	50.00	5.00	8.00	0.02	0.196	9.8
AIDS	0.02	0.03	25.00	5.00	8.00	0.04	0.114	5.7

An empirical example of a large difference between prevalence and lifetime risk is furnished by the example of maternal mortality. In the Matlab study area of Bangladesh, the maternal mortality ratio for the period 1976-89 was 5.1 maternal deaths per 1000 live births. The average total fertility rate during this period was 5.3, so the lifetime risk was about 2.7 per cent[3]. The incidence of maternal mortality may be taken as the maternal mortality rate of 91 per 100,000 woman-years (Fauveau and Chakraborty 1994). Assuming the average survival time for a fatal pregnancy to be nine months or 0.75 years, and the mean time since start of such a pregnancy for a cross-section of women to be half this figure, then with a growth rate of 1.3 per cent per annum, the prevalence of maternal mortality works out at 69 per 100,000 woman-years. Thus the lifetime risk of maternal mortality is nearly 40 times the prevalence of fatal pregnancies in this population.

In order to get realistic values for the age pattern of infection and subsequent mortality, we have used a spreadsheet model originally developed by Zaba (1994) for measuring the demographic effect of AIDS. This model allows for both behavioural and biological factors to influence the way HIV builds up in a cohort, and shows the effect of HIV on the growth and structure of the population. We define prevalence as the proportion HIV-positive among the population aged 15 and over. Lifetime risk is calculated by finding the number of deaths from AIDS in a typical cohort using multiple-decrement life table techniques, rather than the theoretical relationships above. The multiple-decrement approach eliminates the necessity of calculating statistics such as mean time since infection. A full formal specification of the model can be found in Zaba (1994), but a list of variable parameters which govern its operation is shown in Table 2. This table also shows the default values of the parameters and selected statistics for the default population and the epidemic so generated, as well as the ranges of the values used for the simulation models described later in this paper. In all our simulations we generate stable epidemics in stable populations, that is, the results presented show the relationships between prevalence and lifetime risk in a mature epidemic in which prevalence has reached an equilibrium level, and where death rates have stabilized along with the new population age structure.

[3] Estimated using the relationship:

$$lifetime\ risk = 1 - (1 - maternal\ mortality\ ratio)^{total\ fertility\ rate}$$

Table 2
Input parameters and output characteristics of simulation model

Union dynamics			
Input parameters	Default	Range	Notes
Women ever entering union	100%		
Sexual initiation:			
start age	15	13 - 17	Coale & McNeil (1972) marriage
median age	20	18 - 22	model
Union breakdown rate per year	10%	0 - 20%	Independent of age or union duration
Peak annual union re-entry rate	70%	50 - 90%	Bell-shaped distribution
Peak age for union re-entry	30	27 - 33	
Postpartum abstinence	6 months	3m - 9m	Range of variation: 0 - 2 years
Terminal abstinence median age	50	40 - 60	Logistic curve
Coital frequency			
new partner	0.3	.15 - .45	Average declines linearly from
regular partner	0.06	.03 - .09	once every 3 days for new unions to once every 2 weeks for unions lasting five years or more
Output characteristics			(pre HIV)
In active union by age 50	60%		
Mean partners by age 50	2.6		
Proportion partners dead	5%		determined by prevailing mortality rates
Fecundity			
Input parameters			
Mean age at			
menarche	13	11 - 15	Wood & Weinstein (1988) model
menopause	45	40 - 50	
Primary sterility	2%		
Maximum secondary sterility	40%	20 - 60%	Logistic curve
Breastfeeding amenorrhoea	6 months		Shortened after infant death
Postpartum contraception	1.5 yrs	0.0 - 3.0	

Table 2 (cont.)
Input parameters and output characteristics of simulation model

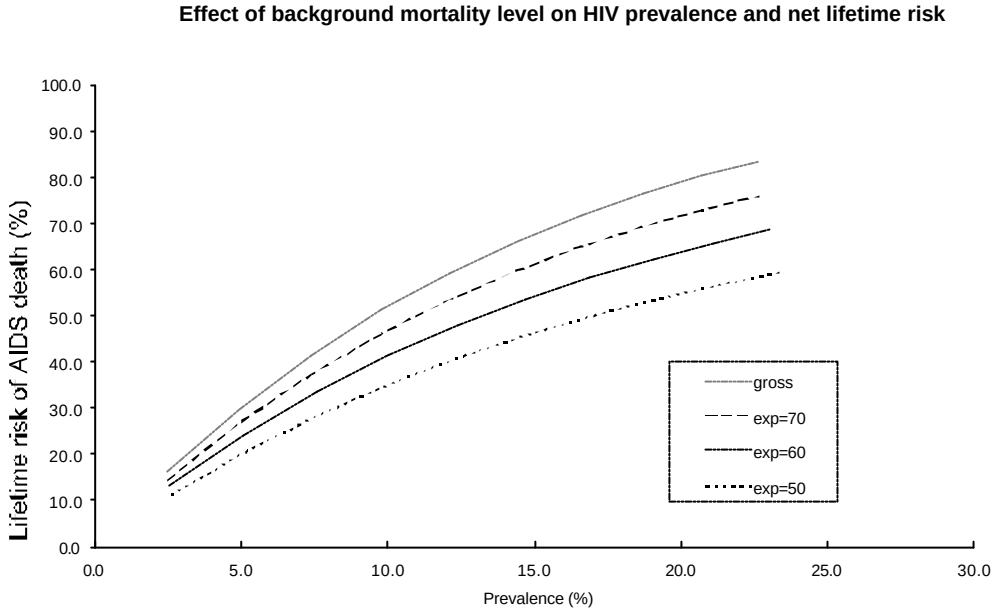
Output characteristics			(pre HIV)
Total fertility rate	5.00		jointly determined by fecundity and union dynamics parameters
Disease severity			
Input parameters			
Mean survival with HIV:			
adult	eight years	4 - 12	exponential decay curve
infant	1 year	0.5 - 1.5	all HIV+ve births die before age 10
Extent of vertical transmission	30%	20 - 40%	
Infection risk per coitus:			
new partner	0.0015	0 - .003	declines linearly over first five
regular partner	0.00015	0 - .0003	years of new partnership; range used in simulations 0.0 to 0.0003 for regular partners, new partner risk is ten times higher
Life expectancy of HIV-negative	60 years		Brass logit relational model
Output characteristics			(post HIV)
Stable prevalence at ages 15-50	17%		
HIV-positive births	6%		
Population life expectancy	39 years		
Mean age of infection schedule	32 years		

Mortality from causes other than AIDS

When discussing lifetime risks, it is necessary to distinguish between gross and net risks. A gross risk is the probability of a person contracting HIV, assuming that no one in the population dies from any other cause. A net risk is the proportion who will die of AIDS when allowance has been made for mortality from other causes. The difference between the gross and the net risks will of course depend on the level of mortality from causes other than AIDS; clearly the higher the mortality from other causes, the more likely will people be to die of something else first.

Since background mortality is a variable which can be manipulated in our model, we have calculated the net lifetime risks assuming that mortality from causes other than AIDS can be represented by model life tables with expectations of life at birth of 50, 60 and 70 years. The other variables were held constant at their default values shown in Table 2. The results are shown in Figure 1. The gross risk increases non-linearly with prevalence, and is over four times as high. The net risks are substantially smaller than the gross, but even with the highest level of background mortality, net lifetime risk is more than twice the prevalence. The lifetime risks shown in the next two figures are all net risks assuming an expectation of life of 60 years for causes other than AIDS.

Figure 1

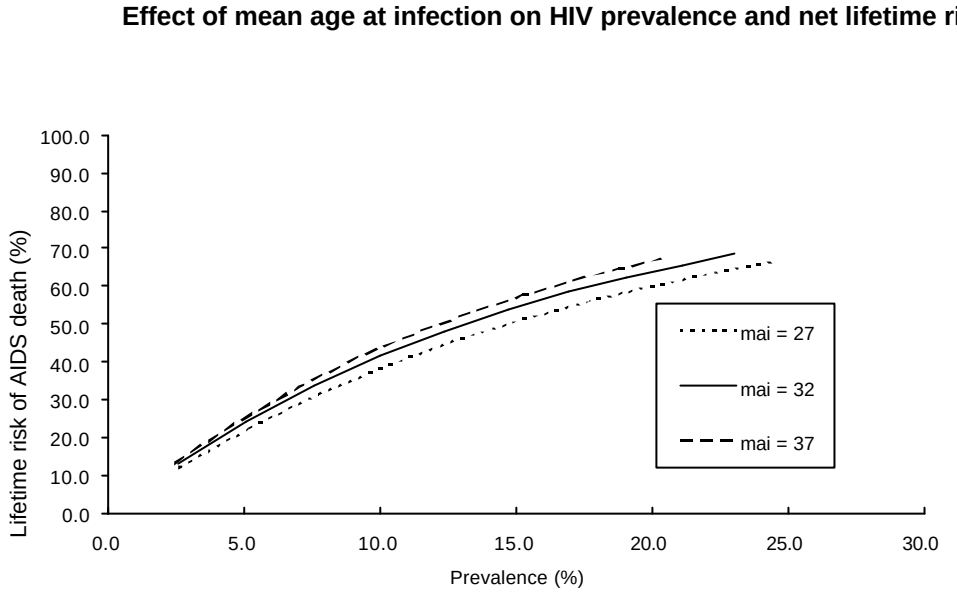


Age at infection

The mean age at infection is primarily determined by the ages at which people become sexually active. It thus varies from population to population, and between males and females, being generally younger among the latter. It is also affected by the age structure of the population, being younger when there is a steep slope on the age distribution. However this last effect can be eliminated from comparisons of populations if we make the calculations, not for the living population at a point in time, but for the schedule of incidence risks by age. The latter will normally be some four or five years older than the former.

Models with mean ages of the risk schedule of 27, 32 and 37 years were constructed. Survival time was held constant at eight years, and total fertility at five births per woman. The results are shown in Figure 2. The effects of the age at infection on lifetime risks are moderate but systematic. The higher the age at infection, the higher the lifetime risk implied by a given prevalence. This is because when age at infection is higher, the infection is concentrated in a smaller proportion of the population, so the same prevalence for the 15+ age range could only be achieved as the result of a higher overall lifetime risk.

Figure 2

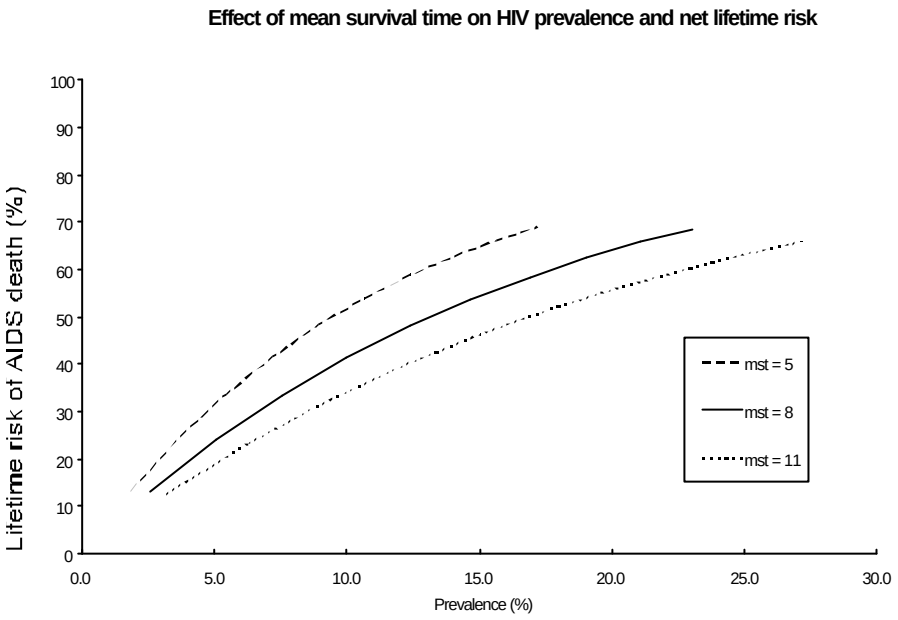


Survival time after infection

Mean survival times of the HIV-positive are difficult to estimate, since this involves very long prospective studies, but quite a lot of evidence is now available about median survival times. Cohort studies of white males in the US have suggested median survival times of ten years (Chin 1995), and drug therapies are now lengthening this time, but there is reason to suppose that in Africa it may be substantially less (Anderson 1989; Gregson 1994). Nunn et al. (1997) estimated median survival time after infection in Masaka district in Uganda as just over five years for persons infected when they were under age 55, and less than three years for those infected at later ages. In a simple exponential model of survival of the HIV-positive, such as used in our model, a mean survival time of eight years is equivalent to a median survival time of 5.5 years.

In order to explore the effect of survival time on the relationship between lifetime risk and prevalence, we have used three values of the mean duration: 5, 8 and 11 years. Mean age of the infection risk schedule was held constant at 32 years. The results are shown in Figure 3. Again the relationship is strongly affected by the survival time: for a given level of prevalence within our range, the lifetime risks were between 30 and 65 per cent higher when the mean duration was five years as against 11 years.

Figure 3

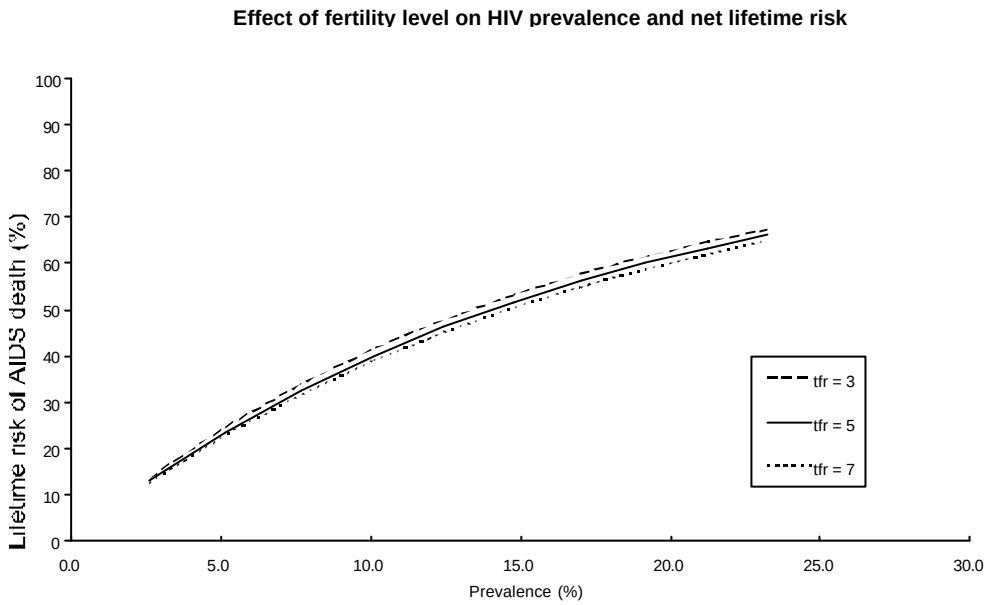


Fertility

As is evident from equation (4), fertility can affect the relationship between prevalence and lifetime risk in different ways. This is because fertility is the primary determinant of the shape of the age distribution: lowering fertility reduces the proportion of young people and increases the proportion of the old. With the mean age at infection held constant at 32 and the mean survival time at eight years, three sets of models were generated with total fertility rates of seven, five and three births per woman. The results are illustrated in Figure 4.

It appears that variation in the level of fertility, and hence in the age structure, has very little effect on the lifetime risk. This means that the relationship in populations with changing fertility levels will be similar to those observed in stable populations with an intermediate level of fertility. The explanation lies in the compensating effects of changes in the proportions of young adults under 25 and of old persons over 50; as one goes up the other goes down, but as both sections have relatively low prevalence of HIV the changes cancel out. If prevalence had been defined as the percentage HIV-positive among persons aged 15-49, a stronger relationship with fertility might have been revealed.

Figure 4



Child and adult risks

The lifetime risks shown in Tables 1 to 4 incorporate both the risks of horizontal transmission between adults and those of vertical transmission from mothers to children. The balance between adult and child risks depends on the likelihood of vertical transmission. In this model we have assumed a 30 per cent probability of an HIV-positive pregnant woman transmitting the virus to her unborn child, or during delivery or through breastfeeding, and we assume that all HIV-positive infants die before reaching adulthood. At this level, the risk of dying from HIV infection in childhood is between seven and ten per cent of the total risk, and the proportion increases slightly with the prevalence. If, however, we assumed a higher or lower vertical transmission ratio (VTR), the proportion of child deaths would increase or decrease correspondingly: with a VTR of 20 per cent childhood deaths constitute 4-6 per cent of total AIDS deaths; whereas if VTR was as high as 40 per cent, between 9 and 13 per cent of AIDS deaths would occur in childhood.

Thus the vertical transmission ratio is an important variable in determining the risk of infection in infancy and childhood, though not in adulthood. As such it is also a component of lifetime risk, albeit a relatively small one. It has virtually no effect on prevalence, which is based on the adult population only.

Applications to developing countries

The question now arises whether these models can be used to make realistic calculations of lifetime risk for Third World countries. Various problems arise in this respect.

In the first place information on at least two of the crucial variables, mean age at infection and survival time, is invariably lacking. We have attempted to overcome this problem by using proxy variables which we expect to be closely related to survival time and mean age at infection. We have investigated two such proxies which can be calculated from the models and which might also be available in real populations: the mean age at death of persons dying of AIDS, and, for females, the mean age at the birth of the first child. Sometimes information is available on the age distribution of AIDS cases rather than on deaths from AIDS, in which case the age at death can be approximated by adding half a year to the mean age of the AIDS cases, since the average survival time after the development of full-blown AIDS is only about one year; for example Boerma et al. (1997) report a mean duration of illness of ten months before an AIDS death.

In Africa AIDS cases and deaths from AIDS tend to be seriously underreported. But unless the degree of underreporting is associated with age, the mean age of the reported cases should give a reasonable approximation to that of all AIDS deaths. However bias could clearly arise if the degree of underreporting is greater in rural than in urban areas, and if the mean age at infection also differs between rural and urban areas.

The mean age of mothers at the birth of their first child should be associated with the mean age of HIV infection since both are the result of sexual activity. We would expect this relationship to be much closer in non-contracepting populations, where both pregnancy and infection are risks associated with sexual activity. But even in non-contracepting populations we would not necessarily expect the relationship to be fixed; if some cultures allow young girls to become sexually active before they become fully fecund, the risk of infection will be raised compared to that of pregnancy. And if sexual activity takes place with more than one partner, this will raise the risk of infection but not of pregnancy. Furthermore a high prevalence of primary infertility and high incidence of abortion would also raise infection risks relative to fertility risks in the population.

The mean age at first birth can readily be obtained from census or survey data on the distribution of women by parity and age group. Thus the mean age at first birth can be calculated from the proportions of childless women in each age group in the same way as the singulate mean age at marriage is obtained from the proportions never-married by age (Hajnal 1953). If there are appreciable numbers of women whose parity was not stated, adjustments by El Badry's (1961) method may be tried.

Information on median survival times may sometimes be available for African populations from small-scale longtitudinal studies, from which an informed guess can be made for the country concerned. On the assumption that the deaths are distributed in an exponential decay function, the mean values implied by the medians can be calculated. We have also explored the possibility of estimating survival times given both the mean age at first birth and mean age at death from AIDS, and have derived the necessary regression equations, using both ordinary least squares regression and a two-stage (instrumental variables) procedure, in which the estimate of mean survival time was not made explicit, but was indirectly fed into the regression estimate of lifetime risk. The results, however, were disappointing. The regression for mean survival based on the mean age at first birth, mean age at death from AIDS and prevalence explained only 67 per cent of its variance. The standard errors of the estimates of mean survival time were disturbingly large, giving 95 per cent confidence limits of the order of ±2 years when we are trying to estimate survival times of the order of eight years. When applied to the Kenya data, described below, they gave a figure of less than three years, which was unacceptably short. We have therefore been reluctantly forced to the conclusion that an independent estimate of survival time is an essential piece of information for our purposes, for which there is no readily available substitute.

A second problem with the practical applications is that the models are of stable populations with constant fertility, mortality and age structure, and in which the HIV/AIDS epidemic has also stabilized, so that prevalence too is constant. In practice we are dealing with situations where the epidemic is still growing and where both fertility and mortality from causes other than AIDS may be changing. In some countries badly affected by the AIDS epidemic, such as Zimbabwe and Kenya, fertility has been falling rapidly in recent years. Current growth rates will therefore differ from the stable population growth rates in our models, and even if recent growth rates can be estimated from successive censuses these will not persist, and would not be suitable inputs into the estimating procedure. These differences between the models and actual populations will affect our calculations in two ways: the underlying age structures will differ, and the mean age at infection will change as the disease spreads from age group to age group.

Changes in age structure will affect the prevalence, but not the lifetime risk, since the latter, like the expectation of life and the total fertility rate, is an index which is independent of the age structure. We have investigated the effect of a changing age structure on prevalence using the model fitted to the Kenya data described below. The age-specific prevalences from the female stable population model were combined with the projected female population of Kenya for 1995 (CBS 1996 b: appendix III); the overall prevalence for the population aged 15 and over worked out at 7.8 per cent as against 7.5 per cent in the model, which implied that we had overestimated lifetime risk by about one percentage point. Such an error can be regarded as trivial compared with the other margins of uncertainty surrounding our estimates.

If the mean age at infection rises as the epidemic spreads, the mean age at death from AIDS will also rise. But, because of the long period of incubation, the current mean age at death will imply an age at infection appropriate for a time several years before (which may partly explain why our regression estimates of survival time for Kenya were unacceptably short). Furthermore the mean age at death will also be affected by changes in the age structure of the population.

To assess the possible nature and magnitude of these changes, we have calculated, first, the mean age of the age-specific AIDS prevalence rates of the model projections made by Anderson (1989: Annex Table 14), and, second, the mean age of the AIDS cases obtained by multiplying their prevalence rates by their projected populations. Thus the first would measure the epidemiological changes only: the way the epidemic spreads from age group to age group; the second will measure the combined effects of the epidemiological and age structure changes.

The mean age of the AIDS prevalences rose by about two years over the 20-year period of the projections. In contrast the mean age of the AIDS cases rose by only 0.3 years over the same period. This implied that the epidemiological and age structure changes were having compensating effects. Their net result would be to introduce a small downward bias in the estimates of lifetime risk, thus compensating for the bias in the prevalences. We may conclude therefore that our stable population models can be used to estimate lifetime risk in current populations, provided they are not regarded as anything more than 'ball-park' estimates.

Linearization of the relationships and regression estimates

In order to make our model useful for estimating lifetime risk from commonly available data, we investigated the relationship between lifetime risk and our chosen indicators in a variety of circumstances. We generated 1000 model simulations with a wide range of values of HIV prevalence, non-AIDS mortality, mean age at infection, mean age at death from AIDS, mean age at first birth, survival time, vertical transmission ratio and fertility. The full model of HIV, population growth and structure is controlled by 22 different parameters describing biological and behavioural factors which govern fertility, mortality and HIV transmission. Even if each

parameter were only allocated one of two values, high and low, to produce simulations covering every possible combination of parameters would require 2^{22} = 4,194,304 different simulations. Since the amount of computer power needed to generate, store and analyse this would be excessive, an alternative strategy was adopted, whereby in each of the 1000 simulations each parameter was assigned a random value between its upper and lower limits.

The scatter diagram of the relationship between lifetime risk and prevalence in the female population derived from these models is shown in Figure 5. It will be seen that the trend is curvilinear, a feature which is already apparent in Figures 1 to 4. The curvature can however be eliminated if we plot the log-odds (i.e. the natural logarithms of the odds-ratios) of the lifetime risks against the corresponding log-odds of the prevalences, as shown in Figure 6. This linearization facilitates the use of multiple regression for analysing the effects of the different variables in the determination of lifetime risk. The independent variables, apart from prevalence, included mean age at first birth, total fertility, mean age at death from AIDS, mean survival time and the vertical transmission rate. An additional term of the product of the life expectancy of non-AIDS mortality and the log-odds of the prevalence was included, since the lines of the log-odds transformation of Figure 1, although straight, were not quite parallel.

The log-odds of the risks generated by the models were then regressed against the log-odds of the prevalences and the other variables. The regression analysis was carried out using ordinary least squares regression in the STATA (1997) package. Three separate regressions were made for each of the net risks considered as dependent variables: log odds of lifetime risk, log odds of risk for adults conditional on surviving to age ten, and the log odds of the net risk of dying from AIDS before age ten. The results are shown in Table 3. Apart from the independent variables listed in this table, we also experimented with the mean age of childbearing in the population, but in all cases this proved statistically insignificant, even at a ten per cent level, and was dropped from the final regressions. Unsurprisingly, the vertical transmission ratio proved to have an insignificant relationship with adult lifetime risk, and childhood risks were not significantly affected by the adult survival time after HIV infection, nor by the cross-product of life expectancy and prevalence. All the dependencies listed in Table 2 were highly significant at probability levels less than 0.1 per cent.

Figure 5

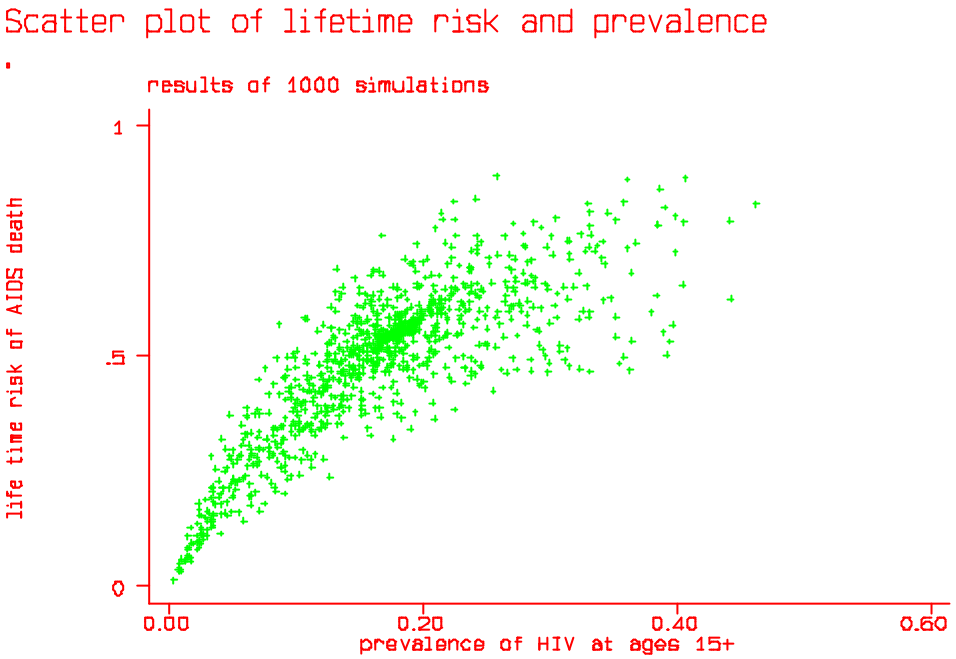
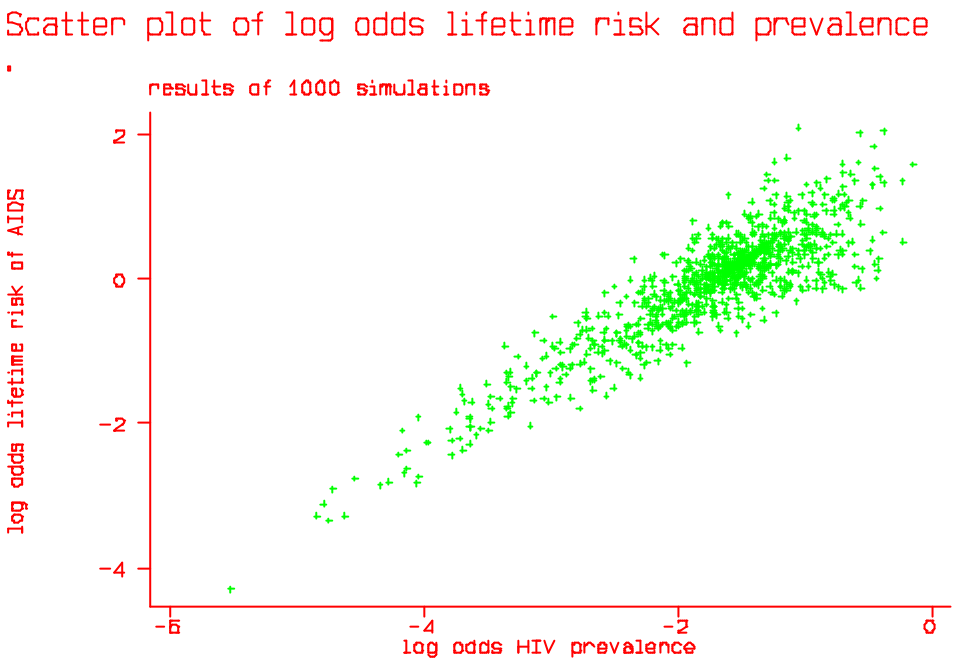


Table 3
Regression coefficients for the estimation of log-odds of net lifetime risk

	Risk at birth	Risk at age 10	Risk of dying from AIDS before age 10
Constant	-2.50	-1.43	-3.47
Log-odds prevalence	0.39	0.49	0.84
Life expectancy	0.06	0.05	0.01
Life exp.x log-odds prevalence	0.01	0.01	
Vertical transmission ratio	0.35		3.62
Mean age at first birth	0.03	0.03	0.07
Total fertility	0.03	0.02	0.06
Mean age at death from AIDS	0.03	0.03	-0.03
Survival time	-0.13	-0.15	
Regression statistics			
R-squared	0.99	0.99	0.99
Standard error	0.10	0.08	0.07

Figure 6

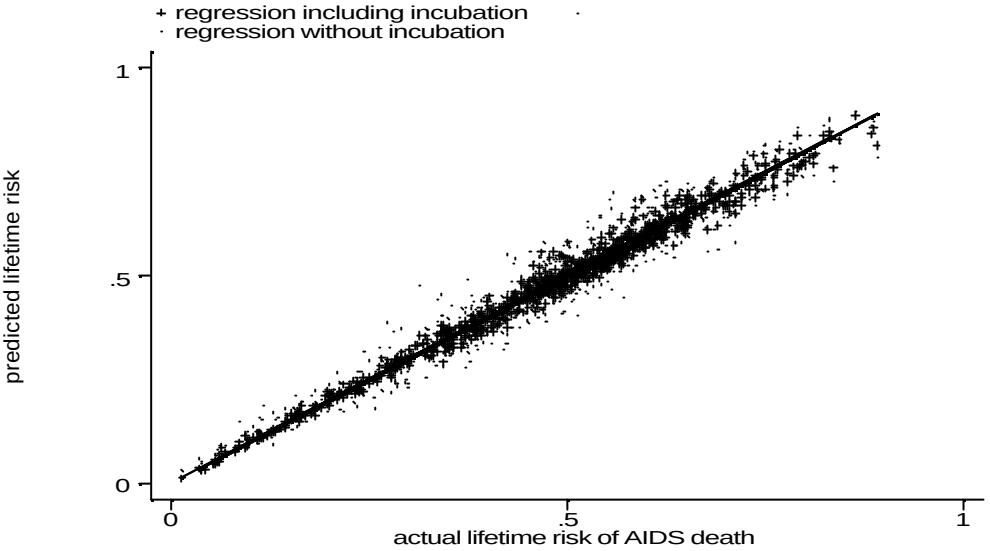


In the case of adult risk, and overall lifetime risk, the factors with the most influence were prevalence, its cross-product with life expectancy, life expectancy, survival time, and mean age at death from AIDS. Dropping the two factors relating to the age pattern of fertility would lower the amount of variance explained from around 99 per cent to 95 per cent, thereby raising the standard errors of the log odds estimates from around 0.09 to 0.17: this is equivalent to an error of 1-2 percentage points in the estimate of lifetime risk when this is of the order of 15 per cent.

Although survival time with HIV is one of the most important factors, both from theoretical considerations (equation 4) and as identified by the regression analysis above, we explored the effects of leaving it out of the regression procedure. The results showed that the amount of variance captured fell to around 90 per cent, and the standard errors of the log odds of the risks rose to 0.24, that is around four percentage points in estimating a risk of the order of 15 per cent. This we deemed unacceptable, which is why the results of regressions without survival time amongst the independent variables are not shown here. Figure 7 shows how well predicted values of lifetime risk based on regressions with and without survival time compare with the actual values generated in the model simulations.

Figure 7

Comparison of regression predictions and model output



A worked example: Kenya

As mentioned above, the National AIDS Control Programme of the Government of Kenya has estimated that the national HIV prevalence in 1995 was 7.5 per cent of the population aged 15 and over (NACP 1996). They have assumed a similar level of prevalence among males and females. They also estimate the vertical transmission ratio to be '30 to 40 per cent'; we have therefore adopted a value of 35 per cent. Figures of registered AIDS cases for the years 1988 to 1996 by sex and age group have also kindly been supplied by the NACP; from these we have estimated the mean age at death from AIDS for females to be 30.6 years.

The expectation of life for females in the absence of AIDS was taken as 61.4 years: this was the value obtained from the model life tables fitted to the data on child survival and orphanhood from the 1979 and 1989 censuses, and represented average values for the intercensal period, when mortality from AIDS could be regarded as negligible (CBS 1996a). Since mortality, both of children and adults, had been falling steadily before the 1989 census, it might seem reasonable to assume that mortality from causes other than AIDS would continue to fall into the 1990s. However this is a questionable assumption, and in the absence of hard evidence we have used the 1979-89 estimates as being the latest available. If, in so doing, we have overestimated non-AIDS mortality, we will have underestimated the lifetime risks of contracting HIV

The cohort mean age of women at the birth of their first child was calculated from the proportions of childless women in each age group (adjusted by the El Badry correction) obtained from the 1989 census, which gave a figure of 20.9 years. Similar calculations using the 1993 DHS data gave an identical result. Total fertility was taken to be 5.13, based on the the analysis of the 1989 census and 1993 DHS (CBS 1996b:13).

Our attempts to estimate mean survival time by regression gave unacceptable results. We know of no longitudinal studies in Kenya, but in Uganda and Tanzania such studies have suggested that seven years would be a plausible guess. The lack of information on survival times in Kenya constitutes the biggest gap in our knowledge for the purpose of these calculations.

When these parameters were multiplied by the regression coefficients shown in Table 3, this produced an estimate of overall lifetime risk of dying from AIDS of 31.3 per cent for a female born in Kenya subject to the current age-specific infection risks. Breaking down the risks separately for child and adult deaths, we find that the risk of dying from vertically transmitted AIDS is 3.6 per cent, and the risk of dying from AIDS for someone who survives to age ten is 32.8 per cent. The reason for the higher probability of dying from AIDS for those who survive to age ten than for the newborn is that infant and child mortality from other causes is also high in Kenya; in the absence of AIDS, we would expect about 12.4 per cent of female children to die before age ten in any case.

As an additional check on these regression results, we have constructed a single stable population model such that prevalence of HIV, non-AIDS mortality, mean age at first birth and at death from AIDS, total fertility rate, survival time and vertical transmission ratio were as close to the Kenya female values as possible. Table 4 shows the female stabilized age distribution, the age-specific prevalences, and the number of infected persons in each age group out of a total of 10,000; and the gross and net risks at different ages. The net lifetime risks at birth and at age ten from this best fitting model are about two percentage points lower than the regression-based estimates. But they confirm the general level of the lifetime risk estimate as around 30 per cent, some four times higher than the prevalence estimate.

Table 4
Age patterns of infection in the model for Kenya females

Age group	Age distribution	Prevalence (%)	Infected persons	Age	Net risk (%)	Gross risk (%)
0- 4	1565	1.50	23	0	29.20	39.40
5- 9	1338	0.00	0	5	29.90	36.80
10-14	1205	0.10	1	10	30.10	36.80
15-19	1077	4.40	47	15	30.30	36.20
20-24	912	12.80	117	20	29.00	33.00
25-29	741	14.40	107	25	24.10	27.00
30-34	604	11.70	71	30	18.10	20.20
35-39	501	8.70	44	35	12.80	14.10
40-44	422	6.70	28	40	8.50	9.30
45-49	357	4.70	17	45	5.10	5.50
50-54	305	2.50	8	50	2.60	2.80
55-59	261	1.20	3	55	1.20	1.40
60-64	220	0.60	1	60	0.60	0.70
65-69	179	0.30	1	65	0.30	0.30
70-74	138	0.10		70	0.10	0.20
75+	175	0.10	0	75	0.00	0.10
Total	10000	4.70	468			
Total 15+	5892	7.50	443			

Table 4 explains this feature which was noted at the beginning of this paper: why the lifetime risk is so much higher than the prevalence. The latter is calculated over the whole 15+

age range, while the infections are concentrated in a relatively short age span. Thus prevalence is low at the extremes of the distribution: in the 15-19 age group which contains many who have not yet been infected but may well be later, and at higher ages (45 and over) where the survivors escaped infection while many of their contemporaries perished and have therefore disappeared from the prevalence calculations.

We may conclude therefore that the estimated prevalence for 1995 of 7.5 per cent, or one person in 13, implies that, of the children born in Kenya, almost one in three will die of AIDS, leaving the other two to die of other causes. Although our calculations cannot do more than indicate a rough order of magnitude, it is nevertheless a formidable figure which we feel deserves wider recognition.

References

Anderson, R.M. 1989. Mathematical and statistical studies of the epidemiology of HIV. *AIDS* 3, 6:333-346.

Boerma, T.J., J. Ngalula, R. Isingo, M. Urassa, K.P. Senkoro, R. Gabone and E.N. Mkumbo. 1997. Levels and causes of adult mortality in rural Tanzania with special reference to HIV/AIDS. This volume.

Central Bureau of Statistics (CBS). 1996a. *Kenya Population Census 1989: Analytical Report.* Vol.3, *Population Dynamics of Kenya*. Nairobi.

Central Bureau of Statistics (CBS). 1996b. *Kenya Population Census 1989: Analytical Report,* Vol. 7, *Population Projections.* Nairobi.

Chin, J. 1995. Scenarios for the AIDS epidemic in Asia. Asia- Pacific Population Research Reports No.2. Honolulu: East-West Center.

El Badry, M.A. 1961. Failure of enumerators to make entries of zero: errors in recording childless cases in population censuses. *Journal of the American Statistical Association* 56,296:909-924.

Fauveau, V. and J. Chakraborty. 1994. Women's health and maternity care in Matlab. Pp. 109-138 in *Matlab: Women, Children and Health*, ed. V. Fauveau. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh.

Gregson, S. 1994. Will HIV become a major determinant of fertility in sub-Saharan Africa? *Journal of Development Studies* 30,3:650-679.

Hajnal, J.1953. Age at marriage and proportions marrying. *Population Studies* 7,2 111-136.

Hoem, J. 1978. Demographic incidence rates. *Theoretical Population Biology* 14:329-337.

National AIDS Control Programme (NACP). 1996. *AIDS in Kenya*. Nairobi: Ministry of Health.

Nunn, A.J., D.W. Mulder, A. Kamali, A. Ruberantwari, J.Kengeya-Kayondo and J. Whitworth. 1997. Five-year HIV-1 associated mortality in a rural Ugandan population. *British Medical Journal*, in press

Preston, S.H. 1987. Relations among standard epidemiologic measures in a population. *American Journal of Epidemiology* 126,2:336-345.

STATA. 1997. Statistical Software, Release 5. Stata Corporation.

Zaba, B. 1994. The demographic impact of AIDS: some stable population simulation results. Research Paper 94-2. London: Centre for Population Studies, London School of Hygiene and Tropical Medicine.

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Levels and causes of adult mortality in rural Tanzania with special reference to HIV/AIDS*

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Abstract

Data from a longitudinal study in northwest Tanzania were used to assess the levels of adult mortality and the leading causes of death. Adult mortality in this rural area was high and 42 per cent of persons aged 15 will die before their sixtieth birthday at current mortality rates. Mortality in this population with an HIV prevalence of about six per cent in 1994-95, has increased by about one-third because of HIV/AIDS, and further increase is likely. Other infectious diseases cause nearly a quarter of deaths and non-communicable diseases are still a relatively minor cause. The occurrence of the AIDS epidemic may have further delayed the onset of the epidemiological transition in many parts of Africa.

Adult health in developing countries has ranked low on the international and national agenda. In part this was due to preoccupation with high levels of child mortality, and child survival programs consumed the bulk of resources. Adults mainly benefited from efforts directed at specific disorders or conditions through programs such as those on safe motherhood or specific tropical diseases.

Demographic changes such as declining levels of child mortality and fertility have led to increasing importance of adult health (Feachem et al. 1992). Changes in disease epidemiology among adults are also taking place in many parts of the developing world and this calls for a major reorientation of health policy (Gribble and Preston 1993). Non-communicable diseases are becoming more important as a cause of adult morbidity and mortality.

In many parts of Africa demographic and epidemiological changes occur at a slower pace than in most other developing countries. A primary obstacle towards developing an effective adult health policy in sub-Saharan Africa is the extreme paucity of data on mortality levels, differentials and causes. Estimates of levels and trends, mostly based on census data,

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yield an incomplete and inaccurate picture of adult mortality in sub-Saharan Africa (Timaeus 1993). Data on causes of death among adults are limited to a few populations or hospitals.

The AIDS epidemic has contributed to the increasing interest in adult mortality; a handful of longitudinal studies in mainly eastern Africa have been initiated to assess the epidemic's causes and consequences. These efforts should lead to more and better data on adult mortality, including non-HIV-related causes of death. This study presents data from one of such efforts in northwestern Tanzania, where the HIV epidemic is now about a decade old. Data from two years of follow-up of a rural population of 20,000 are used to assess the level of mortality, causes of death, and health services utilization during terminal illnesses.

Data and methods

The study was conducted in Kisesa ward in Mwanza Region, Tanzania. The ward has a population of 20,000 and lies about 20 kilometres east of the regional capital Mwanza, along the main road to Kenya. Kisesa ward includes six villages, with about 40 per cent of its population located in a trading centre along the road. More than 90 per cent of the population are Sukuma, the largest ethnic group in Tanzania.

A demographic surveillance system forms the basis of all research activities in Kisesa. In 1994 a baseline census was carried out. All households in Kisesa ward were visited by field workers who listed all persons, and their sex, age (and birth date), schooling, and relationship to the head of the household. Follow-up visits were made every four months. At such visits data were collected on survival status, residence status, pregnancy of women of reproductive ages, and new arrivals. A new person was only listed as a household member if the household respondent had indicated that this person was intending to stay in the household. If the person had left the household by the next round he or she was not considered a resident. By mid-1996 six rounds, including the baseline census, had been completed.

If a death had occurred of a person under 60 years of age the household was visited by an assistant medical officer who interviewed relatives of the deceased or others who knew the deceased well. This interview included an open disease history and a structured questionnaire and was conducted in Swahili, the national language, or in Sukuma, the predominant local language. The questionnaire had been developed following anthropological and clinical research. A qualitative study was conducted in nearby fishing villages to describe local interpretation of illness (Washija and Pool 1996). Within Kisesa, focus-group discussions were held to find the most commonly used terms for signs and symptoms and causes of death . A detailed study of signs and symptoms in adults with HIV/AIDS compared to other illnesses had been completed in the regional referral hospital (Kalluvya et al. 1996). Clinicians of the local referral hospital and public health experts met to develop the questionnaire and algorithms for the most common causes of death.

A survey of all adults 15-44 years was conducted in Kisesa from August 1994 to July 1995. Three-quarters of all eligible adults participated in the survey which included a structured questionnaire and the collection of serum for HIV and syphilis testing (Kisesa Sero-Survey Team 1996). HIV testing was done using at least two ELISA tests, and in case of indeterminate results a Western Blot. Overall HIV prevalence among 5721 adults 15-44 in the ward was six per cent, ranging from four per cent in the five rural villages to ten per cent in the roadside village (Kisesa Sero-Survey Team 1996). Participation was poorer in the roadside village than in the rural villages. If HIV prevalence among non-participants were the same as among study participants from the same village, HIV prevalence would be 7.5 per cent in the ward.

Mortality rates were calculated using the number of deaths occurring between the baseline census and follow-up round 6, and person-years of follow-up. The calculations were

restricted to the resident population. This implied that a person had to be listed at least once as a resident. If a person moved into the Kisesa area after the last round of follow-up and died before the next round, he or she was not included in the analysis. With regard to the calculations of the person-years of observation, the time between the first and last follow-up round in which the person was listed as living in the household was taken for survivors. If a person had moved out for at least two rounds and later returned this person was considered a new resident. Mortality calculations are limited to deaths of those 15-59 years of age, in line with other studies of adult mortality in Africa (Murray, Yang and Qiao 1992; Timaeus 1993).

For the calculations of HIV-specific mortality rates those with indeterminate HIV status were excluded. The follow-up time was calculated from the date of the HIV test until the last round of observation, or in the case of a death, until the midpoint in the interval between two rounds in which the death occurred.

The data from the verbal autopsy questionnaire were analysed using a computer-based diagnosis based on predefined algorithms. For example, to make the diagnosis AIDS the following algorithm was used, based on clinical case definition from WHO (1994): two major symptoms such as at least one month of fever, diarrhoea, or weight loss; and one minor sign: persistent cough, generalized body itching, multiple body swelling as a sign of lymphadenopathy, history of herpes zoster, dysphagia or white spots in the mouth, neck-stiffness, more than five skin abscesses, recent tuberculosis. As for most deceased persons, the sero-survey was carried out after their death and HIV results were available for only 28 per cent of deaths. If HIV results were available (for most deaths the sero-survey was held after the death) these results were used to make the diagnosis. All deaths with positive HIV-antibody test were classified as HIV disease. The diagnosis AIDS was made if other symptoms occurred in addition to HIV infection, in line with the expanded case definition recommended by the World Health Organization (1994).

In addition to the algorithm-based diagnosis, two clinicians were given the disease history, as reported by the respondent, with the structured questionnaires, and made a diagnosis. The three diagnoses were put together and a final diagnosis was made if at least two of the three diagnoses were in agreement. If this was not the case, the cause of death was considered unknown. To simplify the analysis we opted for a single final diagnosis even though the clinicians used multiple causes. Only in the case of HIV/AIDS were multiple conditions recorded. In the analysis the causes were divided into communicable and reproductive causes, non-communicable causes, injuries and undetermined, following Murray et al. (1992).

Results

Mortality rates by age and sex

Table 1 presents the mortality rates for men and women 15-59 years. Overall mortality rates among men and women 15-59 years were 10.8 and 10.0 per 1000 person-years respectively. The age patterns were rather similar for both sexes. These rates can be used to estimate the probability that a man or woman aged 15 dies before his or her sixtieth birthday at current mortality rates ($_{45}Q_{15}$): 42 per cent of men and 43 per cent of women would have died before reaching the age of 60.

There were only minor differences in mortality rates 15-59 between the roadside villages and rural villages within Kisesa (9.8 and 10.6 per 1000 respectively).

Table 1
Mortality rates by age and sex, Kisesa, 1994-96.

Males			
Age	Deaths	PYO	Mortality rate
15-24	18	3156	5.7
25-34	25	2115	11.8
35-44	21	1302	16.1
45-54	14	910	15.4
55-59	3	305	9.8
All 15-59	81	7788	10.4
Females			
Age	Deaths	PYO	Mortality rate
15-24	11	2992	3.7
25-34	29	2350	12.4
35-44	16	1289	12.3
45-54	14	884	15.8
55-59	9	354	25.4
All 15-59	79	7869	10.0

Causes of death

Verbal autopsy interviews were conducted for 141 of 160 deaths (88%). The main reason for not obtaining information for all deaths was absence of the whole household or of key members of the household who were knowledgeable about events before the death. Other reasons included administrative errors (4) and refusal (2). The most common respondents were parents (29%), other relatives (18%), spouse (17%), sibling (16%), friend or neighbour (11%) or child (9%). Interviews were on average conducted 4.9 months after a death (range 2 weeks to 13 months). In addition, three deaths were included for which HIV status but no complete verbal autopsy was available. These deaths were coded as HIV disease (2), or unknown if the HIV test was negative (1).

Table 2
Causes of death among adults 15-59 years, by sex, Kisesa 1994-96

Causes of death	Men		Women		All	
	N	%	N	%	N	%
Communicable and reproductive	47	64.4	43	60.6	90	62.5
HIV/AIDS	22	30.1	28	39.4	50	34.7
Diarrhoea	7	9.6	2	2.8	9	6.3
Tuberculosis	4	5.5	2	2.8	6	4.2
Malaria	4	5.5	3	4.2	7	4.9
Other infections	10	13.7	3	4.2	13	9.0
Maternal causes	-		5	7.0	5	3.5
Non-communicable	9	12.3	10	14.1	19	13.2
Neoplasms	0	0	4	5.6	4	2.8
Endocrine	1	1.4	0	0	1	0.7
Cardiovascular	4	5.5	3	4.2	7	4.9
Respiratory	2	2.7	2	2.8	4	2.8

Digestive / urogenital	2	2.7	1	1.4	3	2.1
Injuries	6	9.2	7	9.9	13	9.0
Unintentional	4	5.5	3	4.2	7	4.9
Intentional	2	2.7	4	5.6	6	4.2
Undetermined	11	15.1	11	15.5	22	15.3
Total	73	100.0	71	100.0	144	100.0

Table 2 presents the probable causes of death for 144 deaths 15-59 years by sex. For 15 per cent of deaths no cause could be ascertained. Communicable and reproductive conditions accounted for 62.5 per cent of all deaths. HIV/AIDS mortality was associated with 35 per cent of all deaths, and was the leading cause for both sexes: 30 per cent of male and 39 per cent of female deaths. Among 50 deaths classified as HIV-associated, there were 30 deaths with clinical AIDS but no tuberculosis, 13 with HIV/AIDS and tuberculosis, and 7 deaths among HIV-positives with no clinical AIDS. These included deaths due to malaria, hepatitis, neoplasm (not cervix carcinoma or Kaposi's sarcoma), and four deaths with no other diagnosis than a positive HIV test. One death due to puerperal sepsis in an HIV-positive woman was classified as maternal death.

Other communicable disease were thought to be the cause of 24 per cent of all deaths. Tuberculosis was associated with 13.2 per cent of all deaths, and two-thirds of those deaths were in persons with HIV/AIDS. Six deaths (4.2%) were ascribed to tuberculosis without HIV infection. The most common diarrhoeal disease was dysentery which was the probable cause of 3.5 per cent of deaths. The most common 'other infections' were meningitis (3.5%) and severe anaemia (2.8 per cent). Strictly anaemia is not an infection, but in the tropics it is frequently the consequence of multiple infections, such as malaria and helminthic infections, and poor nutrition (excluding sickle cell anaemia). No deaths could be ascribed to pneumonia, except as part of clinical AIDS. All deaths with symptoms of respiratory infections of cough, and breathing difficulties, with or without fever, had either prolonged coughing (more than 4 weeks), symptoms of cardiac problems, or signs of tuberculosis, and were classified in the other categories.

Non-communicable diseases were responsible for a relatively small proportion of adult deaths (13%). The most important causes were cardiovascular diseases, neoplasm and liver problems (especially cirrhosis, 5 cases, i.e. 3.5% of all deaths). Thirteen deaths were associated with injuries (9%). Non-intentional injuries included three motor vehicle accidents. There were six homicide cases.

The distributions of causes of death were very similar for men and women. The main differences were more HIV/AIDS-associated mortality among women and more deaths from other infections among men. There were five maternal deaths, which corresponded with seven per cent of all adult female deaths. One of these deaths was due to an induced abortion. Mortality due to non-communicable causes and injuries was equally important for both sexes. Notable was the high number of deaths among women due to homicide (4), compared to two male deaths due to homicide. These women were all over 35 years and in at least three of these four cases alleged witchcraft practices of the deceased were the reason for the homicide.

Table 3
Mean age at death and mean duration of illness by cause of death and sex (standard deviation in parentheses), Kisesa 1994-96.

Cause of death	N	Age at death (years)	Duration of illness (months)	
HIV/AIDS	48	35.1 (8.6)	10.1	(8.6)
Other communicable	40	32.2 (11.6)	2.5	(4.6)

Non-communicable	19	41.8 (12.5)	9.0	(9.1)
Injuries	13	40.7 (13.4)	0.2	(0.3)
Undetermined	21	33.6 (12.6)	7.0	(8.1)
All	141	35.5 (11.4)	6.4	(8.0)

Table 3 shows the mean age at death (in years) for the five groups of causes for 141 deaths with complete verbal autopsy interviews. The overall mean age at death was 35.5 years and the differences between the causes of death were small. Deaths due to infections occurred at an earlier age than those due to non-communicable diseases (32.2 and 41.8 years respectively). The mean age of deaths due to HIV/AIDS was close to the overall mean. Analysis of age at death by sex and cause however showed a significant difference between men and women for HIV/AIDS: 38.7 and 32.6 years respectively (t=2.56 with 56 degrees of freedom; p =.014).

The mean duration of illness, as reported by the respondents, is also shown in Table 3. The mean duration of illness for all deaths was 6.4 months. Persons who died of HIV/AIDS had been sick for 10.1 months, followed by non-communicable diseases (9 months). The terminal illness for HIV-associated deaths lasted four times as long as for deaths due to other communicable and reproductive disorders.

Mortality by HIV status

Mortality rates by HIV status are presented in Table 4 for both sexes combined. There were 22 deaths among those with negative HIV test results, resulting in a mortality rate of 4.1 per thousand person-years among adults 15-44. There were also 22 deaths among HIV-positive persons, which corresponded with a mortality rate of 72.8 per thousand person-years. The age-adjusted mortality rate ratio was 14.5 (95% confidence interval 9.4-33.8). The population-attributable fraction, the proportion of deaths attributable to HIV infection among persons 15-44 years, is 47 per cent.

Table 4
Mortality rates per thousand person-years of observation (PYO) by age and HIV status, Kisesa, 1994-96

Age	HIV negative			HIV positive		
	Deaths	PYO	Mortality	Deaths	PYO	Mortality
15-24	10	2535	3.9	2	59	33.9
25-34	4	1764	2.3	13	160	81.3
35-44	8	1090	7.3	7	83	84.3
All	22	5389	4.1	22	302	72.8[a]

[a] Age adjusted rate ratio 14.5

A more detailed picture by age and sex can be obtained by using the verbal autopsy data and dividing the deaths into three groups: HIV-associated (based on HIV test and/or verbal autopsy), other conditions and unknown (Figure 1). The latter include deaths with no verbal autopsy. Among women HIV-associated mortality peaks at 25-34 years and exceeds mortality due to all other causes. Mortality due to other conditions increased gradually by age. The cause of death was unknown for a large proportion of female deaths over 45 years, making the picture in this age group unreliable. Among men, HIV-associated mortality increased by age and reached its highest level at 45-54 years. Mortality due to other

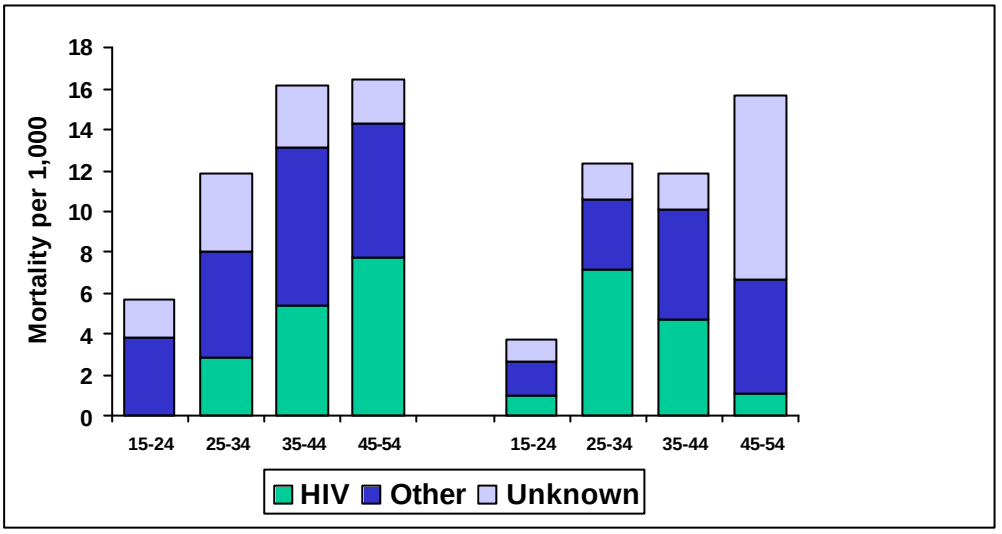
conditions and HIV-associated mortality each account for about half of the male deaths among those aged 35 years and above.

Health services utilization

During the period of terminal illness 40.4 per cent of the deceased had been admitted to a hospital. Among those who died of HIV/AIDS this proportion was 50 per cent, and this did not differ significantly from the proportion admitted among those who died of other communicable or non-communicable diseases (44.1%). There were no differences between men and women.

Utilization of traditional healers was high: 65.3 per cent of all persons who died had visited a traditional healer during the terminal illness and many had stayed for prolonged periods at the traditional healer's premises. Among those with HIV/AIDS deaths 75 per cent had used traditional healers, compared to 57.6 per cent among deaths due to other diseases. Women made more use of traditional healers than men (71.7% and 59.3% respectively for deaths due to diseases), particularly if the cause of death was HIV/AIDS: 85.7 per cent and 60 per cent visited traditional healers respectively. None of these differences however was significant at the 5 per cent level using Fisher's exact test for 2x2 tables.

Figure 1
Mortality rates per 1000 person-years by cause of death and age



During the open-ended section of the verbal autopsy interview many respondents spontaneously mentioned that the cause of death was witchcraft. This occurred more frequently for deaths due to HIV/AIDS than for other deaths (45.8% and 32.2% respectively), but this difference was not statistically significant.

Most deaths took place at home (62.4%), while 13.5 per cent of deaths took place in a health facility. This proportion was similar for deaths due to HIV/AIDS (12.5%). The remaining deaths occurred in another home (family member, traditional healer, 22.0%) or on the road (2.1%).

Discussion

In the Kisesa rural population mortality rates for those aged 15-59 years were 10.8 per thousand for men and 10 for women. This is considerably higher than the mortality estimate of 5.7 per thousand person-years 15-49 for Mwanza Region, in which Kisesa is located, derived from the 1988 national census data (World Bank 1992). Our estimates of ${}_{45}Q_{15}$ (42% for men and 43% for women) were also higher than estimates of 33 per cent for males and females derived from the 1988 census using information on recent death and orphanhood (Timaeus 1993). The Kisesa estimate for male mortality, however, is very close to the results of a follow-up study of more than 10,000 adults in 12 rural communities in Mwanza Region (11 per 1000 person-years for males 15-54), but higher than the female mortality in that study (7.6 per 1000) (Todd et al. 1997). A large study based on vital registration of deaths and annual censuses was carried out in three other locations in Tanzania during 1992-94 (Kitange et al. 1996). Adult mortality rates (15-59 years) were estimated at 8.1 and 6.1 per thousand person-years in Hai district in northern Tanzania, and 15.9 and 13.4 per thousand in rural Morogoro district in central Tanzania, for men and women respectively. In Dar es Salaam the corresponding mortality rates were 10.2 and 10.4 per thousand. Mortality data from longitudinal studies in Uganda were in the same range or higher than in Kisesa. In 15 rural villages in Masaka, southwest Uganda, mortality was 10.3 per thousand person-years among persons aged 13-54 years (Mulder et al. 1994). In a population in Rakai district, Uganda, with

HIV prevalence above 20 per cent, mortality was very high (31.9 per 1000 years of follow-up among persons 15-49) during 1990-91 (Sewankambo et al. 1994).

A problem compounding the measurement of mortality in longitudinal studies is the mobility of sick persons before death. This problem is likely to become larger because AIDS is a chronic illness and gives many sick persons sufficient time to choose a place of dying. In a study in Morogoro district, Tanzania, the homecoming sick constituted 11 per cent of all deaths, and in Hai district this proportion was even higher, 19 per cent (Kitange et al. 1996). In the Kisesa study six deaths were of people who had come home because they were sick. Another two deaths occurred among new arrivals who had been present for only one round and had died at the time of the next visit. The problem of homecoming sick in Kisesa is however not thought to affect mortality estimates or cause-of-death patterns because of the frequency of household visits (three per year) and because of the criteria used for inclusion in the study (at least one round resident and intending to stay). Another problem is the high mobility of HIV-infected persons, perhaps even before they fall sick. In Kisesa mobility was high: about ten per cent of the population moved to another household each year. Mobility among HIV-positive persons may also be higher than among HIV-negative adults. This will only cause a bias if more HIV-positives move out of the study area than into the area. Apart from the migration of the terminally ill out of Mwanza town there is no evidence of such a bias.

The high mortality among HIV-positive persons considerably increases adult mortality rates in African communities. In our study annual mortality rates among HIV-positives 15-44 years were 7.3 per cent compared to 9.3 per cent in rural Mwanza Region (15-44, Todd et al. 1997), 9.6 per cent in Masaka, Uganda (13-44 years, Mulder et al. 1994) and 10.6 per cent in Rakai, Uganda (15-49 years, Sewankambo et al. 1994). The proportion of deaths associated with HIV among persons 15-44 was 47 per cent in Kisesa, compared to 40 per cent in rural Mwanza Region (Todd et al. 1997), 85 per cent in Masaka, Uganda, where mortality among HIV-negatives was very low (Mulder et al. 1994), and 73 per cent in Rakai district, Uganda, where HIV prevalence was very high (Sewankambo et al. 1994).

Mortality rates among HIV-infected persons depend on the stage of the epidemic, as the median duration since infection in the population of infected individuals is shorter in the earlier years of the epidemic. The differences in annual HIV mortality rates between the studies may indicate that HIV-associated mortality in Kisesa may still increase further, not only because of large numbers of HIV-infected persons, but because of the relative dominance of recent infections among HIV-positives. HIV prevalence in rural Mwanza Region may have increased from about 2.5 per cent in 1990/91 to four per cent in 1992 (Grosskurth et al. 1995). Preliminary data from the repeat Kisesa sero-survey also seem to indicate that prevalence is still increasing. The number of seroconversions is higher than the number of HIV-associated deaths, as was also shown in other rural areas in Mwanza region (Todd et al. 1997). This suggests that the epidemic is still in its early stages, with HIV prevalence not yet at its peak and therefore mortality still many years removed from its peak. Simulations have indicated that mortality rates initially increase slowly, followed by a rapid increase 5-10 years into the epidemic, and stabilizing around 15-20 years after the onset of the epidemic in the general population (Gregson, Garnett and Anderson 1994).

The Kisesa cause-of-death structure can be compared with the predicted cause-of-death structures presented by Murray et al. (1992) for different levels of mortality. Even if HIV/AIDS mortality is excluded, the comparison suggests that mortality due to communicable diseases is higher than predicted in the models, particularly for diarrhoea, malaria and other infections. Non-communicable diseases in Kisesa were thus less important than in the models, even if we consider that the deaths from unknown causes may have predominantly been from non-communicable diseases. The hostile tropical environment may

contribute to the relatively high level of mortality from infectious diseases, which was also observed in other studies on causes of death in Africa (Timaeus 1993).

HIV-associated mortality was more common among women: 39 per cent of female and 30 per cent of male adult deaths. The sex difference becomes larger if the age at death is taken into account. Women died of HIV/AIDS at an earlier age than men and also than women dying of other causes. Thus, HIV-associated mortality was responsible for 47 per cent of the years of life lost between 15 and 60 years among women, while the corresponding proportion among men was 25 per cent.

Limitations and procedures for verbal autopsy among adults have been reviewed by Chandramohan et al. (1996). In our study considerable attention was paid to the development of an appropriate survey instrument, from both the anthropological and biomedical perspectives. The open-ended questions turned out to be very useful. The use of an experienced, medically-trained interviewer, knowledgeable about the local culture, was essential. During the course of the study more attention was paid to structuring and recording the medical and social history, as narrated by the respondent. This section now includes information on health services utilization, household social context, consequences of the death for the household, and expenditure during the illness.

In Masaka, Uganda, the diagnosis based on verbal autopsy was compared to HIV status for 155 deaths (Kamali et al. 1996). The results showed high specificity and positive predictive value of the verbal autopsy technique: 92 per cent. In our study such a comparison was not possible because HIV status was available for only 28 per cent of deaths. The verbal autopsy data may have led to an under- or overestimation of the proportion of deaths associated with HIV/AIDS. We have no way to assess the magnitude of such biases, but two potential biases can be described. For 14 per cent of the 50 deaths thought to be associated with HIV no clinical diagnosis of AIDS was made. Some of these deaths may have been primarily due to other causes, but we classified these deaths as HIV/AIDS. On the other hand, while among those with HIV test results in the age group15-44 years 50 per cent of deaths were HIV-associated, only 33 per cent of all deaths in this age group were attributed to HIV/AIDS based on verbal autopsy interviews and HIV data.

In Rakai, Uganda, AIDS was frequently reported as the cause of death during verbal autopsy interviews (Sewankambo et al. 1994). Almost three-quarters of the HIV-associated deaths were identified as AIDS by the respondents, while specificity was also high. Awareness of HIV/AIDS in our study population is high, but HIV prevalence and mortality are considerably lower than in the Ugandan population. Perhaps as a consequence, only eight respondents mentioned AIDS as the cause of death, either spontaneously or when prompted. In seven of these deaths the final diagnosis was AIDS. It can safely be assumed that the HIV status of the deceased was not known to the large majority of respondents as very few people in Kisesa are at present aware of their serostatus. The lack of reporting of AIDS as a cause of death may simply be due to lack of recognition of the symptoms: in 1994-95 64 per cent of respondents 15-44 years said they did not know anyone who had AIDS or had died of AIDS (Kisesa Sero-Survey Team 1996). Alternatively, there may be reluctance to recognize or report AIDS, as it still is a highly stigmatizing condition. Finally, witchcraft plays a very important role in the explanatory models of disease and death of this population. In this study this was evidenced by the high proportion of deaths attributed to witchcraft, the high level of use of traditional healers and the homicide of three older women who were suspected of witchcraft. Anthropological studies have also shown the importance of local belief systems (Washija and Pool 1996).

In sum, adult mortality in this rural area is high and 42 per cent of persons aged 15 will die before their sixtieth birthday at current mortality rates. Mortality in Kisesa, with an HIV prevalence of about six per cent in 1994-95, has increased by about one-third owing to

HIV/AIDS, and further increase is likely. Other infectious diseases cause nearly a quarter of deaths and non-communicable diseases are still a relatively minor cause. The occurrence of the AIDS epidemic may have further delayed the onset of the epidemiological transition in many parts of Africa.

References

Chandramohan, D., G.H. Maude, L.C. Rodrigues and R.J. Hayes. 1994. Verbal autopsies for adult deaths: issues in their development and validation. *International Journal of Epidemiology* 23: 213-222.

Feachem, R.G.A., T. Kjellstrom, C.J.L. Murray, M. Over and M.A. Phillips.1992. *The Health of Adults in the Developing World.* New York: Oxford University Press.

Gregson, S., G.P. Garnett and R.M. Anderson.1994. Is HIV-1 likely to become a leading cause of adult mortality in Sub-Saharan Africa? *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology* 7:839-852.

Gribble, J.N. and S.H. Preston (eds). 1993. *The Epidemiological Transition: Policy and Planning Implications for Developing Countries*. *Workshop Proceedings*. Washington DC: National Academy Press.

Grosskurth, H., F. Mosha, J. Todd, et al. 1995. A community trial of the impact of improved STD treatment on the HIV epidemic in rural Tanzania: 2. Baseline results. *AIDS* 9:927-934.

Kalluvya, S.H., M. Ishengoma, E.N. Mkumbo, A.H. Klokke and J.T. Boerma. 1996. HIV/AIDS in the medical wards of an urban referral hospital, Tanzania. TANESA Working Paper No. 9. Mwanza.

Kamali, A., H.U. Wagner, J. Nakiyingi, I. Sabiiti, J.F. Kengeya-Kayondo and D.W. Mulder. 1996. Verbal autopsy as a tool for diagnosing HIV-related adult deaths in rural Uganda. *International Journal of Epidemiology* 25:679-684.

Kisesa Sero-Survey Team. 1996. Kisesa sero-survey 1994-1995. Report of basic findings. TANESA Internal Report Series No.8. Mwanza.

Kitange, H.M., H. Machibya, J. Black, et al. 1996. Outlook for survivors of childhood in sub-Saharan Africa: adult mortality in Tanzania. *British Medical Journal* 312:216-220.

Mulder, D.W., A.J. Nunn, A. Kamali, J. Nakiyingi, H.U. Wagner and J.F. Kengeya-Kayondo. 1994. Two-year HIV1-associated mortality in a Ugandan rural population. *Lancet* 343:1021-1023.

Murray, C.J.L., G. Yang and X. Qiao. 1992. Adult mortality: levels, patterns and causes. Pp. 23-111 in *The Health of Adults in the Developing World,* ed. R. G. A. Feachem, T. Kjellstrom, C.J.L. Murray, M. Over and M.A. Phillips. New York: Oxford University Press

Sewankambo, N.K., M.J. Wawer, R.H. Gray, D. Serwadda, C. Li, R.Y. Stallings, S.D. Musgrave and J. Konde-Lule. 1994. Demographic impact of HIV infection in rural Rakai district, Uganda: results of a population-based cohort study. *AIDS* 8:1707-1713.

Timaeus, I. 1993. Adult mortality. Pp. 218-255 in *Demographic Change in Sub-Saharan Africa*, ed. K. A. Foote, K.H. Hill and L.G. Martin. Washington DC: National Academy Press.

Todd, J., R. Balira, H. Grosskurth, et al . 1997. HIV-associated mortality in a rural Tanzanian population. *AIDS* 11:801-807.

Washija, N.R. and R. Pool. 1996. Interpretations of illness in the fishing villages on Lake Victoria, Magu district, Tanzania. TANESA Working Paper No. 12. Mwanza.

World Bank. 1992. Tanzania. AIDS assessment and planning study. Washington DC.

World Health Organization. 1994. WHO case definitions for AIDS surveillance in adults and adolescents. *Weekly Epidemiological Record (WHO)* 69:273-280.

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The impact of HIV on morbidity and mortality from tuberculosis in sub-Saharan Africa: a study in rural Malawi and review of the literature*

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Abstract

Since the mid-1980s tuberculosis (TB) case numbers and HIV seroprevalence have both risen sharply in sub-Saharan Africa. Estimates for the relative risk of TB in those infected with HIV have ranged from less than five to more than 20. The proportion of TB cases attributable to HIV (the population attributable fraction) has been calculated for several populations but is difficult to interpret if no account is taken of the age and sex distribution of the cases. In a rural area of Malawi we have studied the proportion of TB attributable to HIV over time. Nearly 40 per cent of smear-positive TB cases in this rural area of Malawi can now be attributed directly to HIV. The actual effect of HIV on TB is even greater than this because increased case numbers increase transmission of tuberculosis infection to both HIV-infected and non-infected sections of the population. We compare our findings with others from sub-Saharan Africa and discuss reasons for the differences, and methodological issues in interpretation

In the presence of the HIV epidemic the number of cases of tuberculosis (TB) reported in sub-Saharan Africa has increased sharply (De Cock et al. 1992). Increases in TB of more than ten per cent per year have been reported from several countries (Cantwell and Binkin 1996). In Malawi notified cases of TB rose from 5334 in 1985 to 19,496 in 1994 (Harries et al. 1996).

Increases in TB have also been reported in many other parts of the world, but the reasons for the increases are not the same in all populations. In Africa the increases are assumed to be due to HIV, but increases are also found in Eastern Europe, in areas relatively untouched by the HIV epidemic (Raviglione et al. 1994). Reasons for reported increases in TB other than through HIV include changes in ascertainment and reporting, and real increases in TB due to poverty, inadequate TB control programs and increasing drug resistance. These effects are not

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independent. For example, a good control program should mitigate the effect of HIV in increasing TB rates, and there is some evidence that it can do so (Cantwell and Binkin 1996). The first effect of an improved control program may be to improve case finding and notification so an increase in reported cases is expected although the actual number may start to go down.

In this paper we examine trends in TB and HIV in a rural area of Malawi. We estimate the impact of HIV on TB morbidity and mortality in this population and elsewhere in sub-Saharan Africa.

Methods

Karonga District, a rural area in Northern Malawi, has been the site of a large epidemiological study of mycobacteria (The LEPRA Evaluation Project/Karonga Prevention Study or KPS) since 1978. HIV infection probably reached the district in the early 1980s and seroprevalence levels between four and 25 per cent have been measured in antenatal clinics in different parts of the district in recent years.

House-to-house total population surveys were carried out in 1980-84 and 1986-89 to ascertain cases of TB and leprosy, the second survey forming the recruitment phase of a trial of repeat BCG and/or BCG plus killed *M.leprae* vaccination (Ponnighaus et al. 1993; Karonga Prevention Trial Group 1996). Since the end of the second survey KPS staff have been stationed in health centres and the district hospital outpatients department to assess all attenders for TB and leprosy. In addition, house-to-house case-finding surveys were conducted in selected areas of the district. In the surveys and health centres individuals were examined for leprosy and asked about cough. Sputum specimens were collected from those with cough of at least three weeks' duration, and biopsies were taken from those with suspected tuberculous lymphadenopathy or cutaneous TB. Throughout the period symptomatic patients could self-present at any time. No matter how they were originally identified, all TB patients treated in the hospital were seen by KPS staff and further specimens for confirmation of the diagnosis were taken if necessary. Since 1988 blood samples for HIV testing have been requested from all adult TB patients.

Sputum specimens were examined by fluorescence and light microscopy in the project headquarters in Malawi. Cultures were set up in Malawi, and those that macroscopically suggested *M.tuberculosis* were sent to the UK PHLS laboratory in Cardiff for species confirmation and drug sensitivity testing. Biopsy specimens were examined in London. Patients were classified as having 'certain or probable' tuberculosis if they had a positive culture or smear, excluding those who only had one smear with fewer than ten acid fast bacilli. Patients with a clinical diagnosis of TB lymphadenopathy, or with any form of extra-pulmonary TB confirmed by biopsy or culture, were also included as 'certain or probable' TB (Karonga Prevention Trial Group 1996). Those who had previously been seen by the project without TB, and had no history of previous TB, were considered incident cases. Age-sex specific rates for smear-positive tuberculosis were estimated using the second survey population as the denominators.

A case-control study of leprosy and HIV was started in the district in 1988; initial results have already been published (Ponnighaus et al. 1991). In 1990 a parallel study of TB was started and is continuing. Cases were certain or probable incident cases of TB (or leprosy) diagnosed in the district among people aged 14 years and above. Controls were selected randomly from the computer database from the second population survey. Controls were matched on sex, age group (within 5 years up to age 35 and within 10 years thereafter) and area of residence (living within the same square kilometre as the case, or adjacent square kilometre if insufficient eligible controls were available). This study will be reported fully elsewhere.

HIV testing was carried out in the project laboratory using a four-test protocol as previously described (Sterne et al. 1995). The total population of controls selected for both leprosy and TB studies, who had HIV results available, has been used to estimate HIV prevalence in the district at different times. It was possible for a control to be chosen more than once, but in this analysis each subject has been included only once, at the time of first selection.

The prevalence of HIV seropositivity in the general population over the age of 14 years was calculated using the age-sex-period specific estimates from the control population, and the age and sex structure of the population recorded in the second population survey (1986-89). The prevalence (p) in different time periods was used, together with the odds ratio calculated from the case-control study as an estimate of the relative risk (RR), to calculate the proportion of TB cases attributable to HIV (the population attributable fraction or PAF) according to the formula:

$$PAF=p(RR-1)/(p(RR-1)+1)$$

This formula has been used in many studies, but may not give the correct answer if, as is the case, both the HIV seroprevalence and the TB incidence vary in different sections of the population, for example, with age and sex. We have therefore also calculated PAF separately for each age-sex-period group and obtained an overall PAF for each time period by weighting these estimates by the number of cases of TB in each age-sex-period group (Schlesselman 1982).

Results

Between 1988 and 1995, 2334 individuals were seen as controls for leprosy or TB patients and had their HIV status recorded. HIV status by age, sex and year is shown in Figure 1. Since relatively few people were seen between 1992 and 1995 the data from this time period have been pooled. The prevalence of HIV has increased markedly except in men under 25 and in the oldest age groups. The infection has been identified in all areas, although Karonga township has been most affected

Figure 1

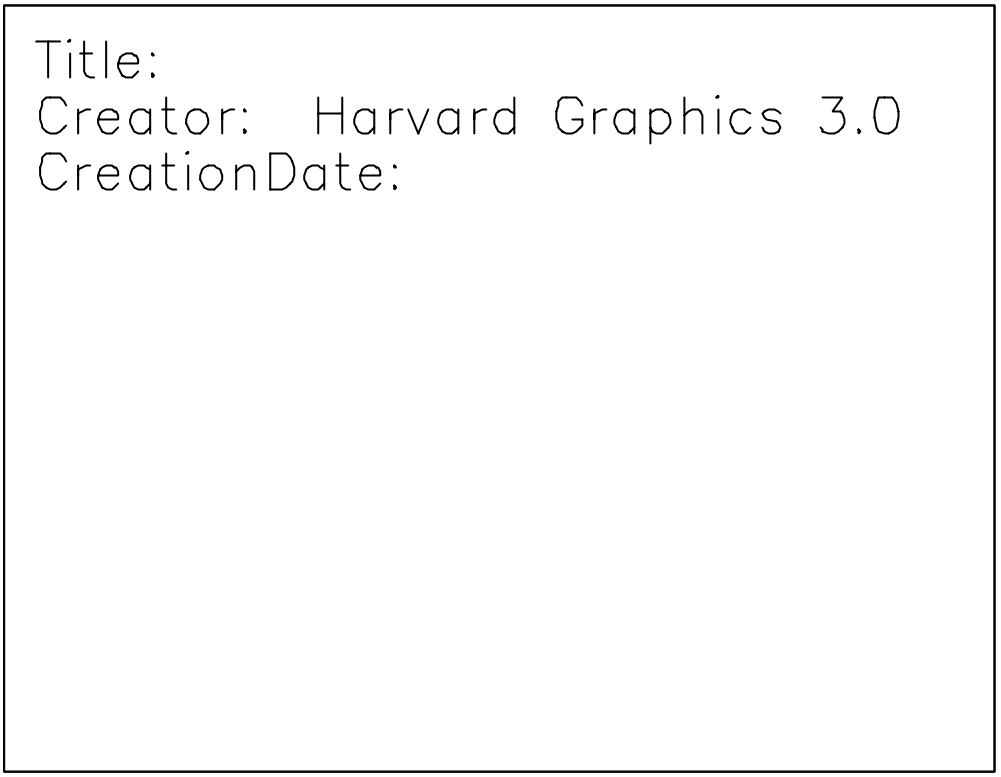


Figure 1. HIV status of individuals blood-tested as controls for leprosy or TB patients in Karonga District, 1988-1995.

TB has increased in the district over the period of study, and the increase has been largest in women between 15 and 44, and men over 25 (Figure 2). The number of 'certain or probable' smear-positive cases diagnosed increased from 50 per year to over 100 per year between 1986 and 1994.

Figure 2

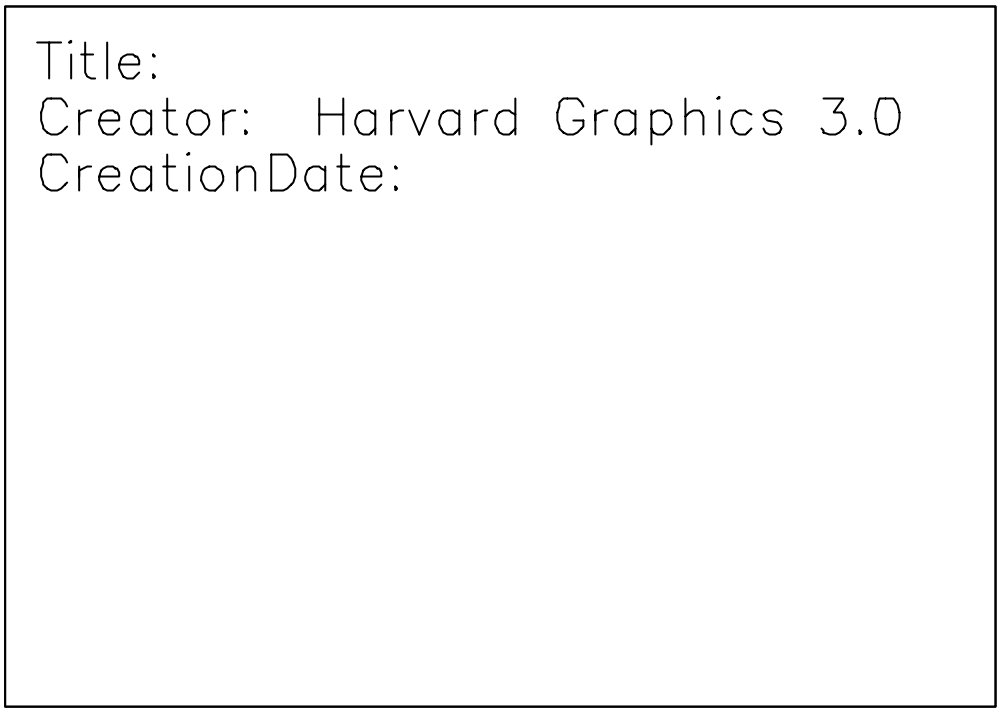


Figure 2. Smear-positive pulmonary TB in Karonga District by age, sex and time period. Cases with only a single scanty smear and no culture evidence of TB are excluded.

Preliminary results from the case-control study show that 101/255 TB cases and 59/558 controls were HIV positive (OR 7.41, 95% confidence interval (CI) 4.41-12.4). The analyses were repeated for the 212 smear-positive pulmonary cases and their controls, giving an odds ratio for HIV of 6.26 (95% CI 3.58-11.0).

PAF was calculated using the odds ratio of 6.26 for smear-positive TB by the two different methods discussed above. The results are shown in Table 1. The proportion of adult TB cases attributable to HIV rose from 17 per cent to 39 per cent over the period studied, after adjusting for age and sex. Figure 3 compares the observed and estimated TB cases without HIV.

Table 1.
The proportion of smear-positive pulmonary TB attributable to HIV (PAF) in the adult population of Karonga District by time period.

Time period	HIV+ (%)	PAF (%) (crude)	PAF (%) (Age-sex adjusted)
1988/89	2.96	13.6	17.0
1990/91	5.26	21.8	19.7
1992/95	10.78	36.4	38.5

Figure 3

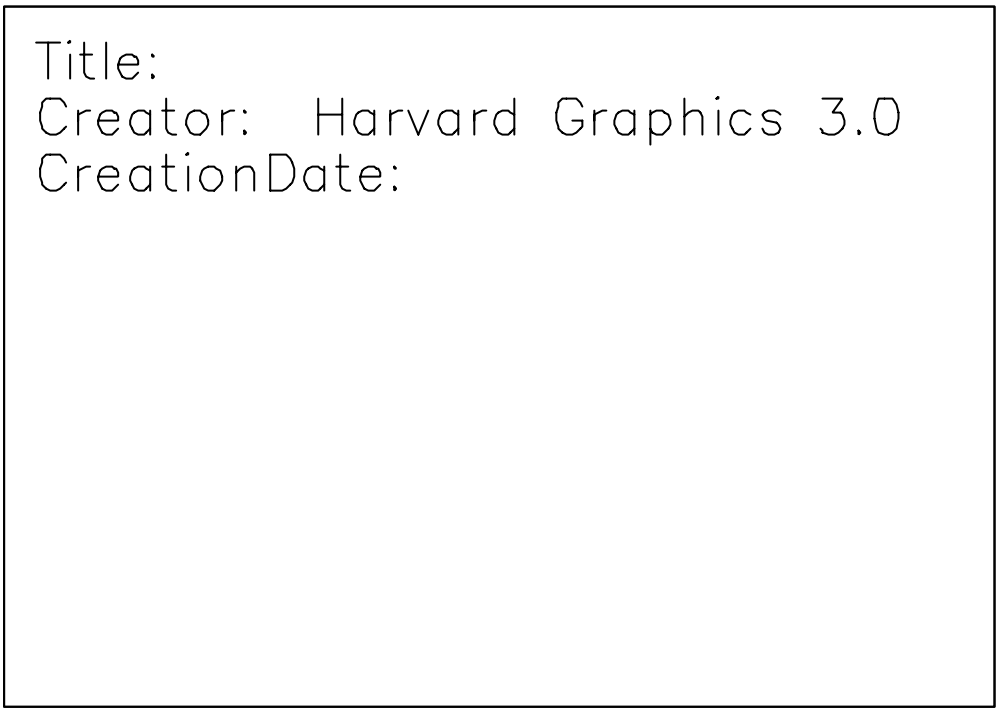


Figure 3. Smear-positive pulmonary TB in Karonga District: observed cases and numbers expected in the absence of HIV.

Discussion

Trends in TB and HIV

HIV seroprevalence has now reached more than 15 per cent in the age groups most at risk in Karonga District: women aged 15-45 and men aged 25-55. This may be an underestimate since the population controls on whom these figures are based were people who were present in the district during the second survey (1986-89) and were then found again in the same area of residence, and moreover agreed to be tested. The prevalence may be higher in more mobile sections of the population and among refusals.

The pattern of increased incidence of TB since the late 1980s reflects the increase in HIV, with large increases being seen in all age groups except the youngest men and oldest women. Changes in ascertainment procedures during the study, from house-to-house surveys to health centre based, are also likely to have influenced the numbers of cases diagnosed in each time period.

HIV-infected TB patients have much higher mortality rates than do those without HIV, so the effect of HIV on TB mortality rates will be even more marked than its effect on incidence rates. In Karonga District, among smear-positive patients, 32/134 HIV-positive and 15/281 HIV-negative patients died during therapy (hazard ratio 5.6, 95% CI 3.0-10 in a Cox regression analysis: Karonga Prevention Study unpublished results). Similarly increased mortality rates have been reported in other studies (Perriens et al. 1991; Nunn et al. 1992; Elliott et al. 1995; Ackah et al. 1995). The mortality rate in HIV-positive patients depends on the degree of immune deficiency (Ackah et al. 1995). Many of the deaths are not directly attributable to TB (Nunn et al. 1992; Elliott et al. 1995) and there is some evidence that TB may accelerate the progression of HIV disease (Goletti et al. 1996).

Relative risk of TB in HIV-positive and HIV-negative persons

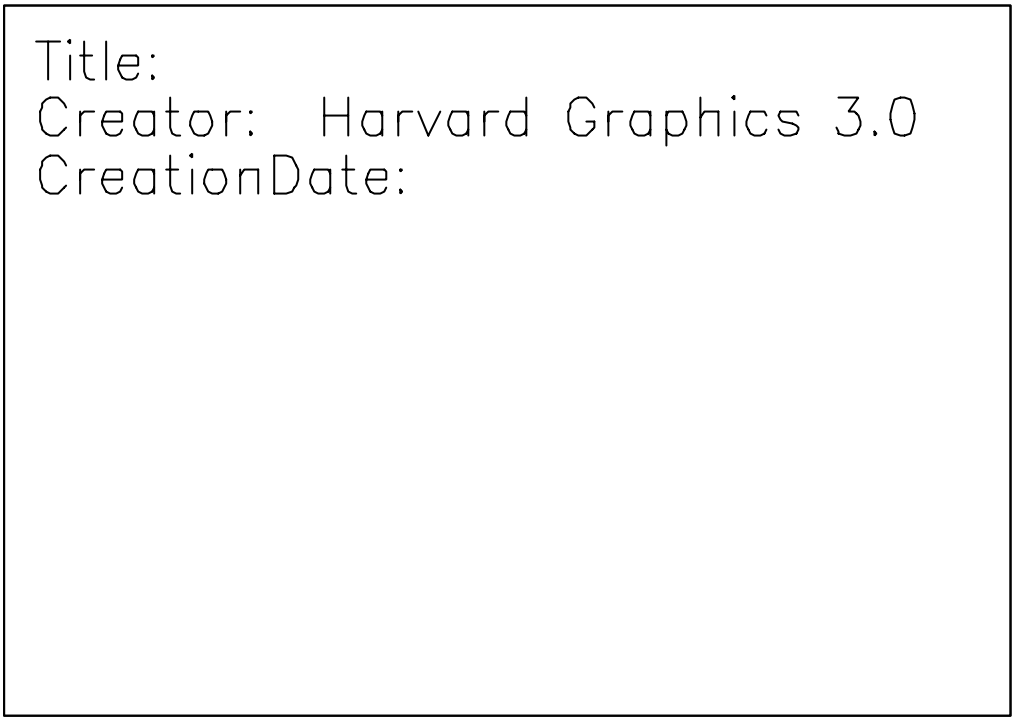
The results of the current case control study are similar to those of a previous study : odds ratio for the association of HIV and TB of 7.4 (95% CI 3.3-16.7) for the period 1988-89 in Karonga District (Ponnighaus et al. 1991). Several previous studies[1] in Africa have also estimated relative risks for the association between HIV and TB, and are summarized in Figure 4. The studies have varied in the certainty of diagnosis of cases of TB, whether they include all types of TB or only pulmonary cases, and in their choice of control groups. These factors are likely to bias the measurements of relative risk in various ways.

Since several HIV-related diseases can be difficult to distinguish clinically from TB, studies which include all diagnosed TB cases rather than bacteriologically confirmed cases are likely to overestimate the relative risk. Several studies have shown higher rates of HIV positivity in patients with extra-pulmonary TB than in those with pulmonary TB (Elliott, Halwiindi et al. 1993), so lower relative risks would be expected in studies which include only pulmonary TB cases. In our preliminary results, and in two other studies from sub-Saharan Africa in which data have been presented separately for all diagnosed TB and bacteriologically confirmed pulmonary TB, lower relative risks were found for confirmed pulmonary TB (Figure 4). In a cohort study in Zaire of HIV-positive and HIV-negative women of childbearing age in which regular follow-up visits were made, the rate ratio was 10

[1] Braun et al. 1991; Ponnighaus et al. 1991; De Cock et al. 1991; Allen et al. 1992; Migliori et al. 1992; Orege et al. 1993; Van den Broek et al. 1993; Morvan et al. 1994; Houston et al. 1994; Leroy et al. 1995; Van Cleeff and Chum 1995; Naucler et al. 1996; Chum et al. 1996.

(95% CI 1.5-47) for smear-positive pulmonary TB and 26 (95% CI 5-125) for all diagnosed TB (Braun et al. 1991). In southern Tanzania, in a study with blood donor controls, the odds ratio was 8.1 (95% CI 4.4-16.3) for smear-positive TB and 11.8 (95% CI 7.9-18.3) for all TB (Van Cleeff and Chum 1995).

Figure 4
Summary of studies of the association between HIV and TB in Africa..



Notes: Studies are presented in groups by study type and comparison group used. For each study the country, years of data collection, and confounders adjusted for are given. Studies referring to all diagnosed TB and those restricted to microbiologically confirmed pulmonary TB are distinguished. For the studies in the Ivory Coast and Guinea Bissau cases and controls infected only with HIV-2 have been excluded. Rate ratios are presented for the cohort studies and odds ratios for the case-control studies, except for the study in Tanzania 91/93 (Chum et al. 1996) in which the risk ratio was given and it was not possible to calculate an odds ratio from the information given.

Blood donors have often been used as controls, but they are not a random sample of the community, and the section of the community they represent will vary from place to place and according to whether they are paid or not. Since donors are likely to be healthy, and in some populations are prescreened for risk factors for HIV, studies which use blood donor controls may overestimate the relative risk. In some populations potential donors with perceived risk factors for HIV infection are excluded from blood donation. On the other hand blood donors may be more likely to be urban dwellers and have higher HIV rates than the general population. Hospital patients are sometimes used as controls, but are likely to have high rates of HIV unless carefully chosen to exclude patients seen for HIV-related conditions, so their use will tend to underestimate the relative risk. Hospital workers have been used as controls, but they may come from a different section of society than do the TB patients and they may

be occupationally exposed to HIV, so they are also unlikely to be a suitable comparison group.

The variation in relative risk seen between the different studies may be due to bias and the lack of adjustment for confounding in many of the studies. On the other hand the confidence intervals on many of the estimates are wide. Most studies are compatible with a 'true' relative risk of about 7, but there may not be a single, constant relative risk in all situations.

There is some suggestion that the relative risk varies with age. The highest relative risks have been reported from three cohort studies of women of childbearing age, one in Zaire (Braun et al. 1991) and two in Rwanda. In Rwanda the rate ratio for all TB was 21.8 (95% CI 5.1-92.9) in one study (Allen et al. 1992) and 18.2 (95% CI 2.4-137) in the other (Leroy et al. 1995).

A case-control study in Mwanza, Tanzania, based on all TB cases, and controls selected from a population database, found an odds ratio of 8.3 (95% CI 6.4-11.0) overall, but a higher odds ratio for the 25-34 year age group (13.4, 95% CI 8.9-20.7) and lower odds ratio for those aged 45-54 (2.9, 95% CI 1.0-7.9) (Van den Broek et al. 1993). Two other Tanzanian studies, which used blood donor controls and did not adjust for any possible confounders, found a similar pattern of higher odds ratios in the 25-34 year age group and lower ones in those over 45 years (Chum et al. 1996; Van Cleeff and Chum 1995). In Harare, Zimbabwe, a study with hospital controls also found the lowest odds ratios in the oldest age group (recalculated as 3.0, 95% CI 1.5-6.1 in those over 45), but the highest was in those aged 35-44 (recalculated as 8.3, 95% CI 4.3-16.3) (Houston et al. 1994). A study of bacteriologically proven TB cases in Kenya using neighbourhood controls found an overall odds ratio of 4.9 (2.6-6.8), with no clear pattern by age group (Orege et al. 1993). And there was no clear trend with age in Abidjan, Ivory Coast, in a study which compared pulmonary TB cases with blood donors, considered HIV-1 and HIV-2 infection, and found an overall odds ratio (recalculated) of 5.1 (95% CI 4.1-6.7) (De Cock et al. 1991).

The true odds ratio may change with time, as the HIV epidemic matures, since the proportions of people with HIV who have different levels of immunosuppression will change. There may also be variation as segments of the population with different underlying risks of TB are affected by HIV. HIV may affect the relative risk of developing primary disease, reactivation disease and reinfection disease differently, so the overall odds ratio would depend on the proportion of the population at risk for the various types of disease. Older people are more likely to have been previously infected with TB so differences in relative risk at different ages may be a reflection of the different influence of HIV on the different pathways to TB disease.

Population attributable fraction

Using a constant relative risk we have calculated the proportion of TB cases attributable to HIV infection in the adult population. Since neither the population prevalence of HIV nor the relative risks were accurately measured, this is inevitably a rough estimate both in our study and in others. The PAFs calculated overall and those adjusted for age and sex are similar. This will not always be the case, if other populations such as children are included in the calculations. For instance, data from the Malawi AIDS program for the first six months of 1996 found that 12.8 per cent of reported AIDS cases in the country were children aged less than 15 years. Using this to estimate the proportion of children HIV-positive in our population and including the (few) smear-positive TB cases seen in children we can recalculate the PAF for the period 1992-95 by the two methods. The all-age population prevalence of HIV is estimated at 6.7 per cent, giving a PAF of 25.9 per cent by the crude method. Using separate PAFs for different age and sex groups gives an age-sex adjusted PAF of 37.7 per cent.

It is often unclear in published studies which age groups have been included in estimating HIV prevalence and PAF. Blood donors, presumably young adults, were used to calculate a PAF of 35 per cent for all adult TB in Abidjan in 1989, for HIV-I and HIV-II combined (De Cock et al. 1991). Estimates based on blood donors and antenatal clinic attenders were used together with census data, TB registers and seroprevalence data among TB patients to calculate PAFs of 36 per cent in 1989 and 40 per cent in 1991 in separate studies in Abidjan (Richards et al. 1995). In Mwanza a PAF of 29 per cent was calculated for those aged 15-54 years with all types of TB (Van den Broek et al. 1993). In a study from southern Tanzania with blood donor controls the estimated PAF rose from 19 per cent to 31 per cent between 1987 and 1990, but it is not clear to what age group the population HIV prevalence figures used refer (Van Cleeff and Chum 1995).

Effect of HIV on TB

The PAF measures only the direct effect of HIV on TB cases. As the HIV epidemic progresses and the number of TB cases including smear-positive TB cases rises, transmission to both the HIV-positive and HIV-negative population must increase. Lienhardt and Rodrigues (1997) have calculated a factor *f* by which the number of cases of TB increases in a population as a direct effect of HIV:

$$f = \frac{\text{risk of TB in general population } (r_t)}{\text{risk of TB in HIV-negative part of population } (r_o)}$$

$$= 1/(1\text{-PAF}) \qquad (\text{since PAF}=(r_t - r_o)/r_t)$$

They go on to argue that, if HIV-infected TB cases are as infectious as HIV-negative TB cases and there is random population mixing, then the annual risk of infection in the short term would be expected to increase by the same factor *f* due to the infections caused by these 'extra' cases. In the longer term there would be a further increase due to transmission from second-generation cases in the HIV-negative population. Based on the above formula, PAFs of between 30 per cent and 40 per cent translate into expected increases in the annual risk of infection of between 40 per cent and 70 per cent above what they would have been in the absence of HIV. This should ultimately increase the number of cases of TB in both HIV-positive and HIV-negative individuals, through infection or reinfection.

More complex models for estimating the effect of HIV on TB have also been developed (Heymann 1993; Schulzer et al. 1994). Models are inevitably constrained by assumptions made in defining their parameters, and in both of these models transmission is also apparently assumed to be the same from HIV-positive and HIV-negative TB cases. The relative infectiousness of HIV-infected TB cases is uncertain, however, making this indirect effect of HIV on TB difficult to quantify.

Three studies in Africa have compared infection with *M. tuberculosis* (as assessed by tuberculin responses) and disease rates in contacts of HIV-positive and HIV-negative index patients with pulmonary TB. In Kinshasa (Klausner et al. 1993) and Nairobi (Nunn et al. 1994) infection rates were similar in the two sets of contacts, and in Lusaka they were lower in the contacts of the HIV-positive group even after adjusting for degree of sputum smear positivity (Elliott, Hayes et al. 1993). In all three studies, BCG vaccination coverage and background TB infection rates were high which will have led to probably non-differential misclassification of tuberculous infection, and is likely to have biased the relative risks towards 1. The rates of TB disease were similar in contacts of HIV-positive and HIV-negative index cases in all the studies, but the numbers were small. In a study in Florida, where BCG is

not used and background TB infection rates are low, lower tuberculin positivity rates were found in contacts of HIV-positive patients than in those of HIV-negative patients (Cauthen et al. 1996). Lower infectiousness of HIV-positive patients may be partly explained by lower degrees of smear positivity (Elliott, Hayes et al. 1993), and, in some places, by closer monitoring of HIV-positive patients leading to earlier diagnosis.

Studies of transmission among contacts inevitably rely on diagnosed patients, who should become non-infectious soon after the start of treatment, but many TB patients go undiagnosed. Since HIV-positive TB patients have a higher mortality rate and are likely to die earlier than do HIV-negative TB patients, transmission from undiagnosed TB cases is likely to be less from the HIV-positive than from the HIV-negative individuals, mainly because the former would have less time during which to transmit.

Conclusions

HIV has led to an increase in TB cases in sub-Saharan Africa, with 30-40 per cent of TB cases, and an even higher proportion of TB deaths, now being directly attributable to HIV in many areas. The indirect effect of this increase in terms of the annual risk of infection and subsequent generations of cases may be less than some have predicted, since HIV-positive TB patients may transmit infection to fewer people than do HIV-negative patients, but some increase in infection and subsequently in disease rates would still be expected. It remains to be seen whether the projected increase in TB can be contained by TB control programs, and whether the increase is sufficient to reverse the long-term downward trend in the annual risk of infection recorded in many populations (Murray, Styblo and Rouillon 1990).

References

Ackah, A.N., D. Coulibaly, H. Digbeu, K. Diallo, K.M. Vetter, I.-M. Coulibaly, A.F. Greenberg and K.M. De Cock. 1995. Response to treatment, mortality, and CD4 lymphocyte counts in HIV-infected persons with tuberculosis in Abidjan, Côte d'Ivoire. *Lancet* 345:607-609.

Allen, S., J. Batungwanayo, K. Kerlikowske, et al. 1992. Two-year incidence of tuberculosis in cohorts of HIV-infected and uninfected urban Rwandan women. *American Review of Respiratory Disease* 146:1439-1444.

Braun, M.M., N. Badi, R.W. Ryder, et al. 1991. A retrospective cohort study of the risk of TB among women of childbearing age with HIV infection in Zaire. *American Review of Respiratory Disease* 143:501-504.

Cantwell, M.F. and N.J. Binkin. 1996. Tuberculosis in sub-Saharan Africa: a regional assessment of the impact of the human immunodeficiency virus and National Tuberculosis Control Program quality. *Tubercle and Lung Disease* 77:220-225.

Cauthen, G.M., S.W. Dooley, I.M. Onorato, W.W. Ihle, J.M. Burr, W.J. Bigler, J. Witte and K.G. Castro. 1996. Transmission of *Mycobacterium Tuberculosis* from tuberculosis patients with HIV infection or AIDS. *American Journal of Epidemiology* 144:69-77.

Chum, H.J., R.J. O'Brien, T.M. Chonde, P. Graf and H.L. Rieder. 1996. An epidemiological study of tuberculosis and HIV infection in Tanzania 1991-1993. *AIDS* 10:299-309.

De Cock, K.M., E. Gnaore, G. Adjorlolo, et al. 1991. Risk of tuberculosis in patients with HIV-I and HIV-II infections in Abidjan, Ivory Coast. *British Medical Journal* 302:496-499.

De Cock, K.M., B. Soro, I.M. Coulibaly and S.B. Lucas. 1992. Tuberculosis and HIV infection in sub-Saharan Africa. *Journal of the American Medical Association* 268:1581-1587.

Elliott, A.M., B. Halwiindi, R.J. Hayes, et al. 1993. The impact of human immunodeficiency virus on presentation and diagnosis of tuberculosis in a cohort study in Zambia. *Journal of Tropical Medicine and Hygiene* 96:1-11.

Elliott, A.M., R.J. Hayes, B. Halwiindi, et al. 1993. The impact of HIV on infectiousness of pulmonary tuberculosis: a community study in Zambia. *AIDS* 7:981-987.

Elliott, A.M., B. Halwiindi, R.J. Hayes, et al. 1995. The impact of human immunodeficiency virus on mortality of patients treated for tuberculosis in a cohort study in Zambia. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 89:78-92.

Goletti, D., D. Weissman, R.W. Jackson, et al. 1996. Effect of *Mycobacterium tuberculosis* on HIV replication: role of immune activation. *Journal of Immunology* 157:1271-1278.

Harries, A.D., L. Nyong'Onya Mbewe, F.M.L. Salaniponi, D.S. Nyangulu, J. Veen, T. Ringdal and P. Nunn. 1996. Tuberculosis programme changes and treatment outcomes in patients with smear-positive pulmonary tuberculosis in Blantyre, Malawi. *Lancet* 347:807-809.

Heymann, S.J. 1993. Modelling the efficacy of prophylactic and curative therapies for preventing the spread of tuberculosis in Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 87:406-411.

Houston, S., S. Ray, M. Mahari, et al. 1994. The association of tuberculosis and HIV infection in Harare, Zimbabwe. *Tubercle and Lung Disease* 75:220-226.

Karonga Prevention Trial Group. 1996. Randomised controlled trial of single BCG, repeated BCG, or combined BCG and killed Mycobacterium leprae vaccine for prevention of leprosy and tuberculosis in Malawi. *Lancet* 348:17-24.

Klausner, J.D., R.W. Ryder, E. Baende, et al. 1993. *Mycobacterium tuberculosis* in household contacts of Human Immunodeficiency Virus type 1-seropositive patients with active pulmonary tuberculosis in Kinshasa, Zaire. *Journal of Infectious Diseases* 168:106-111.

Leroy, V., P. Msellati, P. Lepage, et al. 1995. Four years of natural history of HIV-1 infection in African women: a prospective cohort study in Kigali (Rwanda), 1988-1993. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology* 9:415-421.

Lienhardt, C. and L.C. Rodrigues. 1997. The estimation of the impact of HIV infection on tuberculosis: tuberculosis risks re-visited? *International Journal of Tuberculosis and Lung Disease* 1 (in press).

Migliori, G.B., A. Borghesi, C. Adriko, et al. 1992. Tuberculosis and HIV infection association in a rural district of Northern Uganda: epidemiological and clinical considerations. *Tubercle and Lung Disease* 73:285-290.

Morvan, J.M., G. Auregan, A.J. Rasamindrakotroka, T. de Ravel and J.F. Roux. 1994. L'infection à VIH chez les tuberculeux à Madagascar. Situation en 1993. *Archives d'Institut Pasteur de Madagascar* 61:73-75.

Murray, C.J.L., K. Styblo and A. Rouillon. 1990. Tuberculosis in developing countries: burden, intervention and cost. *Bulletin of the International Union against Tuberculosis and Lung Disease* 65:6-24.

Naucler, A., N. Winqvist, F. Dias, et al. 1996. Pulmonary tuberculosis in Guinea-Bissau: clinical and bacteriological findings, human immunodeficiency virus status and short term survival of hospitalized patients. *Tubercle and Lung Disease* 77:226-232.

Nunn, P., R. Brindle, L. Carpenter, et al. 1992. Cohort study of human immunodeficiency virus infection in patients with tuberculosis in Nairobi, Kenya: analysis of early (6-month) mortality. *American Review of Respiratory Disease* 146:849-854.

Nunn, P., M. Mungai, J. Nyamwaya, et al. 1994. The effect of human immunodeficiency virus type-1 on the infectiousness of tuberculosis. *Tubercle and Lung Disease* 75:25-32.

Orege, P.A., P.E.M. Fine, S.B. Lucas, et al. 1993. A case control study on human immunodeficiency virus-1 (HIV-1) infection as a risk factor for tuberculosis and leprosy in Western Kenya. *Tubercle and Lung Disease* 74:377-381.

Perriens, J.H., R.L. Colebunders, C. Karahunga, et al. 1991. Increased mortality and tuberculosis treatment failure rate among human immunodeficiency virus (HIV) seropositive compared with HIV seronegative patients with pulmonary tuberculosis treated with 'standard' chemotherapy in Kinshasa, Zaire. *American Review of Respiratory Disease* 144:750-755.

Ponnighaus, J.M., L.J. Mwanjasi, P.E.M. Fine, et al. 1991. Is HIV infection a risk factor for leprosy? *International Journal of Leprosy* 59:221-228.

Ponnighaus, J.M., P.E.M. Fine, L. Bliss, et al. 1993. The Karonga Prevention Trial: a leprosy and tuberculosis vaccine trial in Northern Malawi - I: Methods of the vaccination phase. *Leprosy Review* 64:338-356.

Raviglione, M.C., H.L. Rieder, K. Styblo, et al. 1994. Tuberculosis trends in Eastern Europe and the former USSR. *Tubercle and Lung Disease* 75:400-416.

Richards, S.B., M.E. St Louis, P. Nieburg, I.M. Coulibaly, D. Coulibaly, L. Abouya, H.D. Gayle and K.M. De Cock. 1995. Impact of the HIV epidemic on trends in tuberculosis in Abidjan, Côte d'Ivoire. *Tubercle and Lung Disease* 76:11-16.

Schlesselman, J.J. 1982. *Case-control Studies. Design, Conduct, Analysis*. New York: Oxford University Press.

Schulzer, M., M.P. Radhamani, S. Grzybowski, E. Mak and J.M. Fitzgerald. 1994. A mathematical model for the prediction of the impact of HIV infection on tuberculosis. *International Journal of Epidemiology* 23:400-407.

Sterne, J.A.C., A.C. Turner, P.E.M. Fine, et al. 1995. Testing for antibody to Human Immunodeficiency Virus type 1 in a population in which mycobacterial diseases are endemic. *Journal of Infectious Diseases* 172:543-546.

Van Cleeff, M.R.A. and H.J. Chum. 1995. The proportion of tuberculosis cases in Tanzania attributable to human immunodeficiency virus. *International Journal of Epidemiology* 24:637-642.

Van den Broek, J., M.W. Borgdorff, N.G. Pakker, H.J. Chum, A.H. Klokke, K.P. Senkoro and J.N. Newell.1993. HIV-1 infection as a risk factor for the development of tuberculosis: a case-control study in Tanzania. *International Journal of Epidemiology* 22:1159-1165.

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HIV and fertility change in rural Zimbabwe*

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Abstract

Fertility transition and HIV epidemics are currently running parallel in some sub-Saharan African populations. Interactions between the two at the individual and population levels could accentuate or moderate the resulting demographic trends. We review a number of mechanisms through which an HIV epidemic and responses to it can affect birth rates, through the biological and behavioural proximate determinants. Uninfected as well as infected people can be affected and many of the changes could have unintended consequences for fertility at the individual level. Results from a small-scale in-depth study in two rural areas of Zimbabwe are reviewed. These indicate that the local HIV epidemic has begun to influence the proximate determinants of fertility. If observed trends persist, a modest acceleration in the recent decline in birth rates seems plausible.

Fertility transition and deterioration in mortality due to HIV[1] epidemics are perhaps the two most important demographic factors affecting socio-economic development in central, eastern and southern Africa today. Individually, but especially in combination, they can cause dramatic and substantial shifts in the demographic profile of a population, including major changes in population growth and structure, orphanhood, widowhood and household composition. Each is driven itself by socio-economic conditions, including cultural influences, levels of education and income, and patterns of income generation. These conditions affect the proximate determinants of fertility and HIV transmission, between which there is a considerable degree of overlap. Because of this overlap, biological and behavioural changes arising from HIV epidemics can have consequences for fertility trends and family planning practices. Equally, differing or changing attitudes towards fertility and family building strategies can influence the actions taken to reduce risks of HIV infection. An obvious example of this is that the desire to bear children limits the possibilities for avoiding HIV transmission within stable unions.

In this article, we consider the demographic effects of HIV epidemics in sub-Saharan African contexts with particular reference to fertility. We focus on identification of possible ways in which an HIV epidemic and responses to it can affect birth rates, through the proximate determinants; and we review evidence from a small-scale study in rural Zimbabwe

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[1] Throughout this paper HIV refers to HIV-1.

which tentatively suggests that early changes are indeed under way, the net effect of which seems likely to be an intensification of pre-existing downward pressures on total fertility.

HIV as a determinant of socio-demographic change

Theoretical evidence (Anderson, May and McLean 1988; Gregson, Garnett and Anderson 1994; United Nations 1991) and a growing body of empirical evidence (De Cock et al. 1990; Garenne et al. 1995; Gregson, Anderson et al. 1997; Mulder et al. 1994; Sewankambo et al. 1994) demonstrates that HIV epidemics will have a profound effect on the demography of many sub-Saharan populations. However, there is incomplete understanding of the processes through which this effect will occur. An initial framework for examining these processes is set out in Figure 1.

In summary, an HIV epidemic exerts an upward pressure on morbidity and mortality. This has a direct effect on death rates, population structure and population growth, which, in turn, causes changes in the extent and age-pattern of orphanhood and widowhood, and changes in household composition. The epidemic may bring about biological changes which affect birth rates. At the population level, fertility may also be affected by the adjustments in population structure, widowhood patterns and age and sexual-activity-selective mortality.

At the same time, the epidemic triggers responses at the individual and community levels. Changes in social attitudes and cultural norms can lead to behaviour changes which influence birth rates. From a female perspective, these could have both positive and negative effects. For example, reductions in access to education, due to increased emphasis on the female role as carer for the sick (De Bruyn 1992), would be negative and could result in an upward pressure on fertility, through earlier marriage and reduced contraception. On the other hand, the new situation may encourage women to challenge male domination in the spheres of sexual behaviour and family planning (Ankrah 1991). This could lead to lower rates of partner acquisition and/or an increase in contraceptive use, which could reduce fertility. In either case, changes in attitudes and behaviour to avoid HIV and AIDS could lead to changes in the proximate determinants of fertility.

Finally, any changes in attitudes and behaviour brought about by increased sickness and death and AIDS control initiatives are likely to affect the future course of the HIV epidemic: a feedback process which will determine the long-term demographic effect of the epidemic. We will examine possible changes in fertility determinants resulting from HIV epidemics in more detail, but first discuss briefly why it is important to take account of underlying trends in fertility when considering the demographic impact of HIV epidemics.

The influence of fertility change on the demographic effect of HIV epidemics

Fertility declines are now widely accepted as being under way in a number of the more developed areas of sub-Saharan Africa. HIV epidemics could serve to intensify or restrict these declines, but whether or not this is the case, patterns of fertility change will be vital in determining the demographic effect of HIV epidemics.

Figure 1
HIV as a determinant of socio-demographic change

The significance of contemporary fertility trends is illustrated in Figure 2. The graphs show levels of population growth (rate of natural increase) and maternal orphanhood projected using a published mathematical model and epidemiological and demographic estimates for Zimbabwe (Garnett and Anderson 1993, 1994; Gregson et al. 1996). Four scenarios are illustrated. In each case, migration is ignored and non-HIV-related mortality is taken to remain constant. The first is a baseline scenario in which population growth and orphanhood are assumed to remain constant at mid-1980s levels. The second scenario illustrates the effects of fertility decline and is based on the assumption that birth rates are reduced in a linear fashion between 1986 and 1998 at a similar rate to that recorded in the 1988 and 1994 Zimbabwe Demographic and Health Surveys (Mandishona 1989; Parirenyatwa 1995). Here population growth declines from just under three per cent per annum to two per cent but there is little effect on orphanhood. The third scenario assumes constant fertility but takes into account the HIV epidemic which is assumed to peak at around 25 per cent among adults aged 15-49 years in the late 1990s. Population growth declines rapidly before levelling off at around one per cent and there is a steady and substantial increase in orphanhood. Taking fertility decline and the HIV epidemic together—the fourth scenario—population growth dips below zero and orphanhood reaches extremely high levels, because fertility decline intensifies the impact of the HIV epidemic on the age-structure of the population. Among children aged under 15 years, a greater proportion are aged over ten years and thus have a higher cumulative risk of their mothers' having died.

If women with HIV infection have reduced birth rates (Serwadda et al. 1997), the effect of the epidemic on population growth would be slightly greater than indicated, the reduction in fertility outweighing the drop in early-childhood mortality, but the rise in orphanhood would be less marked (Gregson et al. 1994:84).

In the cases of population growth and orphanhood, the effect of fertility decline is to magnify that of HIV-related mortality. However, in other respects the effects can offset each other. For example, declining fertility tends to moderate the extent to which an HIV epidemic reduces the mean age of the working population (say 15-64 years).

Socio-economic and proximate determinants of fertility and HIV and AIDS epidemics

In demography, there is a well established theory of intermediate or proximate determinants of fertility through which the influence of the ultimate structural and socio-economic determinants is mediated (Davis and Blake 1956; Bongaarts and Potter 1983; Bongaarts, Frank and Lesthaeghe 1984). Possible pathways through which an HIV epidemic could influence fertility through these proximate determinants are examined in the next section. However, there is a similar set of biological and behavioural factors which mediate the spread and effect of HIV infections within a population (Mason 1994). The relationship is summarized in Figure 3. As with fertility, for any given underlying demographic profile, one or more of the proximate determinants must differ or vary if the impact of HIV is to differ between populations or within the same population over time. Variations in levels of proximate determinants such as sexual behaviour patterns and the length of the incubation and infectious periods account for differences in the speed, shape and ultimate size of HIV and AIDS epidemics in different populations (Jacquez et al. 1988; Anderson, Gupta and Ng 1990; Gregson et al. 1994). To have an impact, AIDS prevention strategies must bring about changes in at least one of the proximate determinants of HIV epidemics by appropriately addressing the underlying socio-economic causes.

Figure 2
Demographic impact of HIV and fertility decline

Figure 3
Socio-economic and proximate determinants of HIV-1 and AIDS epidemics

A list of the principal proximate determinants of HIV and AIDS epidemics is given in a previous paper (Blair Research Institute 1996; Gregson et al. 1996). Our interest here is in highlighting the areas of overlap between proximate determinants of HIV and AIDS epidemics, on the one hand, and those of fertility, on the other. The extent of overlap is considerable (Table 1) and similar socio-economic factors such as culture, education, religion and income differentials are likely to be important in each case. Clearly changes in the proximate determinants of HIV and AIDS are likely to result in changes in fertility and *vice versa*.

Table 1
Common proximate determinants of HIV-1 and fertility

Determinants	Role re HIV-1	Role re fertility
Sexual unions	High numbers of partners tend to increase the chance of acquiring infection; extent depending on sexual mixing patterns	Fertility generally concentrated in stable unions; polygyny can result in lower fertility
Sexual abstinence and coital frequency	Low frequency of sex reduces the risk of infection	Low frequency of sex reduces the chance of conception
Lactation	Increases chances of vertical transmission	Extends postpartum amenorrhoea
Condom use	Regular use reduces the risk of infection	Regular use reduces the chance of conception
Injections	Can transmit HIV-1 if needles are contaminated from earlier use	Injectables can be used as contraceptives
Sterility and natural fecundity	STDs' co-factor role in sexual transmission	STDs - and possibly HIV-1 - cause sterility
	Malnutrition may increase susceptibility to infection and reduce the incubation period to AIDS	Malnutrition may reduce natural fecundity

The effects of each factor are considered here in isolation. In practice, of course, they may interact.

Pathways for an influence of HIV on fertility

There is mounting evidence for associations between HIV infection and reduced fertility in sub-Saharan African populations (Batter et al. 1994; Sewankambo et al. 1994; Serwadda et al. 1997). Infertility may be a risk factor for HIV infection (Boerma, Urassa and Isingo 1996). However, it also seems likely that HIV itself can result in lower fertility among infected individuals. In a recent analysis of a large-scale population-based study in Uganda, Serwadda et al. (1997) found a 52 per cent lower adjusted risk of pregnancy among women with HIV infection (but without syphilis) compared to women without HIV. As was the case in an earlier small-scale study in Zaire, women with more advanced disease had the lowest pregnancy rates but there was also evidence for subfecundity among those with asymptomatic infections (Ryder et al. 1991). The authors suggested that higher levels of spontaneous

abortions and stillbirths could be important in explaining their findings. Other possibilities include increased amenorrhoea (Widy-Wirski et al. 1988), reduced spermatozoa among male partners (Martin et al. 1991) and reductions in coital frequency during periods of sickness.

At the population level, lower fertility among women with HIV infection can, in some circumstances, result in an overall increase in birth rates, over time (Zaba and Collumbien 1996). For example, this could occur where women with lower fertility due to other sexually transmitted infections are more likely to acquire HIV infection and thus suffer higher mortality. It is also important to recognize that biomedical and behavioural responses to HIV epidemics which affect birth rates can extend to persons not infected themselves. The substantial reduction in fertility experienced by HIV-infected women notwithstanding, behaviour changes could emerge as being more important than biological changes at the population level: more people being involved and the effects being longer-term. Relatively few people infected with HIV in sub-Saharan Africa are likely to be aware of their condition until the later stages, which are typically very short. Unless counselling and testing services become widely available and accepted, it seems unlikely that behaviour change will be significantly more extensive among people infected with HIV than in the rest of the population.

At the individual level, it is helpful to differentiate between deliberate and unintended fertility effects. It is sometimes suggested that couples will seek to accelerate their childbearing for fear that they might not live throughout the normal reproductive lifespan (Setel 1996). This seems especially plausible in societies which emphasize the importance of childbearing. However, other concerns such as reduced survival chances of the child, the adverse effects of orphanhood on the child and the broader health consequences of rapid childbearing for the woman may often be paramount, particularly where the woman or couple already has children. Most cultures emphasize succession by living children and in Zimbabwe, at least, while most people have heard that HIV can be transmitted from a mother to a child at birth, relatively few realize that there is a more than 50-50 chance that this will not occur. In many populations, conscious control of fertility remains unusual and the possibilities for deliberate acceleration of childbearing are clearly limited.

Irrespective of whether individuals alter behaviour with the intention of influencing their rate of childbearing, broader changes, instituted primarily to reduce the risks of HIV transmission, may have this consequence. Increases in abstinence and condom use would tend to reduce fertility, particularly where effective contraceptives have not been widely used in the past. Conversely, earlier and more effective treatment for other sexually transmitted diseases or early termination of breastfeeding would tend to increase fertility. It seems quite plausible that it will be changes of this nature, which will involve currently uninfected as well as infected individuals, which will be most significant in determining the extent of effect of the HIV epidemic on fertility at the population level.

In an earlier paper we identified a large number of potential mechanisms through which an HIV epidemic could come to influence birth rates at the population level through the proximate determinants (Table 2). On the basis of the very limited empirical evidence available at the time, it appeared that HIV epidemics were most likely to have a depressing effect on fertility, at least in the short to medium term. Increasing use of contraception, shorter periods within childbearing unions and reductions in fecundity seemed most likely to outweigh reductions in breastfeeding and postpartum abstinence (Gregson 1994). However, for any given population, the effect on fertility and the significance of different pathways would depend on socio-economic factors and the current stage of fertility transition within that population.

Table 2
Hypothesized mechanisms for interaction between the HIV-1 and AIDS epidemics and the proximate determinants of fertility in sub-Saharan African settings

Proximate determinants of fertility	Possible mechanism for interaction	Hypothesized effect on fertility
Marriage: exposure to sexual relations		
	Delayed onset of sexual relations	-ve
	Reduction in premarital sexual relations	-ve
	Delayed marriage-possibly resulting in increased non-marriage	-ve
	Reduced polygyny	+ve
	Increased divorce	-ve
	Increased widowhood	-ve
	Reduced remarriage	-ve
Contraception and abortion		
	Reduced desired family size due to fear of passing on infection or leaving orphans	-ve
	Increased desired family size to ensure survival of preferred minimum and increasing 'replacement effect' due to HIV-1 related child mortality	+ve
	Increased condom use to protect against HIV-1/STDs	-ve
	Switching of family planning method from pill to condom	+ve
	Increased abortion to avoid bearing an infected child or potential orphan	-ve
Breastfeeding and postpartum abstinence		
	Reduction in breastfeeding due to concerns re vertical transmission of HIV-1 infection	+ve
	Reduction in postpartum abstinence to avoid regular partners having other relationships and contracting HIV-1 infection	+ve
	Reductions in breastfeeding and postpartum abstinence due to increase in infant mortality (natural 'replacement effect')	+ve
Pathological sterility		
	HIV-1 induced sterility	-ve
	Reduction in existing STDs due to greater condom use, lower partner change rates and increased access and resort to treatment	+ve
Natural fecundity		
	Increase in spontaneous abortions and stillbirths	-ve
	Reduced coital frequency due to increased morbidity	-ve
	Reduction in spousal separation due to HIV-1 control interventions by employers	+ve
Reduced nutrition, deteriorating health and decreased spermatozoa		-ve

Adapted from Gregson (1994)

HIV and fertility change in rural Zimbabwe

Manicaland study of HIV and fertility

An epidemiological and demographic study was conducted in two rural areas, the Honde and Rusitu valleys, of Manicaland, the eastern province of Zimbabwe, between 1993 and 1995. The objectives of the study were to establish and describe the extent and demographic impact of the HIV epidemic in rural areas and to improve understanding of factors affecting the spread of the epidemic into and within rural populations. Concerning demographic impact, there was particular interest in investigating possible effects of the HIV epidemic on the proximate determinants of fertility and a number of results were obtained which may provide pointers to the nature of these effects.

The principal survey, carried out between November 1993 and June 1994, comprised an initial *de jure* household census in the growth points (small commercial centres) at Hauna and Dzingire and the immediately surrounding areas in the Honde and Rusitu valleys, followed by detailed interviews with women of childbearing age, 13-49 years, on fertility and HIV-related topics. Four hundred and thirty-one households were enumerated in the Honde Valley and 498 in the Rusitu Valley. Coverage was estimated at over 95 per cent in both locations. In Honde 593 women (95% of those listed as eligible in the household questionnaires) and in Rusitu 644 women (97%) were interviewed in depth.

The household questionnaire covered basic socio-demographic details such as age, sex, education level, for each person enumerated, and background socio-economic indicators for the household. Individual questionnaires were modelled on forms used in Demographic and Health Surveys (IRD/Macro International Inc. 1990), subject to the substitution of more detailed knowledge, attitudes, beliefs and practices (KABP) questions on HIV and AIDS for the usual section on infant and child health (Gregson et al. 1996). The individual questionnaires included standard DHS birth history schedules, from which fertility and infant mortality indices were calculated for recent periods.

At the same time that the population-based survey was being conducted, HIV surveillance was undertaken at antenatal clinics in each of the study areas. For ethical reasons, it was not possible to link these data to the main survey data, but some basic details on socio-demographic characteristics—age, education, marital status and parity—were obtained for each participant. Inability to link HIV results with data from the population-based survey was a disadvantage but not a fundamental one, since our principal aim was to investigate the impact of the HIV epidemic on the demography of the population as a whole.

A follow-up survey of all households containing children in 1994 (745/929) was conducted between March and April 1995. In this survey, information was obtained on orphanhood, together with details of the survivorship status of individuals resident in each household in 1994. This information was used to estimate contemporary and past levels of adult mortality.

Parallel with these surveys, a small number of in-depth studies were carried out using key-informant interviews, focus-group discussions, personal observation and other ethnographic techniques. Findings from these studies were used in the design of the survey questionnaires and in interpretation of the statistical results. Finally, data were extracted from secondary sources, principally vital registration records, national and provincial Census reports, and Demographic and Health Survey reports, so that the highly localized results could be placed in a broader context.

Extent and mortality effect of the HIV epidemic in rural Zimbabwe

Full details of all methods used in data collection and analysis and detailed accounts of the results on HIV prevalence and mortality change are available elsewhere (Gregson et al. 1996; Gregson, Anderson et al. 1997). However, given that our interest here is in examining

fertility-related behaviour changes which may have resulted from the HIV epidemic, and that the extent of the epidemic and its effect on morbidity may be important in influencing the timing and nature of these changes, these changes are briefly summarized.

HIV prevalence levels among pregnant women were 24.1 per cent in the Honde Valley and 14.2 per cent in the Rusitu Valley. Among these women, those in their twenties and divorcees and single women were particularly likely to be infected. It is questionable how reliable these estimates are of levels in the general population (Gregson et al. 1995). However, given subfecundity among women with HIV infection of the order of that recorded in Uganda (Serwadda et al. 1997), the net bias due to selection effects would be modest. Mortality has increased in both study areas over the last five years, with the HIV epidemic being the most likely cause. The increases are most pronounced in the Honde Valley, where HIV levels are higher, and among men in the principal sexually-active age groups. District registry records indicate that most of the excess mortality is attributable to HIV-associated diseases. There was also evidence of a recent rise in orphanhood in the study populations (Gregson, Anderson et al. 1997).

Recent trends in fertility and its determinants

Survey results indicate that birth rates have been declining in rural Manicaland during the late 1980s and early 1990s (Figure 4). Total fertility (TFR) up to exact age 45 years declined from 5.84 live births per woman in 1984-89 to 4.98 in 1989-94 in the Honde Valley. The equivalent figures for the Rusitu Valley are 6.51 and 5.15. In each area, birth rates were lower during the most recent period at all ages. Fertility declines were seen in more and less urban locations and across women of different education levels and religious groups (Table 3). These findings are in broad agreement with recent national-level results for rural areas of Zimbabwe (Parirenyatwa 1995).

While there are indications that HIV and AIDS will become more influential in the future and may serve to intensify existing downward pressures on fertility, there appears to have been relatively little impact on birth rates in rural areas to date. This is plausible in that the epidemic seems to have spread into these areas very recently (since the late 1980s) and has only begun to affect morbidity and mortality in a noticeable way in the last two to three years. More important factors underpinning the recent decline in birth rates in rural Zimbabwe appear to have included increased female education, improvements in awareness of family planning methods following national media-based campaigns, and increased availability of modern contraceptives through local health clinics and community-based distributors. Religion may act as an obstacle to the use of effective contraception in some localities.

Table 3
Total fertility rate (ages 15-44) by period, site, religion, education, and proximity to growth point

Control Variable	Honde Valley			Rusitu Valley		
	1984-89	1989-94	Women in 1994	1984-89	1989-94	Women in 1994
All women	5.84	4.98	540	6.51	5.15	573
Religion						
Apostolic and Zionist	8.08	7.01	165	7.41	5.24	221
Other churches	4.96	3.95	375	6.19	4.96	352
Education						
Primary or less	6.50	5.56	269	7.00	5.65	305
Secondary or higher	5.23	3.67	241	4.56	3.92	238
Growth point						
Inside	4.7	4.0	198	5.81	4.16	64
Outside	6.2	5.2	307	6.61	5.20	472

In a Poisson regression analysis, female education ($p = 0.002$) and religious affiliation ($p = 0.014$) were found to be associated with differentials in numbers of births in the last five years, among women currently aged 15-49 years. Regular access to newspapers and radio showed non-significant negative influences in the analysis. More educated women were most likely to have started to reduce their fertility earliest and continue to have lower birth rates. The overall decline in fertility reflects a substantial increase in the proportion of women experiencing secondary education since Independence in 1980, as well as a diffusion effect to less educated women. In both study areas, only one in ten of the women aged over 35 years had received any secondary education, as compared to two-thirds of those aged under 25 years. The higher birth rates among women from Apostolic and Zionist churches reflect disapproval by leaders of some of these churches of the use of medicines, including contraceptives (Gregson, Zhuwau et al. 1997).

There was no significant relationship between HIV-related variables and birth rates in the five years preceding the survey. However, there were statistical associations with some of the proximate determinants of fertility. The HIV variables used and the nature of the associations recorded are reviewed in the following two sections.

Indices of knowledge, experience and risk of HIV and AIDS

Indices were constructed from survey data to reflect respondents' knowledge (KNOWLEDGE), personal experience (EXPERIENCE) of HIV and AIDS, and sense of personal risk of infection (RISK). These indices were tested for possible associations with the proximate determinants of fertility, controlling for other factors typically associated with these determinants. Such associations would provide *prima facie* evidence for emerging changes in behaviour which would affect fertility; evidence which, while indirect, would not be biased by any tendency to exaggerate behaviour changes made in response to AIDS.

Figure 4
General fertility rate (GFR) for women aged 13-39 years, by study site, 1972-94: 3 year averages

The KNOWLEDGE variable was constructed as an index (range 0.00-1.00) using responses to questions on risk factors and modes of HIV transmission, symptoms of AIDS, time taken for AIDS to develop and the existence of a cure for AIDS. For the questions on modes of transmission, women were asked first to say how they thought HIV could be transmitted. Where a possible mode of transmission (including a number of false modes) was not mentioned spontaneously, the woman was asked whether she thought HIV could be transmitted in this way. If she said 'yes' or 'no' this was recorded as a prompted response. Otherwise her response was recorded as 'don't know'. Many of the women may have made a guess when prompted, so that greater weight in the index was given to 'spontaneous-yes' and 'prompted-no' responses.

The median score on the KNOWLEDGE index was 0.43. In a multivariate logistic regression analysis, greater knowledge about HIV and AIDS was associated with increased education ($p < 0.001$), regular newspaper reading ($p < 0.001$), radio and television exposure ($p = 0.002$), and travel to urban areas ($p < 0.001$). For women in a stable union, partner's travel to urban areas was also associated with greater knowledge ($p = 0.015$). Members of Apostolic churches generally had poorer knowledge ($p < 0.001$).

The EXPERIENCE variable was also constructed as an index (range 0.00-1.00), this time using responses to questions on knowledge of persons with AIDS or having died from AIDS. Greater weight was given within the index to knowledge of household members or close family members who had AIDS. A score was also given when the respondent said she was caring for an orphan whose last parent died of AIDS (0.20) or from an illness with HIV-related symptoms (0.05-0.10). Higher scores were recorded for women who live in more urban locations ($p = 0.040$) and who have regular access to radio or television ($p = 0.035$).

In the questions on personal risk perception, respondents were asked initially whether they felt in danger of becoming infected with HIV themselves. Women who indicated that they did feel in danger were asked to state the principal reason, as this was considered to be a crucial factor in determining any possible behaviour response. Precoded response options given on the questionnaire were: (1) respondent has had multiple sex partners; (2) husband has had multiple sex partners; (3) friends and relatives dying of AIDS; and (4) other reasons. The RISK variable was set to unity, from a default value of zero, where a positive response was received for the initial question and response (3) was given to the subsidiary question. Two per cent of women felt in danger of HIV infection because they had multiple sex partners, and 19 per cent because their regular partners had multiple sex partners. Fifty-seven per cent said they did not feel in danger and ten per cent said they felt in danger because friends and relatives were dying (RISK).

The RISK variable was defined in this way so as to distinguish the effects on the behavioural proximate determinants of fertility, of general awareness of the risk of HIV infection from the effects of a sense of danger due to the respondents' own personal circumstances: the question is whether fewer people in the population, as a whole, are practising activities which carry a high risk of HIV transmission because of increasing awareness, rather than whether people who feel in danger of infection are those who practise high-risk activities. As the level of general awareness of the effects of HIV and AIDS rises, the effect of associations between greater awareness and lower levels of high-risk activity at the population level might be expected to grow. To the extent that these new patterns of behaviour affect the proximate determinants of fertility, it would also be expected that birth rates would be affected.

In a logistic regression analysis, women who are not currently married (*p < 0.001*), women with regular access to radio or television (*p = 0.015)*, and women with recent contact with medical services *(p = 0.014)* all tended to be more conscious of the risks of HIV and AIDS. After controlling for these other variables, better knowledge - KNOWLEDGE - (*p = 0.076*) and closer personal experience of AIDS - EXPERIENCE - (*p = 0.043*) were both weakly associated with greater awareness of the risks.

Associations between HIV variables and the proximate determinants of fertility

Associations in the survey data between HIV and AIDS-related variables and attitudes and practices affecting the proximate determinants of fertility are summarized in Table 4. In each case, controls were introduced for age, education, religion and regular newspaper reading, other factors associated with differentials in the outcome variables. In most cases, the RISK variable showed the strongest associations.

Table 4
Associations between increased knowledge and enhanced sense of personal risk of HIV-1 infection and attitudes and practices in regard to fertility, among women aged 15-49 years, Manicaland, Zimbabwe, 1994

Fertility attitudes and practices	n	KNOWLEDGE		RISK	
		OR	p[a]	OR	p[a]
Attitudes					
Desired family size under 4	1054	3.2	0.088	1.8	0.031
Preference for delay in timing of next birth after an infant death	1099	2.3	0.096	-	NS
Practices					
Experienced first sexual intercourse	447	-	NS	0.2	0.005
Experienced first marriage	437	-	NS	0.3	0.005
Remarriage following divorce in the last five years	64	-	NS	4.8	0.046
Condom use for family planning	315	23.4	0.034	-	NS
Breastfeeding: did not breastfeed last or previous birth	563	-	NS	4.4	0.015
Postpartum abstinence period curtailed	429	-	NS	3.5	0.014

[a]Multivariate logistic regression analyses controlling for age, education, religion and regular newspaper reading. Women aged 15-22 years and 16-23 years, respectively, were included in the tests on experience of first intercourse and first union (cohabiting or over 6 months). Interactions between the effect of the KNOWLEGE and RISK variables were tested but proved non-significant in each case. Personal EXPERIENCE of AIDS was positively associated with condom use (p=0.031) but not with any of the other fertility determinants. NS indicates results which were not significant at the p<0.1 level. Results where p>0.05 are provided for information, but are not regarded as being statistically significant.

Sexual unions

Marriage, broadly defined to include cohabiting and long-term sexual unions (6 months or more), is a universal experience for women in both study areas. Only one woman over 35 years of age was recorded as never having married. Survey results suggest that the recent trend has been towards later entry into a union: 53 per cent of women currently in their twenties had entered a union by age 20 years compared to 61 per cent of women now aged over 30 years ($p < 0.025$). Reductions in the frequency of marriage due to concerns about AIDS have been reported by respondents in focus-group studies in Uganda (Mukiza-Gapere and Ntozi 1996). In the current case, higher levels of female education appear to have been the principal factor but results from a logistic regression analysis suggest that the HIV RISK variable has a strong independent effect. This effect was strongest in less educated women. Very similar results were obtained for age at first sexual intercourse.

Five per cent of women aged 13-49 years were divorced or separated from their spouses at the time of the survey. In Rusitu Valley, this figure rises to ten per cent among women in their twenties and thirties. The mean age at divorce was 26.1 years in Honde and 27.6 years in Rusitu. Six per cent of women in Honde and 11 per cent in Rusitu reported a divorce or separation in the five years preceding the survey. The lower rate in Honde Valley reflects the greater representation of a particular Apostolic church which prohibits divorce.

Women who experienced a divorce were more likely to report a sense of personal risk of infection than other women ($p < 0.001$). Divorcees are also more likely to become involved in prostitution, so it is difficult to tell whether this association results from experiences before or after divorce. However, it is interesting that divorcees who felt at 'RISK' were more likely to have remarried (Table 4). Older and more educated women were less likely to have remarried. Overall, fewer than 25 per cent of divorcees remarried within three years, a similar rate of remarriage to that recorded for widows. Risk of HIV transmission was the most common (unprompted) reason given for why remarriage after divorce might be difficult. However, most women thought there would be no problem and the absolute proportions are relatively small: nine per cent of all women; five per cent of divorcees. Eleven per cent of respondents thought remarriage after widowhood would be inadvisable because of the HIV risk compared to 45 per cent who said it would be inadvisable because of concerns about their children. Concerns about the HIV risk were higher among single women (19%) who are typically younger and more educated, and have no children.

Fertility preferences and use of contraception

The mean numbers of children wanted in a lifetime, reported by women in the Honde and Rusitu valleys, were 4.43 and 5.15, respectively. In both areas the modal value was four. More educated women generally wanted fewer children, while older women and Apostolic women tended to have higher desired family sizes. When asked directly, half of the women interviewed said they now wanted to have fewer children because of AIDS and almost as many (46%) said they would now prefer to have their next child later. Very few (3%) reported an increase in desired family size, despite the widespread belief that infant survival chances have been reduced by AIDS. While these responses may reflect the pre-AIDS trend towards wanting fewer children and the feeling that 'times are hard', which have been increased by the 1992 drought and the Economic Structural Adjustment Programme, the admittedly weak associations between the HIV KNOWLEDGE and RISK variables, and lower desired family sizes and preference for delay in the timing of the next birth after an infant death (Table 4), lend a degree of credence to the possibility of an additional AIDS effect.

The lack of evidence for a conscious replacement effect was unexpected but is supported by other aspects of the data. Only 13 per cent of women said they would want to have the next birth sooner if an infant or child died. Women who had experienced a miscarriage, abortion or stillbirth in the last five years were twice as likely to be using a modern method of contraception as other women ($p = 0.025$). A similar result was obtained in a study in the Gambia, where women said they wished to delay the next pregnancy to allow their bodies to recover from whatever illness had caused the death of the child (Bledsoe 1995). In focus groups in the Honde and Rusitu valleys, participants stated that infant and child deaths could be caused by angry spirits. They believed that a subsequent child might also die if a reasonable time period was not allowed for the spirit to be appeased. The thought that the child could have died of AIDS and that subsequent children would suffer the same fate could be another, possibly related, explanation for preference for a delay in the current context.

Most respondents (83%) said women with HIV should stop childbearing because of the risks of vertical transmission and the adverse consequences of orphanhood. Only one per cent said women should accelerate their childbearing if they discovered they were infected. However, in a real-life situation, where traditional religion holds that dying without leaving successors bars acceptance as an ancestral spirit, women without children may still be expected to attempt to accelerate childbearing.

Use of modern methods of contraception is now common in Zimbabwe, even in rural areas (Parirenyatwa 1995). Over half of non-Apostolics interviewed in the study had used a modern method at some time in their lives. Almost a third were currently using a modern method. In younger cohorts, the figures are even higher: for example, 85 per cent of women aged 25-29 years in Rusitu reported ever-use and 54 per cent reported current use. The contraceptive pill is the most commonly used method (37% ever-use) but there is some evidence that condoms are becoming more popular. Demographic and Health Surveys in Zimbabwe have recorded an increase in ever-use of condoms from 13 per cent to 21 per cent between 1988 and 1994 (Mandishona 1989; Parirenyatwa 1995). In our own survey, 21 per cent of women reported lifetime use for family planning purposes and four per cent reported current use. Women who were better informed about HIV and AIDS (KNOWLEDGE) and who were currently using a modern method were more likely to have chosen condoms as their method of contraception (Table 4). In the same logistic regression analysis, closer personal experience of AIDS (EXPERIENCE) was also positively associated with choice of condoms for family planning ($p = 0.031$). A further three per cent of respondents reported current use of condoms in the family planning section of the interview as protection against HIV or other STDs. Forty-three women (6%) who currently had a sexual partner reported a change of contraceptive method since hearing about AIDS. Of these women 41 were using condoms after the change—15 in combination with the contraceptive pill—compared to five before.

Breastfeeding and postpartum abstinence

Breastfeeding and postpartum abstinence are both widely practised in Zimbabwe. Median periods of breastfeeding following penultimate births occurring within the five years before the survey extended to 19 months in each study area. The median period of abstinence was six months. In each location, younger women and more educated women tended to breastfeed and abstain for shorter periods.

Seventy-one women (6%) stated without prompting that vertical transmission of HIV infection could be a problem with breastfeeding. A similar proportion mentioned the concern that breastmilk might be bad for the health of the child because of contamination following sexual intercourse. Women who mentioned the risk of HIV transmission were more likely to have avoided breastfeeding a child born in the previous five years (Table 4).

The principal reasons given for postpartum abstinence were maternal health (26%), child health (33%) and temporary absence of partner (20%). Women who had experienced a birth in the previous five years were asked how long they had abstained after the most recent birth and then what they considered to be the ideal abstinence period. Thirty per cent (134/439) of these women reported recommencing sexual activity before the ideal time interval had passed; 49 of these women (9%) said without prompting that they had done this to avoid their husbands having other partners.

Given the high level of tolerance of men's extramarital partners in the pre-AIDS era, it may be supposed that fear of HIV infection was a common concern here. On the other hand, in an environment where polygyny is practised and tensions between wives are common, some women may have feared that their husbands could take additional wives. Tests were therefore carried out to see whether women who reported a sense of risk of HIV infection were more likely to resume sexual relations earlier than they would ideally have wanted. Separate tests were conducted for all most recent births and for births where sex had been resumed. The possible effect of perceived risk of infection was tested for all instances where sex had been resumed early and for those where this was said to have been done because of the risk of the husband having other partners. Even when the restriction was applied, women who felt at risk of HIV infection (RISK) were more likely to have resumed sex early ($p = 0.038$).

Finally, the reality of women's concerns regarding their husbands' infidelity should be stressed. In focus-group discussions with men in the Rusitu Valley, abstinence on the part of their wives was one of the two reasons cited for their need to have casual affairs. In many cases, these affairs were with prostitutes contacted while drinking at beer halls.

Pathological sterility and natural fecundity

Sexually transmitted diseases (STDs) such as *Neisseria Gonorrhoea* and *Chlamydia Trachomatis*, which are common in sub-Saharan countries, including Zimbabwe, can result in pelvic inflammatory disease and lead to primary or secondary infertility (Frank 1983; Cates, Rolfs and Aral 1993). In 1990, more than one million STD infections were reported as treated at public health facilities in Zimbabwe. High incidence rates have been reported in rural as well as urban areas (Le Bacq et al. 1993). Many additional cases go unrecognized and untreated owing to the absence of visible symptoms. However, many of the reported cases are repeat cases. In Manicaland province, the reported case rate among men and women aged 15-59 years is almost 15 per cent per annum (National AIDS Co-ordination Programme 1994). Urethral discharges and genital ulcers are both common. In the study areas, 11 per cent of women reported that they or their partners had attended an STD clinic at some point in their lives. The figure was low in both areas, possibly reflecting under-reporting due to the stigma of these diseases, but especially so in the Honde Valley (9% as against 13% in Rusitu), where Apostolics are unlikely to attend clinics for treatment and may, in any event, be less affected by STDs because of their more restrictive behaviour code (Gregson, Zhuwau et al. 1997).

Of the 421 women in the survey aged 30 years or over, only five reported not having had any children (1%). This finding is consistent with results from other studies in sub-Saharan Africa, including Zimbabwe, which have shown low levels of primary sterility, even in the presence of STDs (Mandishona 1989; Larsen 1994). An analysis of the prevalence of secondary sterility has not been attempted in the current study. Nationally, it has been estimated that age-specific sterility rates may be substantial (Larsen 1994). Not all of this sterility is due to STDs, of course: at later ages natural infecundability becomes an increasingly important factor (Knodel and Wilson 1981). Nonetheless, earlier and more effective treatment of STDs, on a widespread basis, introduced as part of the drive to counter

the spread of HIV, clearly has the potential to increase fecundity levels significantly. HIV prevention programs have achieved substantial reductions in STD cases in some locations in Zimbabwe (Wilson et al. 1994), but, to date, these programs have generally been limited to urban areas.

HIV infection has been found to be associated with adverse pregnancy outcome in a number of studies (Johnson et al. 1986; Ndinya-Achola et al. 1990; Miotti et al. 1991). In the current survey, 12 per cent of women aged 15-49 years reported ever having had an abortion or stillbirth. Women who reported ever visiting an STD clinic or who said they felt in danger of HIV infection due to multiple partners—taken here as risk factors for HIV infection—were more likely to report a pregnancy loss of this kind (OR: 2.68, $p = 0.024$, n = 743).

The HIV-related variables were tested in a model of the socio-demographic determinants of coital frequency but there was no evidence of an association. The principal hypothesis here was that increased morbidity would reduce coital frequency among persons infected with HIV. However, HIV-related morbidity remains low in the study populations, in absolute terms, and it was not possible to analyse the data by current serostatus directly.

Discussion and conclusions

In this article we have shown that the demographic effects of HIV epidemics in sub-Saharan African populations can be extensive and complicated. Contemporary fertility trends can have a major influence on the nature of many of these effects, through the complexities of population dynamics. Furthermore, attitudes and practices surrounding reproduction can influence the pattern of risk-behaviour for HIV transmission and the nature of the behaviour changes most likely to be adopted. Equally, behaviour, and indeed biological, changes associated with HIV prevention can result in shifts in the proximate determinants of fertility. We have shown that, in theory at least, there are numerous pathways through which such shifts can arise. These can include changes which affect birth rates deliberately and in unwanted or otherwise unintended ways. In many instances, uninfected individuals are as likely to modify behaviour as infected people.

In a case study in rural Zimbabwe we found associations between HIV variables and aspects of the proximate determinants of fertility which provide tentative *prima facie* evidence for causal linkages. These associations are consistent with findings from a recent study in Uganda, where delayed sexual activity and marriage and increased condom use, particularly in casual relationships, were recorded in areas of high but declining HIV prevalence (Asiimwe-Okiror et al. n.d.). However, firm conclusions cannot be drawn in the current case, in view of the methodological problems in deriving results on behaviour change from cross-sectional survey data using HIV indicators of the form used here. For example, women with greater knowledge and awareness of HIV could be more modern or 'progressive' in outlook and thus more likely to marry later, bottlefeed rather than breastfeed and so on. This possibility has been addressed in the analyses by introducing controls for age, education, religion and newspaper reading, but some residual effect may remain. A further problem is that we have had to use current status indicators for knowledge, experience and awareness of risk for levels in the recent past. In mitigation, the retrospective periods of study are relatively short, five years or less in most cases, while public awareness campaigns have been under way in Zimbabwe since 1987. Furthermore, recent changes would generally be expected to make associations with the proximate determinants of fertility *more* difficult to detect. A strength of the approach adopted is that the outcome fertility-related variables are based on information given in interviews before the subject of AIDS was raised. This avoids a common problem in KABP studies, whereby questions on behaviour are linked in respondents' minds to information received on steps they should take to prevent HIV infection.

The surveys carried out in the current study are small-scale, reporting bias may affect some of the results and the underlying social processes are known to be complex. While HIV prevalence has reached high levels in women at antenatal clinics, the cumulative effect of the epidemic on morbidity and mortality has been modest so far. Personal experience and awareness of the risks of infection are likely to be heightened and become more widespread as the AIDS epidemic develops. Gaps in knowledge will be filled. The changes in attitudes and behaviour observed here may therefore become more common but there may also be modifications as the epidemic intensifies. At the same time, other fertility determinants, notably education and religion, will remain influential. Over the next 10-15 years, increasing education levels among older women will depress birth rates.

The ultimate extent and net effect of changes in the proximate determinants on fertility can only really be guessed. Compensating adjustments might be expected, particularly in view of the existing high levels of contraceptive use in Zimbabwe. Given the natural desire to have children—reinforced in Shona culture by the ancestral spirit belief system—but typically moderate contemporary desired family sizes and birth rates, any deferral of initial sexual activity may result in delayed marriage and later commencement of childbearing, but have little net effect on total fertility.

Husbands appear to be infected earlier in the epidemic than wives in most cases (Gregson, Anderson et al. 1997). Significant increases in the incidence of widowhood therefore seem inevitable. However, many of the women who experience widowhood may not live very long after the deaths of their husbands. The effect on divorce is not yet clear. In both cases, remarriage is currently relatively unusual. Widows appear even less likely to remarry in the AIDS era. Divorcees may be more likely to remarry. If this turns out to be the case, the net effect on time spent outside stable unions—where birth rates were recorded as being lower—would be relatively small. Total fertility would be little affected, although increased proportions of women would not survive the full potential childbearing lifespan.

Contraceptive use is already increasing. This trend may be accelerated by more open discussion of contraception in public and in private, because of AIDS education programs. To the extent that couples come to suspect or be told that they are infected with HIV, contraceptive use to prevent further births may become more common. This effect may be seen more widely if it is true that the HIV epidemic is causing couples to want fewer children. Significant reductions in contraceptive use due to couples seeking to 'insure' against increased infant and child mortality or 'replace' children who died appear to be unlikely. Nonetheless it is hard to believe that women or couples with no children will not seek to ensure that they have some successors, particularly when it becomes more widely known that there is perhaps a two-in-three chance that the child of an infected mother will not itself be infected. Any natural replacement effect, due to increased early childhood mortality, will be modest in populations where contraceptive use is common.

Similarly, women who breastfeed or abstain for shorter periods after giving birth may be expected to use contraception to delay the next birth. Few women in the current study relied on the contraceptive effect of breastfeeding and a number of women were breastfeeding and using modern family planning methods simultaneously. If the wider health benefits of breastfeeding are stressed in future HIV control and family planning programs in line with current World Health Organization (1992) guidance it may be possible to forestall further reductions. However, this is an issue that will require careful and sensitive handling. Further declines in postpartum abstinence will be relatively unimportant in relation to fertility in Zimbabwe, as periods are already short and contraception is frequently adopted on resumption of sexual activity.

Increases in fecundity could occur where better treatment of STDs is instituted. However, where STD control is achieved through increases in condom, and thus contraceptive, use,

there could be little net effect on birth rates. On the contrary, some reductions in fecundity are most likely, because of reduced coital frequency among sick individuals and the biological effects of HIV infection.

The above is essentially informed speculation. If the interpretation suggested is true, the most plausible outcome seems to be a modest acceleration in the pace of fertility decline in rural Manicaland. This could result from delayed marriage and childbearing, further increases in contraceptive use and reduced fecundity among those with HIV infection. There may also be an increased concentration of births into the age-range 25-34 years: a slight upward shift in the peak age at childbearing. In an extended epidemic, this would result in more women dying before completing their intended childbearing and a further downward pressure on the crude birth rate and population growth. This prognosis is largely society-specific. Similar changes could be seen elsewhere but the net effect may differ substantially. For example, populations where contraceptive availability and acceptance are low could experience increases in fertility, if breastfeeding and abstinence periods were to be eroded and behaviour changes led to reductions in STDs. This would be especially likely in populations where there is a strong conscious replacement effect.

Further studies are required to confirm or correct these findings and to assess the effect of HIV on fertility at different stages of epidemics, in different cultural settings, and in populations at different stages of the fertility transition. Prospective comparative studies which monitor not only changes in birth rates among infected and uninfected individuals but also changes in the proximate determinants of fertility within each group may prove most fruitful. Where such studies are attempted, they should incorporate effective controls for possible confounding factors, such as the presence of other STDs and socio-demographic risk-factors for HIV infection and fertility change.

References

Anderson, R.M., S. Gupta and W. Ng. 1990. The significance of sexual partner contact networks for the transmission dynamics of HIV. *Journal of Acquired Immune Deficiency Syndromes* 3: 417-429.

Anderson, R.M., R.M. May and A.R. McLean. 1988. Possible demographic consequences of AIDS in developing countries. *Nature* 332: 228-234.

Ankrah, M. 1991. AIDS and the social side of health. *Social Science and Medicine* 32: 967-980.

Asiimwe-Okiror, G., A.A. Opio, J. Musinguzi, G. Tembo and M. Carael. n.d. Change in sexual behaviour and decline in HIV infection among young pregnant women in urban Uganda. Submitted for publication.

Batter, V., B. Matela, M. Nsuami, et al. 1994. High HIV-1 incidence in young women masked by stable overall seroprevalence among childbearing women in Kinshasa, Zaire: estimating incidence from serial seroprevalence data. *AIDS* 8: 811-817.

Blair Research Institute and Oxford University. 1996. *The Early Socio-Demographic Impact of the HIV Epidemic in Rural Zimbabwe*. Harare.

Bledsoe, C. 1995. Numerators and denominators in the study of high fertility populations: past and potential contributions from cultural anthropology. Paper presented at Seminar in Honour of John C. Caldwell, Australian National University, August.

Boerma, J.T., M. Urassa and R. Isingo. 1996. Female infertility and its association with sexual behaviour, STD and HIV infection in Tanzania. Working Paper No. 14. Mwanza: TANESA.

Bongaarts, J., O. Frank and R. Lesthaeghe. 1984. The proximate determinants of fertility in sub-Saharan Africa. *Population and Development Review* 10: 511-537.

Bongaarts, J. and R.G. Potter. 1983. *Fertility, Biology and Behaviour, Studies in Population.* New York: Academic Press.

Cates, W., R.T. Rolfs and S.O. Aral. 1993. The pathophysiology and epidemiology of sexually transmitted diseases in relation to pelvic inflammatory disease and infertility. Pp.101-125 in *Biomedical and Demographic Determinants of Reproduction,* ed. R. Gray, H. Leridon and A. Spira. Oxford: Clarendon Press.

Davis, K. and J. Blake. 1956. Social structure and fertility: an analytical framework. *Economic Development and Cultural Change* 4: 211-235.

De Bruyn, M. 1992. Women and AIDS in developing countries. *Social Science and Medicine* 34: 249-262.

De Cock, K.M., B. Barrere, L. Diaby, et al. 1990. AIDS - the leading cause of adult death in the West African city of Abidjan, Ivory Coast. *Science* 249: 793-796.

Frank, O. 1983. Infertility in sub-Saharan Africa: estimates and implications. *Population and Development Review* 9: 137-144.

Garenne, M., M. Madison, D. Tarantola, B. Zanou, J. Ada and R. Dogore. 1995. *Conséquences Démographiques du SIDA en Abidjan, 1986-1992*. Vol. 10. Paris: Centre Français sur la Population et le Développement.

Garnett, G.P. and R.M. Anderson. 1993. Factors controlling the spread of HIV in heterosexual communities in developing countries: patterns of mixing between different age and sexual activity classes. *Philosophical Transactions of the Royal Society of London* Series B, 342: 137-159.

Garnett, G.P. and R.M. Anderson. 1994. Balancing sexual partnerships in an age and activity stratified model of HIV transmission in heterosexual populations. *IMA Journal of Mathematics Applied in Medicine and Biology* 11: 161-192.

Gregson, S. 1994. Will HIV become a major determinant of fertility in sub-Saharan Africa? *Journal of Development Studies* 30: 650-679.

Gregson, S., R.M. Anderson, J. Ndlovu, T. Zhuwau and S.K. Chandiwana. 1997. Recent upturn in mortality in rural Zimbabwe: evidence for an early demographic impact of HIV infections? *AIDS* (in press).

Gregson, S., G.P. Garnett and R.M. Anderson. 1994. Assessing the potential impact of the HIV-1 epidemic on orphanhood and the demographic structure of populations in sub-Saharan Africa. *Population Studies* 48: 435-458.

Gregson, S., G.P. Garnett, R. Shakespeare, G. Foster and R.M. Anderson. 1994. Determinants of the demographic impact of HIV-1 in sub-Saharan Africa: the effect of a shorter mean adult incubation period on trends in orphanhood. Pp.65-92 in *AIDS Impact and Prevention in the Developing World: Demographic and Social Science Perspectives,* ed. J. Cleland and P. Way. Supplement to *Health Transition Review* 4. Canberra: Australian National University.

Gregson, S., T. Zhuwau, R.M. Anderson and S.K. Chandiwana. 1996. The early socio-demographic impact of the HIV-1 epidemic in rural Zimbabwe. Harare: Blair Research Institute.

Gregson, S., T. Zhuwau, R.M. Anderson and S.K. Chandiwana. 1997. Apostles and Zionists: the influence of religion on demographic change in rural Zimbabwe. *Population Studies* (forthcoming).

Gregson, S., T. Zhuwau, R.M. Anderson, T. Chimbadzwa and S.K. Chandiwana. 1995. Age and religion selection biases in HIV prevalence data from antenatal clinics in Manicaland, Zimbabwe. *Central African Journal of Medicine* 41: 339-345.

IRD/Macro International Inc. 1990. *Demographic and Health Surveys - Phase II: Model 'A' Questionnaire with Commentary for High Prevalence Countries*. Columbia MD.

Jacquez, J., C. Simon, J. Koepman, L. T. Sattenspiel and T. Perry. 1988. Modeling and analyzing HIV transmission: the effect of contact patterns. *Mathematical Biosciences* 92: 119-199.

Johnson, W.D., M. Stanback, R. Verdier, et al. 1986. Fetal and postnatal mortality associated with HTLV-III infection. Paper presented to International Congress of Antimicrobial Agents and Chemotherapy, New Orleans.

Knodel, J. and C. Wilson. 1981. The secular increase in fecundity in German village populations: an analysis of reproductive histories of couples married 1750-1899. *Population Studies* 35: 53-84.

Larsen, U. 1994. Sterility in sub-Saharan Africa. *Population Studies* 48, 3: 459-474.

Le Bacq, F., P.R. Mason, L. Gwanzura, V.J. Robertson and A.S. Latif. 1993. HIV and other sexually transmitted diseases at a rural hospital in Zimbabwe. *Genitourinary Medicine* 69: 352-356.

Mandishona, G.M. 1989. *Zimbabwe Demographic and Health Survey, 1988*. Harare: Zimbabwe Central Statistical Office.

Martin, P.M.V., G. Gresenguet, V.M. Herve, G. Renom, G. Steenman and A.J. Georges. 1991. Decreased number of spermatozoa in HIV infected individuals. *AIDS* 6: 130-131.

Mason, K.O. 1994. HIV transmission and the balance of power between men and women: a global view. Pp.217-240 in *AIDS Impact and Prevention in the Developing World: Demographic and Social Science Perspectives*, ed. J. Cleland and P. Way. *Health Transition Review* 4 (Supplement). Canberra: Australian National University.

Miotti, P.G., G. Dallabetta, E. Nvodi, G. Liomba, A.J. Saah and J. Chiphangwi. 1991. HIV-1 and pregnant women: associated factors, prevalence, estimate of incidence and role of fetal wastage in Central Africa. *AIDS* 4: 733-736.

Mukiza-Gapere, J. and J.P.M. Ntozi. 1996. Impact of AIDS on marriage patterns, customs and practices in Uganda. Pp. 201-208 in *The Third World AIDS Epidemic*, ed. I. O. Orubuloye et al. *Health Transition Review* 5 (Supplement). Canberra: Australian National University.

Mulder, D.W., A.J. Nunn, A. Kamali, J. Nakiyingi, H. U. Wagner and J. F. Kengeya-Kayondo. 1994. Two year HIV-1-associated mortality in a Ugandan rural population. *Lancet* 343, i: 1021-1023.

National AIDS Co-ordination Programme. 1994. HIV and AIDS Surveillance Annual Report. Harare: Zimbabwe Ministry of Health.

Ndinya-Achola, J.O., I.A. Wamola, N. Nagelkerke, F.A. Plummer, R.C. Brunham, M. Temmerman and P. Piot. 1990. Maternal STD/HIV infection as a risk factor for spontaneous non-septic abortion and HIV infection as a risk factor for adverse pregnancy outcome. *Kenya AIDS Technical Bulletin* 1: 5.

Parirenyatwa, C.N. 1995. *Zimbabwe Demographic Health Survey, 1994*. Harare: Zimbabwe Central Statistical Office.

Ryder, R.W., V.L. Batter, M. Nsuami, N. Badi, L. Mundele, B. Matela, M. Utshudi and W. L. Heyward.1991. Fertility rates in 238 HIV-1 seropositive women in Zaire followed for 3 years post-partum. *AIDS* 5: 1521-1527.

Serwadda, D. , R. H. Gray, M. J. Wawer, et al. 1997. HIV, STDs and fertility: a population-based study in Rakai district, Uganda.

Setel, P. 1996. The effects of HIV and AIDS on fertility in east and central Africa. Pp.179-190 in *The Third World AIDS Epidemic*, ed. I. O. Orubuloye et al. *Health Transition Review* 5 (Supplement). Canberra: Australian National University.

Sewankambo, N.K., M.J. Wawer, R.H. Gray, D. Serwadda, C. Li, R.Y. Stallings, S.D. Musgrave and J. Konde-Lule. 1994. Demographic impact of HIV infection in rural Rakai District, Uganda: results of a population-based cohort study. *AIDS* 8: 1707-1713.

United Nations. 1991. *The AIDS Epidemic and its Demographic Consequences*. New York.

Widy-Wirski, R., S. Berkley, R. Downing, et al. 1988. Evaluation of the WHO clinical case definition for AIDS in Uganda. *Journal of the American Medical Association* 260: 3286-3289.

Wilson, D., B. Nyathi, N. Lamson, G. Foster and F. Dakwa. 1994. *Community Interventions in Zimbabwe: Analysis of a Multi-Site Replication.* Harare: University of Zimbabwe.

World Health Organization. 1992. Consensus statement from the WHO/UNICEF consultation on HIV transmission and breast-feeding. *Weekly Epidemiological Record* 67, 24: 177-184.

Zaba, B. and M. Collumbien. 1996. HIV and fertility: modelling the effects of changes in union dynamics. Paper presented to British Society for Population Studies, St Andrews.

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Estimates of the impact of HIV infection on fertility in a rural Ugandan population cohort*

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Abstract

Fertility rates in a population-based cohort of over 3500 women aged 15-49 years living in rural southwest Uganda are described and examined in relation to infection with HIV. Over a six-year follow-up period (1989/90 to 1995/6) the average general fertility rate was estimated as 199 births per thousand woman-years of observation (95 % confidence interval 191 to 207) with a total fertility rate of 6.2 births per woman. The overall prevalence of infection with HIV was 12 per cent and remained relatively stable during follow-up. With the exception of women aged 15-19 years, women who were not infected with HIV had higher fertility than HIV-infected women. The overall age-adjusted fertility rate in HIV-infected women was 0.74 of that of uninfected women (95% confidence interval 0.63 to 0.87, P<0.001) and this result was unaffected by additional adjustment for marital status. When combined with an overall HIV prevalence rate of 12 per cent, this corresponds to a three per cent reduction in fertility rates in the whole population. The lower fertility in HIV-positive women is unlikely to be explained by increased use of contraception, as use of modern contraceptive methods in rural Uganda is low and fewer than ten per cent of women are aware of their HIV-serostatus. More likely explanations are reduced sexual activity due to clinical symptoms associated with HIV infection or lower fertility associated with co-existing infections with other sexually transmitted diseases, such as syphilis. A reduction in fertility caused by HIV infection itself cannot be excluded. The implications of these findings for the use of antenatal clinic data to provide population estimates of HIV prevalence are discussed.

Understanding the effect of HIV[1] infection on fertility in populations in sub-Saharan Africa is vital for predicting the likely demographic impact of the epidemic and for predicting the future socio-demographic burden on communities of increased numbers of children infected by vertical transmission of HIV or subsequently orphaned by the death of their HIV-infected

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[1] HIV refers to HIV-1 throughout this paper.

mother. There have, however, been relatively few reports of fertility rates in HIV-infected and uninfected women in Africa. Lower fertility among HIV-infected women has been observed in women in Zaire, Uganda and Rwanda (Ryder et al. 1991; Allen et al. 1993; Sewankambo et al. 1994) although the studies have generally been too small to yield statistically significant results. Significantly reduced rates of pregnancy were however observed in HIV-infected women in a cross-sectional study carried out in Rakai district, Uganda (Serwadda et al. 1997). Whilst lower fertility rates have also been described in HIV-infected women in developed countries (Selwyn et al. 1989; Stephenson et al. 1996; De Vincenzi et al. 1997; Thackway et al. 1997) these are unlikely to be applicable to African populations because, unlike most women in developing countries, the women studied knew their HIV status, and modern contraceptive methods and pregnancy termination were more readily available and acceptable.

Estimates of the levels and trends in HIV prevalence and incidence among women in many African countries increasingly rely on sentinel surveillance data collected from antenatal clinics (Kigadye et al. 1993; Batter et al. 1994; Asiimwe-Okiror et al. 1995; Fylkesnes et al. 1997). HIV prevalence rates in women attending an antenatal clinic in Tanzania were found to be lower than those found in a population survey carried out in the same geographical area (Kigadye et al. 1993). This suggests that HIV-infected women may be less likely to give birth than uninfected women. Opportunities for similar comparisons investigating the validity of sentinel surveillance data of childbearing women are, however, rare. If real, the reduced fertility in HIV-infected women has important implications for monitoring the HIV epidemic in developing countries (Boisson et al. 1996). Further studies of fertility rates in relation to HIV infection are required in order to assess the likely magnitude of the potential bias in estimating HIV prevalence from surveillance data.

In this paper we describe fertility rates in a general population cohort in relation to infection with HIV. The cohort is located in Masaka district, southwest Uganda, and includes over 3500 women of childbearing age. Prevalence and incidence of HIV, and of mortality rates during 1990 to 1996, in this cohort have previously been reported (Mulder et al. 1995; Kengeya-Kayondo et al. 1996; Nunn et al. 1997). The present analysis is based on data for the first six years of follow-up, based on annual surveys conducted between 1989/90 and 1995/6.

Methods and materials

The Medical Research Council general population cohort is located in rural southwest Uganda and comprises approximately 10,000 people residing in 15 neighbouring villages in a subcounty in Masaka district. The inhabitants are mainly subsistence farmers who produce small-scale cash crops such as bananas and coffee. The population was first surveyed in 1989-90 and has since been resurveyed annually. Detailed description of the cohort study design and methods is given elsewhere (Mulder et al. 1994). Briefly, the survey procedure at each round commences with updating of detailed village maps to identify new or demolished houses since the previous round. All households are then visited by a census taker who interviews one member of each household to enumerate all *de jure* residents, including joiners and births since the previous round, and to obtain information about leavers and those who have died. Following this, the medical team aims to interview all adult members (aged 13 years and over) and obtain a blood specimen from all consenting individuals. HIV serological status is determined by two independent enzyme-linked immuno-absorbent assays (EIA) and confirmed by Western Blot where necessary.

The present fertility analyses are based on data collected from the first seven survey rounds obtained from four sources. First, at each annual round, a census was conducted in an attempt to identify all newborns (including those who have died) since the last census and

relate them to their mothers if present in the household at the time of census. Second, since the third survey round, census takers have inquired for each woman aged 15-49 years whether they gave birth in the last 12 months, and have recorded the names of any newborns reported. Third, a monthly birth and death register, which has been maintained by village registrars since the third survey round, is used to record births which might otherwise have been missed by the census team (such as babies who were born and died before census). Fourth, at the second and seventh survey rounds, additional questions were included in the questionnaire administered by the medical survey team which asked women about their reproductive histories. Using data from these four sources it was possible to individually link all but ten babies born in the study area to resident mothers. Babies were not found for five mothers residing in the study area who reported giving birth. Five babies born to women aged under 15 years were excluded from all analyses.

Statistical analysis

All resident women aged between 15 and 49 years at one or more censuses contributed to the analyses. Women resident at two consecutive censuses contributed one year, those who joined or left between censuses contributed a half-year, and those who joined and left between censuses contributed one-third of a year to the denominator (woman-years of observation) for the calculation of fertility rates. Woman-years of observation and births occurring between each pair of censuses were stratified according to five-year age groups, marital status and HIV-status as determined at the first of the two censuses. Women who changed age group, marital status or HIV status during follow-up contributed woman-years to more than one stratum. Births were assigned to the stratum corresponding to the age, marital status and HIV status of the mother for the year in which the birth occurred. For joiners, women-years of observation and births recorded before their first census were assigned to the age, marital status and HIV category as recorded at the first survey round at which they were censused. Where possible, HIV serostatus for women resident but not blood-tested at a given round was inferred from serological results obtained at earlier (if previously HIV-positive) or later (if subsequently HIV-negative) rounds.

General fertility and total fertility rates were calculated using standard techniques (Newell 1988). Confidence intervals for general and age-specific fertility rates and rate ratios for fertility in HIV-positive versus HIV-negative women were derived from the quadratic approximation to the Gaussian log likelihood (Clayton and Hills 1993). P-values for comparing rates were based on the chi-square distribution. Rate ratios adjusted for age and marital status, together with approximate 95 per cent confidence intervals and tests of statistical significance, were derived from Poisson regression models. Odds ratios for HIV-infection in women giving birth versus women not giving birth were estimated with adjustment for age using the Mantel Haenszel techniques (Hennekens and Buring 1987). All statistical analyses were performed using the STATA computer package (StatCorp 1995).

Results

The number of women aged 15-49 censused as resident in the study area at each survey round ranged between 2000 and 2250 (Table 1). Altogether, a total of 11,420 woman-years of observation were accrued during the six-year period of follow-up. Just under one-third of women (31 per cent) were aged 15-19 years (average over six years). Information on marital status was available for 98 per cent of women. On average, 31 per cent of all women were single, 52 per cent were married, 4 per cent were widowed and 11 per cent were divorced. Among women aged 15-19 years, 72 per cent were single, 25 per cent were married and 2 per

cent were divorced. The percentage of women remaining single was 28 per cent at ages 20-24, 13 per cent at ages 25-29 and 6 per cent at ages 30 years and over.

Table 1
Study population by age, marital status and year of follow-up among women aged 15-49 at census

	Year of follow-up						Total	
Age group	1	2	3	4	5	6	Number	%
15-19	612	691	702	681	676	654	4016	31
20-24	403	389	406	406	398	404	2406	18
25-29	341	365	369	332	323	276	2006	15
30-34	216	243	255	275	280	299	1568	12
35-39	171	171	165	170	169	188	1034	8
40-44	178	168	191	198	200	168	1103	8
45-49	144	156	155	150	151	151	907	7
Marital status								
Single	694	665	654	657	662	691	4023	31
Married	1110	1140	1192	1134	1142	1085	6803	52
Widowed	70	69	75	78	89	90	471	4
Divorced	140	250	310	282	263	246	1491	11
Not known	51	59	12	61	41	28	252	2
Total number	2065	2183	2243	2212	2197	2140	13040	100
Woman-years of observation	1826	1906	1942	1948	1920	1878	11420	100

HIV serostatus was determined for 78 per cent of women surveyed (average over six years). Lowest HIV coverage was obtained for women aged 15-19 years (70%) and highest among women aged 40 years or more (85%) (Table 2). HIV prevalence was highest among women aged 20-24 years (20%). Prevalence of HIV according to marital status varied according to the age of the woman: for example, among single women aged 25 and over HIV prevalence was 20 per cent compared to 5 per cent for those aged 15-24 years, whereas for married women rates for those aged 15-24 years and 25 years and over were 8 per cent and 16 per cent respectively. Prevalence rates were generally higher in women who had been widowed or divorced. The overall prevalence rate was 12 per cent and remained relatively constant throughout the period of follow-up. While there was some evidence of a decrease in HIV prevalence across survey rounds in women aged 15-24, a reverse pattern of increasing prevalence emerged for women aged 25 years and over (Table 2). Both trends were of only borderline statistical significance (P=0.04).

Table 2
HIV coverage and positivity rates by age, marital status and year of follow-up among women aged 15-49 at census (average across six years of follow-up)

	% (Number) with known serostatus		%HIV +		
	%	(Number)	All women	15-24 yrs	25-49 yrs
Age group					
15-19	70	(2816)	5		
20-24	75	(1816)	20		

25-29	81	(1625)	18		
30-34	84	(1315)	14		
35-39	80	(828)	8		
40-44	85	(938)	9		
45-49	85	(774)	6		
Marital status					
Single	70	(2798)	7	5	20
Married	81	(5525)	11	16	8
Widowed	85	(398)	26	31	25
Divorced	82	(1225)	19	24	18
Not known	66	(166)	23	24	21
Year of follow-up					
1	87	(1803)	12	13	11
2	81	(1772)	12	12	11
3	77	(1732)	12	11	13
4	74	(1645)	11	10	12
5	75	(1641)	12	10	13
6	71	(1519)	12	10	14
Total	78	(10112)	12	11	12

A total of 2274 births were recorded among women resident in the study area. The majority of births (74%) occurred to married women and 15 per cent of births were reported by single women (Table 3). Of the 353 births recorded during the sixth year of follow-up, 219 (62%) were recorded by both the census team and village registrars, 115 (33%) were reported by census alone and 19 (5%) were identified by the village registrars alone.

Table 3
Percentage distribution of births by marital status by round among women aged 15-49 at census

	Year of follow-up						Total
	1	2	3	4	5	6	%
Marital status							
Single	20	19	13	12	11	14	15
Married	74	69	77	77	73	76	74
Widowed	1	3	1	1	2	3	2
Divorced	4	6	10	7	9	6	7
Not known	1	2	0	3	4	2	2
Total (%)	100	99	101	100	99	101	100
Total (Number)	423	432	339	350	377	353	2274

General and total fertility rates are presented in Table 4 by marital status and year of follow-up. Overall, the general fertility rate was 199 per 1000 woman-years (95% confidence interval 191 to 207) and the total fertility rate was 6.2 births per woman. Highest fertility rates were observed among married women (Table 4). Fertility rates declined during the period of follow-up; the decline was statistically significant both for general fertility (X^2 for trend = 12.2, P<0.001) and age-adjusted fertility rates (X^2 for trend= 10.5, P=0.001). The decline in general fertility was particularly marked among single women where rates declined from 136 per 1000 in year 1 to 85 in year 6 (X^2 for trend= 18.0, P<0.001). For married women, the decline in fertility was less marked (X^2 for trend= 3.63, P=0.06) but for widowed, divorced and those with unknown marital status there was no evidence of a trend in rates across time (X^2 for trend= 0.01, P=0.9)

Table 4
General and total fertility rates by year of follow-up and marital status among women aged 15-49 years at census

	General fertility rate per 1000 births				Total
Year of follow-up	Single	Married	Other	All women	fertility rate
1	136.1	312.1	121.1	231.7	7.0
2	147.5	292.0	155.0	226.6	7.0
3	78.7	242.5	108.1	174.0	5.3
4	76.5	260.7	105.7	180.2	5.5
5	77.1	269.7	171.1	196.3	6.2
6	85.2	269.2	117.1	188.0	6.1
Total	100.9	274.0	116.9	199.1	6.2

Figure 1:
Age-specific fertility rates in all women aged 15-49 at census for women and according to HIV status (women with a definitive serological status only)

Figure 1 shows age-specific fertility rates in the total study population and for women with a definitive serostatus. Among HIV-negative women and all women combined, highest fertility rates were observed in women aged 20-24 years. For HIV-positive women, the highest fertility rate was observed at ages 15-19 years. In contrast with HIV-negative women, age-specific fertility rates in HIV-positive women showed a monotonic decline in rate with increasing age.

The general fertility rate was 195 per 1000 woman-years in women testing positive for HIV and 212 per 1000 woman-years in negative women (Table 5). Lower fertility rates were observed in HIV-positive women in each age group with the exception of women aged 15-19 years where fertility rates in HIV-positive women were 1.54 times those of uninfected women (95% confidence interval 1.07 to 2.21). Fertility rates in HIV-positive women were significantly below those of HIV-negative women at ages 20-24, 25-29 and 30-34 years. At ages above 34 years, rate ratios were also below 1.0 but the reduced fertility was not statistically significant: only eight births were observed among HIV-positive women in this age band. The age-adjusted fertility rate ratio for HIV-positive versus HIV-negative women was 0.74 (95% confidence interval 0.63 to 0.87). This result is very highly statistically significant ($P<0.001$).

Table 5
Age-specific fertility rates (ASFR), general fertility rates and rate ratios (RR) for fertility in HIV-positive versus HIV-negative women aged 15-49 at census

	HIV-positive			HIV-negative			RR HIV+ versus HIV-		
Age group	Births	Woman-years	ASFR/1000	Births	Woman-years	ASFR/1000	RR	95% CI	
15-19	31	111.1	279.0	418	2299.4	181.8	1.54	1.07	2.21
20-24	73	282.2	258.7	460	1310.4	351.0	0.74	0.58	0.94
25-29	54	236.6	228.2	392	1233.4	317.8	0.72	0.54	0.95
30-34	16	146.3	109.4	270	1051.7	256.7	0.43	0.26	0.71
35-39	6	49.7	120.7	128	732.8	174.7	0.69	0.31	1.57
40-44	2	74.4	26.9	59	819.3	72.0	0.37	0.09	1.53
45-49	0	33.9	0.0	4	705.7	5.7	0.00	-	-
Total	182	934.2	194.8[a]	1731	8152.7	212.3 [a]	0.74[b]	0.63	0.87

[a] General fertility rate

[b] Rate ratio in HIV-positive versus HIV-negative women adjusted for age

Fertility rate ratios adjusted for age alone, and for age and marital status, are presented according to year of follow-up in Table 6. Lower fertility in HIV-positive women was evident in all six years but was only statistically significantly below that of HIV-negative women in year 3 (P=0.02). In year 4, age-adjusted rates in the two groups were similar (RR=0.93) but diverged again during the last two years of follow-up. Additional adjustment for marital status had little effect on the overall rate ratio estimate.

Table 6
Rate ratios (RR) for fertility in HIV-positive women versus HIV-negative women by year of follow-up. Adjusted for age alone and age and marital status

	Age-adjusted fertility rate ratio (RR)			
Year of follow-up	RR	95%CI		P-value
1	0.72	0.50	1.02	0.07
2	0.81	0.58	1.15	0.24
3	0.60	0.39	0.93	0.02
4	0.93	0.64	1.35	0.70
5	0.74	0.50	1.10	0.14
6	0.69	0.45	1.06	0.09
All years: adjusted for age	0.74	0.63	0.87	P<0.001
All years: adjusted for age and marital status	0.75	0.64	0.88	P<0.001

Data were not collected on age at first sexual intercourse or age at marriage. In order to examine whether HIV-positive women started sexual activity earlier than HIV-negative women we therefore compared the marital status distribution of women aged 13-19 years. In this age group, HIV-positive women were twice as likely to be married as HIV-negative women (Table 7). The difference in marital status distributions was highly statistically significant (P<0.001).

Table 7
Marital status of women aged 13-19 according to HIV status

	HIV-positive		HIV-negative	
Marital status	Number	%	Number	%

Single	55	38	1884	72
Married	71	49	685	26
Widowed	3	2	5	0.2
Divorced	17	12	56	2
Total	146	101	2630	100

X^2 for difference (df=3) = 113.0; P < .001.

Age-specific HIV prevalence rates are presented separately for women who gave birth during the study period and those who did not (Table 8). Overall, women who gave birth had 44 per cent lower odds of being HIV-positive than women who did not give birth, after adjusting for age (OR=0.56, 95% confidence interval 0.48 to 0.67). Lower HIV prevalence among women giving birth was observed in all age groups except ages 15-19 years where the odds of being HIV-positive were 43 per cent higher in women who gave birth.

Table 8
HIV prevalence by age in women who gave birth during period of observation and those who did not

	Number (%) HIV positive					
Age group	Gave birth		Did not give birth		Odds ratio[a]	P-value
13-19	448	(6.9)	2368	(4.9)	1.43	0.09
20-24	533	(13.7)	1283	(22.8)	0.54	<0.001
25-29	446	(12.1)	1179	(20.4)	0.54	<0.001
30-34	286	(5.6)	1029	(16.7)	0.30	<0.001
35-39	134	(4.5)	694	(8.2)	0.52	0.14
40-44	61	(3.3)	877	(9.7)	0.32	0.1
45-49	4	(0.0)	770	(5.7)	-	-
Total	1912	(9.5)	8200	(12.3)	0.56[b]	<0.001

[a] Odds ratio for being HIV-positive in women giving birth versus those not giving birth

[b] Odds ratio adjusted for age

Discussion

In this cohort study of over 3500 women of reproductive age located in rural southwest Uganda, the general fertility rate was 199 per 1000 woman-years (95% confidence interval 191-207) and the total fertility rate was 6.2 births per woman. Fertility rates declined significantly during the six-year period of follow-up (1989/90 to 1995/6), from 232 per 1000 (7 births per woman) to 188 per 1000 woman-years (6.1 births per woman). These rates are somewhat lower, and have declined faster, than those reported in two recent Demographic Health Surveys carried out in Uganda where total fertility rates over a similar period declined from 7.3 in 1989 to 6.8 in 1995 (Statistics Department, Uganda 1996). Fertility rates reported in the 1991 Population and Housing census for the whole of rural Uganda were also higher: TFR=7.3 births (Statistics Department, Uganda 1995). A recent study found that 8.7 per cent of men and 9.6 per cent of women in rural Masaka made use of modern contraceptive methods compared to 1.8 per cent of men and 1.7 per cent of women in rural Lira, located in northern Uganda (Blanc et al. 1996). These findings suggest that contraceptive use in rural Masaka may be higher than average for Uganda and this could explain the lower fertility rates found in the present study population. The decline in fertility observed over the period of

study was particularly evident among single women. This may reflect behaviour change, with single women increasingly postponing childbearing until after marriage, and increased use of contraception among single women. Data on contraceptive use were not collected during the study period, so it is not possible to investigate this issue further.

One of the major objectives of the study was to describe fertility rates in relation to infection with HIV. The overall prevalence of HIV infection in the present study population was 12 per cent and remained relatively stable during the study period. During the six-year period of follow-up, the general fertility rate was 212.3 births per 1000 woman-years in women without HIV infection and 194.8 per 1000 in HIV-infected women. After adjustment for age, the fertility rate in HIV-infected women was estimated as 0.74 of that in uninfected women (95% confidence interval 0.63 to 0.87; $P<0.001$). When combined with an overall HIV prevalence rate of 12 per cent, this corresponds to a three per cent reduction in fertility rates in the whole population.

Additional adjustment for marital status had little effect on estimated reduction in fertility in HIV-infected women but we were not able to adjust for the effects of several other potential confounding factors. In this context, the lack of data on sexually transmitted diseases is notable. Active syphilis, in particular, has been associated with reduced pregnancy rates in African women (Serwadda et al. 1997). As syphilis is also likely to be more common in HIV-infected women, the lack of adjustment would tend to slightly exaggerate the reduced fertility in HIV-infected women found here. We plan to carry out serological testing for active syphilis using stored sera for a sample of women in this population for use in further analyses in order to investigate this further.

Our estimate of 26 per cent lower fertility associated with HIV infection is broadly similar to the reduction of 20 per cent found in a one-year follow-up of a cohort of Ugandan women residing in the neighbouring district of Rakai (Sewankambo et al. 1994) and of 23 per cent among Zairean women followed for three years postpartum, after adjustment for birth control use (Ryder et al. 1991). In contrast, a substantially larger study carried out in Rakai district found that women with HIV infection, but without serological syphilis, had 52 per cent lower pregnancy rates than women without HIV infection or serological syphilis, after adjusting for age, marital status, gravidity, contraceptive use, lactation, history of subfertility and recent sexual history (Serwadda et al. 1997). The difference between the current findings and those of Serwadda et al. is surprising, given the close proximity of the two study populations, and is unlikely to be due to chance. It is possible that some births may have been missed in our study, but we would need to speculate a greater loss among HIV-negative women to explain the more modest effect of HIV on fertility found here. We have no data with which to assess this possibility but it seems biologically implausible. A more likely explanation is that the present population-based fertility estimates do not take account of the period following birth during which women are not at risk of getting pregnant. We would expect adjustment for this period to increase fertility rates in HIV-negative women relative to those of HIV-positives because of the larger number of births in HIV-negative women.

In contrast with the general pattern of lower fertility among HIV-infected women, infected women aged 15-19 had significantly higher fertility than uninfected women (RR=1.54, 95% confidence interval 1.07 to 2.21). This may reflect earlier sexual activity among women who became infected with HIV. Although data were not available on age at first sexual intercourse or age at marriage, HIV-infected women in this age group were almost twice as likely to be married as uninfected women, which lends support to this suggestion.

Despite current uncertainty about the exact magnitude of the effect, our findings support the suggestion that infection with HIV appears to be associated with substantially reduced fertility in sub-Saharan populations. There are several possible reasons why this might be, including biological, social and clinical (Gregson 1994). As far as biological factors

are concerned, it may be that HIV infection itself results in infertility or early foetal loss. HIV infection has been associated with non-ulcerative sexually transmitted diseases in women (Laga et al. 1993) which can result in pelvic inflammatory disease and consequent infertility (Frank 1983; Cates, Rolfs and Aral 1993). Women infected with HIV may also have higher rates of spontaneous abortions and stillbirth due to HIV transmission *in utero* (Langston et al. 1995). While plausible, the role of such biological factors is difficult to address in an epidemiological study such as ours with a large sample size.

As far as social factors are concerned, women infected with HIV may voluntarily reduce their fertility by changing their sexual behaviour or by increased use of contraception. Some data on sexual behaviour were collected during the fourth and fifth survey rounds and we will investigate this issue in further analyses. In the current study, however, the vast majority of women were unaware of their HIV status and greater use of voluntary fertility reduction among HIV-positive women therefore seems unlikely. Moreover, while we did not collect data on use of contraception for the current study, independent data from Masaka district suggest that use of modern contraceptives is less than ten per cent in this population (Blanc et al. 1996).

While many women may not know their HIV serostatus, those with advanced disease (particularly AIDS-defining symptoms, such as weight loss or fever), or those whose partner has recently died of AIDS, may suspect it and alter their sexual behaviour accordingly. Symptomatic women may reduce or stop sexual activity because of symptoms associated with advanced disease while those experiencing significant weight loss may become amenorrhoeic. Data from an ongoing natural history cohort study of a subset of HIV-positive women in the present study population suggest that around half have symptoms compatible with WHO clinical stage 3 or 4, which includes significant weight loss and chronic fever (D. Morgan, personal communication). Both asymptomatic and mildly symptomatic HIV-infected women have been found to experience significantly more episodes of amenorrhoea (Chirgwin et al. 1996). A reduction in fertility caused by HIV infection itself cannot therefore be ruled out.

The use of HIV prevalence in pregnant women to predict prevalence in women in the general population is subject to a number of culturally and socially-specific biases, of which differential fertility in HIV-positive and negative women is an important one (Boisson et al. 1996). The current finding of lower fertility in HIV-infected women has important implications for the interpretation of estimates of HIV prevalence resulting from sentinel surveillance data. With the exception of teenage women, the odds of HIV infection in women who gave birth during the study period were consistently lower than that of women who did not give birth. Odds ratios for HIV infection in women aged 20 or more (Table 8) suggest that HIV prevalence derived from antenatal clinic data may underestimate infection in the population by as much as 50 per cent. Recent surveillance data from Uganda have been interpreted as suggesting that prevalence in urban areas may be declining (Asiimwe-Okiror et al. 1995). Although lower prevalence of HIV infection in women giving birth may have less serious implications for extrapolating trends in antenatal clinic data to the general population, such predictions may not be completely free from bias (Serwadda et al. 1997). Biases associated with extrapolating from HIV prevalence in antenatal clinic attenders to the general population warrant further study.

Conclusion

In this cohort of over 3500 women of childbearing age in rural southwest Uganda infection with HIV was associated with 26 per cent lower fertility, after adjusting for age ($P<0.001$). When combined with an overall HIV prevalence rate of 12 per cent, this corresponds to a three per cent reduction in fertility rates in the whole population. These findings are unlikely

to be explained by differences in marital status, or increased use of contraception among uninfected women, as use of modern contraceptive methods in rural Uganda is low and fewer than ten per cent of women were aware of their HIV-serostatus. More likely explanations are reduced sexual activity due to clinical symptoms associated with HIV infection or lower fertility associated with co-existing infections with other sexually transmitted diseases, such as syphilis. A reduction in fertility caused by HIV infection itself cannot be excluded.

References

Allen, S., A. Serufilira, V. Gruber, S. Kegeles, P. Van de Perre, M. Carael and T. J. Coates. 1993. Pregnancy and contraception use among urban Rwandan women after testing and counselling. *American Journal of Public Health* 83:705-710.

Asiimwe-Okiror, G., J. Musinguzi, G. Tembo, et al. 1995. Declining trends in HIV infection in urban areas in Uganda. In *IX International Conference on AIDS in Africa, Kampala* (Abstract WeC206).

Batter, V., B. Matela, M. Nsuami, et al. 1994. High HIV-1 incidence in young women masked by stable overall seroprevalence among childbearing women in Kinshasa, Zaire: estimating incidence from serial seroprevalence data. *AIDS* 8:811-817.

Blanc, A.K., B. Wolff, A. J. Gage, A. C. Ezeh, S. Neema and J. Ssekamatte-Ssebuliba. 1996. *Negotiating Reproductive Outcomes in Uganda.* Calverton MD: Macro International Inc.

Boisson, E., A. Nicoll, B. Zaba and L. C. Rodrigues. 1996. Interpreting HIV seroprevalence data from pregnant women. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology* 13:434-439.

Cates, W., R.T. Rolfs and S.O. Aral. 1993. *The Pathophysiology and Epidemiology of Sexually Transmitted Diseases in Relation to Pelvic Inflammatory Disease and Infertility. Biomedical and Demographic Determinants of Reproduction.* Oxford: Clarendon Press.

Chirgwin, K.D., J. Feldman, O. Muneyyirci-Delale, S. Landesman and H. Minkoff. 1996. Menstrual function in Human Immunodeficiency Virus-infected women without Acquired Immunodeficiency Syndrome. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology* 12:489-494.

Clayton, D. and M. Hills. 1993. *Statistical Models in Epidemiology*. New York: Oxford University Press.

De Vincenzi, I., C. Jadand, E. Couturier, et al. 1997. Pregnancy and contraception in a French cohort of HIV-infected women. *AIDS* 11:333-338.

Frank, O. 1983. Infertility in sub-Saharan Africa: estimates and implications. *Population and Development Review* 9:137-144.

Fylkesnes, K., R. Mubanga Musonda, K. Kasmuba, Z. Ndhlovu, F. Mluanda, L. Kaetano and C. C. Chipaila. 1997. The HIV epidemic in Zambia: socio-demographic prevalence patterns and indications of trends among childbearing women. *AIDS* 11:339-345.

Gregson, G. 1994. Will HIV become a major determinant of fertility in sub-Saharan Africa? *Journal of Developmental Studies* 30, 3:650.

Hennekens, C. H. and J. E. Buring. 1987. *Epidemiology in Medicine*. Boston: Little Brown and Co.

Kengeya-Kayondo, J.-F., A. Kamali, A.J. Nunn, A. Ruberantwari, H.-U. Wagner and D. W. Mulder. 1996. Incidence of HIV-1 infection in adults and sociodemographic characteristics of seroconverters in a rural population in Uganda:1990-1994. *International Journal of Epidemiology* 25:1077-1082.

Kigadye, R.-M., A. Klokke, A. Nicoll, et al. 1993. Sentinel surveillance for HIV-1 among pregnant women in a developing country: 3 years' experience and comparison with a population serosurvey. *AIDS* 7:849-855.

Laga, M., A. Manoka, M. Kivuvu, et al. 1993. Non-ulcerative sexually transmitted diseases as risk factors for HIV-1 transmission in women: results from a cohort study. *AIDS* 7:95-102.

Langston, C., D. Lewis, H. Hammill, E. J. Popek, C. A. Kozinetz, M. W. Kline, I. C. Hanson and W. T. Shearer. 1995. Excess intrauterine fatal demise associated with maternal immunedeficiency virus. *Journal of Infectious Diseases* 172:1452-1460.

Mulder, D., Andrew Nunn, A. Kamali and J. Kengeya-Kayondo. 1995. Decreasing HIV-1 seroprevalence in young adults in a rural Ugandan cohort. *British Medical Journal* 311:833-6.

Mulder, D.W., A. J. Nunn, H.-U. Wagner, A. Kamali and J. Kengeya-Kayondo. 1994. HIV-1 incidence and HIV-1 associated mortality in a rural Ugandan population cohort. *AIDS* 8:87-92.

Newell, C. 1988. *Methods and Models in Demography*. Chichester: Wiley.

Nunn, A.J., D.W. Mulder, A. Kamali, A. Ruberantwari, J.-F. Kengeya-Kayondo and J. Whitworth. 1997. Five-year HIV1 associated mortality in a rural Ugandan population. *British Medical Journal* (in press).

Ryder, R.W., V.L. Batter, M. Nsuami, N. Badi, L. Mundele, B. Matela, M. Ntshudi and W. L. Heyward. 1991. Fertility rates in 238 HIV seropositive women in Zaire followed for 3 years post-partum. *AIDS* 5:1521-1527.

Selwyn, P.A., E.E. Schoenbaum., K. Davenny, et al. 1989. Prospective study of human deficiency virus and pregnancy outcomes in intravenous drug-users. *Journal of the American Medical Association* 261:1289-1294.

Serwadda, D. , R.H. Gray, M.J. Wawer et al. 1997. HIV, STDs and fertility: a population-based study in Rakai district, Uganda.

Sewankambo, N.K., M. J. Wawer, R.H. Gray, D. Serwadda, C. Li, R.Y. Stallings, S. D. Musgrave and J. Konde-Lule. 1994. Demographic impact of HIV infection in rural Rakai District, Uganda: results of a population-based cohort study. *AIDS* 8:1707-1713.

StatCorp. 1995. Statistical Software: release 4.0. College Station TX: Stata Corporation.

Statistics Department, Uganda, and Macro International Inc. 1996. *Uganda Demographic and Health Survey, 1995.* Calverton MD.

Stephenson, J.M., A. Griffioen and the Study Group for the Medical Research Council Collaborative Study of Women with HIV. 1996. The effect of HIV diagnosis on reproductive experience. *AIDS* 10:1683-1687.

Thackway, S.V., V. Furner, A. Mijch, et al. 1997. Fertility and reproductive choice in women with HIV-infection. *AIDS* 11:663-667.

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HIV infection in rural households, Rakai District, Uganda

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Abstract

The Rakai Project conducted a population-based cohort study in rural Rakai District, Uganda, a region with high rates of HIV prevalence. The cohort population described here was followed between 1990 and 1992 and consisted of all residents aged 15 years or more living in 1945 households in 31 community clusters. A detailed census was conducted at baseline in every study household. Census data were updated annually, and all inter-survey deaths, births, and migrations were recorded. Immediately following each annual census, all consenting adults were administered a socio-demographic, behavioural and health survey, and provided a blood sample for HIV testing.

HIV prevalence in the study population was high, with 19.1 per cent of adults aged 15 or more years being HIV-positive. By household, the burden of infection was even more pronounced: 31.3 per cent of households had at least one HIV-infected resident adult. Twenty seven per cent of heads of households were also HIV-positive. Overall, 3.6 per cent of study households experienced the death of an HIV-positive adult per year, and another two per cent lost an HIV-negative adult. HIV-related adult mortality had substantially more effect on subsequent household dependency ratio and on material possessions than the death of an HIV-uninfected adult, in part because the former deaths were concentrated in adults aged 15-49, the most economically active age group in this rural population. Just under 15 per cent of children aged 14 years or less had lost one or both parents, and approximately half of these parental losses are estimated to be associated with HIV infection. Nineteen per cent of study households reported at least one resident child who had lost one or both parents. Although there is evidence that loss of a parent is associated with lower school attendance, orphans overall continue to be absorbed by community households which are headed by adults.

HIV infection is very prevalent among adults in Rakai and the associated mortality imposes a substantial social and economic burden on households in the district.

Unlike most infectious diseases, HIV/AIDS especially affects adults in the prime of life, and in many African countries, large proportions of the adult population are infected. In the absence of very costly therapy, the inevitable outcomes of HIV/AIDS are substantial

morbidity and death. Because of its epidemiology, natural history and demographic distribution, the HIV epidemic can be expected to have substantial effects on households by, for example, increasing the numbers of orphans, altering dependency ratios and influencing economic well-being.

HIV/AIDS is the principal cause of adult mortality in a number of African settings including Abidjan, Côte d'Ivoire (de Cock et al. 1990), and Masaka and Rakai Districts, both situated in Uganda (Mulder et al. 1994; Sewankambo et al. 1994). In rural Mwanza, Tanzania, where only four per cent of adults were found to be HIV-positive, a third of adult deaths have been attributed to HIV infection (Todd et al. 1997). In studies by Mulder et al. (1994), Sewankambo et al. (1994) and Todd et al. (1997), all of which were population-based, mortality among the HIV-infected was found to be 9-10 per hundred person-years of observation (PYO), substantially higher than is generally reported among HIV-positive adults in developed countries.

In Masaka, Kamali et al. (1996) reported that just over ten per cent of children under age 15 years had lost one or both parents: in almost two-thirds of these cases, the father was the deceased parent. The Masaka data suggest that approximately half of recently orphaned children lost their parent or parents as a result of HIV infection. Although orphaned children continued to be cared for by the community, the concern was raised that community and household coping mechanisms could become overextended. Foster et al. (1995) in Mutare, Zimbabwe, found that 18.3 per cent of households included orphans, of whom half were also estimated to have lost one or both parents as a result of HIV infection. As in Masaka, orphaned children in Mutare were being absorbed into existing households, but given that heads of households which included orphans tended to be older and less well-educated, the researchers expressed concerns regarding households' ability to continue providing for all members in the absence of adequate support services.

The mortality data, coupled with existing information on orphanhood, suggest the need for additional research on HIV/AIDS at the population and household level. There are, however, few population-based studies which can provide direct information on HIV in households. In this paper, we present selected data from a community-based HIV cohort study conducted in rural Rakai District, southwestern Uganda.

Rakai District is bordered by northern Tanzania and Lake Victoria. The district has a population of 385,000, living in three geographic strata (Wawer et al. 1991). In the first stratum, main-road trading centres are accessible by tarmac road and service local, domestic and international traffic; these communities have a predominantly market economy which includes bars, discothêques, small hotels or lodges and multiple shops. Communities in the intermediate trading village stratum, situated on secondary dirt roads, have a mixed agricultural and market economy, serve as foci for local trade and communications, and contain small shops but almost no bars or lodges with sleeping facilities. Rural agrarian villages are located off main and secondary roads, and their economy is based almost exclusively on agricultural production, much of it for household consumption or barter. Approximately five per cent of the district's population resides in the trading centres, four per cent in the trading villages, and 91 per cent in the agricultural villages. The majority of Rakai residents are Baganda. As described by Seeley et al. (1993), the descent system among the Baganda is patrilineal and women move into the husband's home upon marriage, in many cases at some distance from their natal home. The extended family group can be dispersed over several villages, as young men do not necessarily set up new households near their parents or brothers.

AIDS was first recognized in the district in the mid-1980s (Serwadda et al. 1985). Data from the 1989-1992 Rakai Project cohort indicate that HIV prevalence in the district is high and varies considerably by geographic stratum: in 1990, prevalence rates in persons aged 15

years and over were 35 per cent in the trading centres, 23.1 per cent in the intermediate trading villages and 11.8 per cent in the rural villages ($p < 0.001$) (Sewankambo et al. 1994). Extrapolating to the district as a whole, we estimated that 13 per cent of persons aged 13 years or more were HIV-infected in 1989-1990 (Wawer et al. 1991), a rate consistent with the results of the 1987 Ugandan National Serosurvey, which reported a prevalence of 12.1 per cent for rural central Uganda (Kaheru 1989), the region in which Rakai is situated.

Mortality among adults with HIV infection, at 119.9 per 1000 person-years of observation, was almost ten times that of uninfected subjects (RR = 9.5, 95%CI = 6.0-14.9) (Sewankambo et al. 1994). The mortality rate among HIV-positive males was almost 50 per cent higher than in HIV-positive females (147 per 1000 PYO compared to 98, $p < 0.05$). In keeping with the higher HIV prevalence in trading centres than in rural communities, HIV-related deaths occurred disproportionately among trading centre residents. Similarly, HIV was more prevalent in better educated persons and in those who worked in the cash economy (Serwadda et al. 1992), and these distributions were also reflected in HIV-related death rates (Sewankambo et al. 1994). Extrapolating from data collected in the Rakai cohort, we estimate that HIV-negative adults, who constitute approximately 87 per cent of all adults in the district, contribute an estimated 2100 deaths per year. HIV-infected adults, who constitute only 13 per cent of the adult population, experience almost 3000 deaths per year in the district. The Rakai setting thus offers the opportunity to observe selected socio-demographic effects of the HIV epidemic on rural households in three geographic settings.

Method

The smallest political unit in Rakai District is the Level One Resistance Committee (RC1), each containing 100 households on average. In 1989, we established the Rakai Project population-based cohort study using two-stage stratified cluster sampling. In the first stage, 21 RC1s were chosen at random from the complete list of district Committees. In order to ensure adequate representation of potentially high-prevalence communities, trading centres and trading villages were oversampled in relation to their relative populations in the district: six clusters were drawn from each of the trading-centre and trading-village strata, and nine were located in agrarian villages. Clusters were created by selecting one household at random in each community; this household and 39 contiguous residences situated in a concentric circle around the index household were enrolled in the study. To ensure adherence to the sampling protocol, survey supervisors drew detailed community maps before sampling. A household was defined as comprising persons who slept under one roof and ate out of a common pot. In the communities under study, households tend to be well defined and to have individual, separate houses.

In 1990, the cohort was enlarged by expanding each of the 21 existing clusters by 20 contiguous households (for a total of 60 households per cluster), and by adding ten new clusters of 60 households each. The new clusters were randomly selected from the trading-centre and trading-village strata, for a total of 11 clusters in each of these strata and nine in the rural, agrarian stratum. In addition, in cases where a member of an existing cohort household set up a new residence (for example, a son or daughter married and established their own family), the new household was also included in subsequent survey rounds. In 1990, 85 such households were included in the annual enumeration and survey, for a total of 1945 study households in that year. If an existing study cluster household moved as a unit to a new location within the community or to another community which contained a study cluster, cohort follow-up was maintained. Each study household received a baseline visit, and was subsequently followed with annual visits over two years (1990-1992). The data presented in this paper are drawn from the baseline and two-year follow-up of the expanded cohort study.

During the baseline and annual follow-up visits, an enumeration team visited all study households and updated a list of all eligible study subjects, defined as adults and children resident in the household for at least three months of the previous year, whether present at the time of the enumeration or not (*de jure* population). The broad classification of residence was used to ensure inclusion of the majority of persons having frequent and substantial contact with household members. Basic demographic data (age, sex, place and household of residence, relationship of each member to the head of the household) were recorded for all cohort subjects, as well as reasons for any absence. Household-level data were also collected on characteristics such as quality of housing and household possessions. Following the enumeration, knowledge and behavioural data, as well as a whole blood sample, were collected from all consenting subjects. In 1990, 9275 *de jure* residents were enumerated in the study households, of whom 50.5 per cent were aged 15 and over. Seventy per cent (6473) of the enumerated population were present in the study clusters at the time of the 1990 enumeration-survey, and we obtained a serological sample and interview data on a total of 5973 persons (90% of those present in the clusters at the time of the survey), of whom 2929 were aged 15 years and over. Of these persons, 1525 were identified through the census as being the head of the household. Thus, 78.4 per cent of household heads within the study clusters were included in the survey. In the remaining 420 households enumerated in 1990, 368 heads of households were absent at the time of the survey and 52 did not consent to participate. The overall levels of absenteeism and refusal among heads of households was thus somewhat lower than in the general adult study population.

Interview information was collected regarding socio-demographic characteristics, behaviour and health status. Venupuncture whole-blood specimens were spun in the field in a portable centrifuge, and the separated serum was frozen in liquid nitrogen. The frozen specimens were transported to the Uganda Virus Research Institute, a World Health Organization HIV reference laboratory in Entebbe, for storage at -20 C°. Initial serum screening was carried out using a commercial ELISA assay (Recombigen EIS, Cambridge Biosciences), and all samples found positive on ELISA were confirmed by Western Blot (Biorad, USA).

In 1991 and 1992, the enumeration and survey were repeated in all study households in the 31 clusters. During the 1991 and 1992 visits, data were also collected on changes in household composition and possessions that occurred during the follow-up year. Events of interest included deaths, births, marriage and in- and out-migrations since the time of the previous enumeration-survey. Follow-up information, a repeat questionnaire or survival status as reported by other household members, was acquired on 93 per cent of persons on whom we had 1990 serological and health data. Follow-up information on individuals was unavailable in cases where a person had migrated and the remaining family reported no contact, or where the entire household was absent and no data could be obtained from extended family members. In addition, information regarding 1991 survival status was available on 86 per cent of the enumerated adults from whom we had not received a serological specimen or interview in 1990 owing to absence or refusal. With respect to households, 1667 (85.7%) of the 1945 enrolled in 1990 remained in the study between 1990 and 1992.

The present data analysis focuses on HIV-prevalence among adults and among heads of households at baseline, as well as on the proportion of households with at least one HIV-positive adult. Follow-up data on mortality at the household level among all adults and among heads of households are also presented. Finally, the proportion of households with resident orphans under 15 is examined.

Results

The age and sex structure of the cohort population was typical of rural African communities. Individuals under the age of 15 constituted 49.5 per cent of the enumerated cohort, and women of reproductive age (15-49) made up 21.3 per cent of the study population, consistent with 1991 National Census data for Rakai. The one under-represented group was young men aged 15-29, an age where mobility and migration related to work, military service and schooling resulted in approximately 25 per cent fewer male residents than females in the same age group. Most of the male absenteeism occurred in the trading centres and trading villages.

Average household size in Rakai rural villages, at 5.3 persons, was larger than in either the intermediate trading villages (4.7) or the trading centres (4.3), and rural households had more children under the age of 15 (mean 2.5) than did trading-centre households (mean 2.0). The adult population in the trading centres was proportionately composed of more women and was younger, with two-thirds of the adults aged 15-29, than were the populations of the intermediate villages or rural agrarian villages, where one-half of adults were aged 15-29. The data are consistent with the different economies of the three strata: work in shops, bars, lodges, truck driving and vending in the trading centres; traditional agricultural practices in the rural villages; and a mix in the intermediate trading villages.

Over a quarter (28.4%) of study households were headed by women, with marked differentials in the distribution of female heads by geographic stratum. In the trading centres, 43.8 per cent of households were headed by women, whereas in the trading villages 22 per cent and in the rural communities 18.5 per cent were female-headed. The majority of female household heads (66.5%) were widowed, separated or divorced; 19.9 per cent were married, and 13.6 per cent were single. Among male heads of households, most (83.8%) were married, another 11.2 per cent were widowed, separated or divorced, and only five per cent were single.

HIV prevalence: individual and household data

As indicated earlier, in 1990, the first year of the expanded cohort study, HIV prevalence among persons aged 15 and over was 35 per cent in the trading centres, 23.1 per cent in the trading villages, and 11.8 per cent in the rural, agrarian villages. Overall, 19 per cent of cohort adults were HIV-positive. Ninety-seven per cent of all adult HIV infection was concentrated in persons aged 15-49 years, the economically active age group. By household, the burden of HIV infection was even greater than is reflected in the population prevalence rates. Table 1 illustrates individual adult HIV-prevalence by stratum (1990 data), compared to the proportion of households having at least one HIV-positive adult. In the trading centres, where 35 per cent of adults were infected, fully 47 per cent of households had at least one HIV-positive adult resident. In rural villages, the proportion of households with a seropositive adult, 20.3 per cent, was almost double the adult HIV-prevalence, 11.8 per cent. In those households with at least one HIV-infected adult, the mean number of seropositive adults was 1.3.

The distribution of HIV infection was also examined among heads of households (Table 1). In the trading centres, and to a lesser degree in the rural communities, HIV-prevalence was higher among heads of households than in the general adult population. Table 2 illustrates HIV-prevalence among heads of households by sex and by marital status. In all strata, female heads of households were more likely to be HIV-positive than male heads, although the difference was significant only in the trading centres. Thus, in the latter communities, 52.8 per cent of female household heads were HIV-positive, compared to 33.7 per cent of male heads ($p < 0.001$); in the trading villages, the respective female and male prevalence rates were 28.7 and 22.2 per cent (not significant); and in the rural villages, the rates were 17.9 and 12.5 per

cent (not significant). This variation in sex-specific HIV rates among household heads reflects differences in HIV-prevalence by the marital status of heads in the different strata. Overall, HIV-prevalence was highest among female heads of households who were currently single (57%) or married (53.5%), and it was 31.2 per cent among female household heads who were currently divorced, separated or widowed (Chi^2 for trend $p < 0.001$). Far fewer male than female heads of households reported being currently separated, divorced or widowed. However, men in this relationship category had the highest HIV-prevalence among male household heads.

Table 1
HIV prevalence (%) in all study subjects aged 15 years or more and in household heads, males and females combined; and percentage of households with one or more HIV+ adults: baseline 1990 data, by stratum.

	HIV prevalence among all adults 15+(%)	HIV prevalence among household heads (%)	Households with a least one HIV+ adult (%)
Main road, trading centres	35.0	42.1	47.0
Trading villages, secondary roads	23.1	23.6	31.6
Agrarian villages	11.8	13.5	20.3
All (cohort)	19.1	26.9	31.3
District (extrapolated)	13	15	22
Number of observations	2929	1525	1945

For both male and female household heads, positive HIV serostatus was associated with smaller household size. HIV-negative male-headed households had a mean of 5.3 persons, whereas the mean size of households headed by HIV-positive males was 4.4; among women, the mean household size was 4.3 for HIV-negative and 3.2 for HIV-positive women. This difference in household size was seen in all strata, and was due predominantly to differences in the mean number of children by HIV-status of the head of household. HIV-negative male-headed households included an average of 2.6 children, compared to two children in households with HIV-positive male heads. HIV-negative females headed households with a mean number of 2.2 children, whereas HIV-positive women headed households with only 1.5 children on average. In keeping with these observations, households headed by HIV-positive persons had a lower total dependency ratio, defined, in this rural setting, as the mean number of adults aged 50 and over and of children under age 15 per economically active adult aged 15-49. For the cohort overall, the dependency ratio in households headed by HIV-negative males was 1.3, compared to 1.0 in households headed by HIV-positive males; and 1.5 for households headed by HIV-negative women, compared to 1.2 in households headed by HIV-positive women.

Table 2
HIV prevalence of household heads by sex, place of residence and marital status; baseline data, 1990.

	Male household heads (N=1092)		Female household heads (N= 433)		Total both sexes (N=1525)	
	HIV+/N	Per cent	HIV+/N	Per cent	HIV+/N	Per cent
Trading centres						
Single	6/21	28.6	30/45	66.7	36/66	54.5
Married	82/246	33.3	43/65	66.1	125/311	40.2
Div/sep/wid.	10/24	41.7	47/117	40.2	57/141	40.4
Total	98/291	33.7	120/227	52.9	218/518	42.1
Trading villages						
Single	4/26	15.4	3/12	25.0	7/38	18.4
Married	72/342	21.0	0/9	-	72/351	20.5
Div/sep/wid.	20/64	31.2	32/101	31.7	52/165	31.5
Total	96/432	22.2	35/122	28.7	131/554	23.6
Agricultural villages						
Single	0/8	-	½	50.0	1/10	5.0
Married	37/327	11.3	3/12	25.0	40/339	11.8
Div/sep/wid.	9/34	26.5	11/70	15.7	20/104	19.2
Total	46/369	12.5	15/84	17.9	61/453	13.5
All strata combined						
Single	10/55	18.2	34/59	57.6	44/114	38.6
Married	191/915	20.8	46/86	53.5	237/1001	23.7
Div/sep/wid.	39/122	32.0	90/288	31.3	129/410	31.5
Total	240/1092	22.0	170/433	39.3	410/1525	26.9

We have previously reported that individuals with higher education were significantly more likely to be HIV-infected (Serwadda et al. 1992); as would be expected, the same trend was observed in heads of households. In the 213 heads with secondary education, 36.6 per cent were HIV-positive, compared to only 15.8 per cent HIV-positive among heads with no education ($p < 0.001$). Household heads with some or completed primary schooling had an intermediate HIV-prevalence of 29.7 per cent. Women heads of household with secondary or higher education were more likely to be HIV-positive (60% prevalence) than men with similar levels of education (31.2% prevalence); this is partly related to the fact that educated female heads of household resided almost exclusively in trading centres where exposure to HIV infection is most pronounced. Households with HIV-infected adults tended, at baseline, to have characteristics associated with higher socio-economic status. Households with a radio, wooden or cement floors, iron roofs and a higher number of rooms per member were significantly more likely to have an HIV-positive head of household.

Selected effects of HIV-related mortality on households

As illustrated in Table 3, a substantial proportion of Rakai study households experienced the death of an adult as a result of HIV infection (1990-1991 data). Just over five per cent of all cohort households experienced an adult death in that study year: two-thirds of these deaths occurred among HIV-positive adults. Four per cent of households lost an economically active adult each year: 82 per cent of these deaths were associated with HIV infection. Similarly, the great majority (71%) of deaths of heads of households were associated with HIV. Extrapolated to the district as a whole, that is, adjusting for the oversampling of trading centres and trading villages in the Rakai cohort, we estimate that 2.4 per cent of all Rakai households experienced an HIV-related adult death per year and that 2.3 per cent of all Rakai households lost an adult in the most economically active age range each year as a result of the epidemic. Of households which did experience adult deaths, 92.9 per cent reported only one adult death during the two-year follow-up, six per cent experienced two adult deaths, and 1.1 per cent three adult deaths.

Table 3
Percentage of all study households experiencing an adult death, 1990-1991 (N=1945).

	Deceased was HIV -	Deceased was HIV+	Any death (HIV+/HIV- combined)
Any adult death (age 15+)	2.0	3.6	5.5
Death of adult 15-49	0.7	3.4	4.1
Death of head of household	0.8	2.0	2.8

In households with no adult death, household size remained essentially unchanged between baseline and follow-up; in households with an adult death, household size declined on average, by 1.7 persons (adults and children combined and inclusive of the deceased person). The probability that a given household would remain in the study community during follow-up was affected both by initial household size and by subsequent adult deaths. Households with only one resident adult were highly mobile, and 32 per cent migrated out of the study communities during follow-up (1990-1992), even in the absence of adult death; virtually all such households migrated or were subsumed following the death of the adult member. Of households with two adults at baseline, 14 per cent migrated out in the absence of any adult death, 24 per cent migrated out following an HIV-unrelated death, and fully 57 per cent moved out of the community following the death of an HIV-positive adult. Households with three adults at baseline were substantially less likely to move: seven per cent with no adult death moved out, seven per cent moved out following a non-HIV death, and nine per cent migrated following the death of an HIV-positive adult member.

As shown in Table 4, among those households which remained in the cohort at the time of follow-up, a greater proportion (14 %) of those which had experienced the death of an HIV-positive adult reported no adult in the age group 15-49 years at follow-up, compared to households with no adult death or an HIV-negative adult death. Similarly, the dependency ratio increased in those households which experienced the death of an HIV-positive adult, compared to households with no adult death or those with the death of a seronegative adult (Table 5).

Table 4
Percentage of households with no resident adults aged 15-49 at baseline and at follow-up, by adult mortality which occurred in the household between 1990 and 1992, for households followed during that period (N= 1667).

	No adult death N = 1483	Death of HIV- adult N = 74	Death of HIV+ adult N = 110
Baseline	6	0	2
Follow-up	7	3	14

Table 5
Dependency ratio, defined as the mean number of children under age 15 plus adults aged 50 or more, per adult aged 15-49, in study households at baseline (1990) and at follow-up (1992), by adult mortality experienced by the household during follow-up.

	No adult death N= 1483	Death of HIV- adult N = 74	Death of HIV+ adult N= 110
Baseline	1.35	1.30	1.16
Follow-up	1.34	1.34	1.47

Households reported substantial financial outlays on HIV-related illness and burial. Two-thirds of households experiencing an HIV-related adult death reported selling property to pay for medical treatment and burial costs. Extended family members outside the immediate household were reported to have contributed to medical and burial costs in 40 per cent of cases. Local community and non-governmental organizations provided additional short-term support for medical costs in 18 per cent of cases, primarily in the form of medication; and support for burial costs in 68 per cent of cases, primarily in the form of food donated for the burial service.

Among households with no adult deaths, the proportion owning basic possessions increased over two years of observation (Table 6). Households experiencing an adult death, particularly if the deceased was HIV-positive, reported at follow-up fewer possessions including such economically important items as bicycles (Table 6). The data suggest a negative economic effect related to medical and burial costs and, possibly, to loss of earnings with the death of an economically active person.

Table 6
Percentage of households reporting selected possessions at baseline (1990) and at follow-up (1992), by adult mortality (age 15+) experienced in the household during follow-up

Possession		No adult death N= 1483	Death of HIV- adult N = 74	Death of HIV+ adult N= 110
Bicycle	Baseline	34	38	39
	Follow-up	41	38	33
Radio	Baseline	30	36	42
	Follow-up	38	35	36

Orphans

Only 1.9 per cent of HIV-uninfected adults aged 15-49 reported having a deceased spouse, compared to 8.3 per cent of HIV-infected adults (RR = 4.4, 95% CI 2.8-7.0). All children under the age of 15, or their guardians, were questioned regarding the status of the child's biological parents. Children who had lost one or both biological parents are considered orphans for the purposes of this paper, in keeping with the definition used in other African studies (Foster et al. 1995; Kamali et al. 1996). In 1990, 4.5 per cent of children had a deceased mother, 7.9 per cent had a deceased father, and 2.4 per cent had lost both parents, for a total of 14.8 per cent under the age of 15 who had lost one or both parents. Nineteen per cent of cohort households reported at least one resident child who had lost one or both parents, with no marked differentials by geographic stratum.

Despite HIV-related and other adult mortality, most Rakai households continue to be headed by adults: 97 per cent of cohort households had at least one resident aged 17 years or more, an age at which persons may be considered fully economically independent adults in this rural area. If age 20 is considered to represent economic adulthood, 94 per cent of all households contained at least one resident at or above this age. Just under one per cent of households were headed by children under the age of 15.

From project data, we estimate that there are approximately 29,000 children resident in the district who have lost at least one parent; 4500 to 5000 of these children have lost both parents. These 29,000 children represent 15 per cent of all children under age 15 and 7.5 per cent of the total population of the district. Furthermore, we estimate that by 1992, approximately half of surviving orphans under age 15 had lost one or both parents to HIV; among the youngest orphans, those under five, as many as two-thirds may have lost at least one parent as a result of HIV.

Within households, there is some evidence that orphaned children are at a disadvantage compared to children with two living parents. Households with orphans tend to be headed by females: 62 per cent of households with orphans have a female head of household, compared to only 30 per cent of households without orphans. Households with orphans tend to have lower adult per capita incomes than do households with no orphans, in part probably as a result of being female-headed: the difference in per capita income is approximately 15 per cent. Fifty-six per cent of orphaned children aged 15 and under are enrolled in school, compared to 64 per cent of children with both parents living ($p < 0.05$).

Discussion and conclusions

Adults in Rakai district have high rates of HIV infection, but the current analysis indicates that the household burden of HIV infection is even higher: 19.1 per cent of all adult cohort subjects were HIV-infected, whereas 31.3 per cent of study households had at least one HIV-positive resident adult. Similarly, HIV prevalence among heads of households, many of whom have substantial economic and organizational responsibility for the household, was higher than in the general adult study population, with 26.9 per cent of household heads being infected. The higher rate of HIV at the household than at the individual level is due to the fact that many households contain only one infected person but multiple HIV-negative residents. For example, among marital couples in the Rakai cohort, approximately 14 per cent were HIV-concordant-positive (i.e. both positive), but almost an equal proportion were HIV-discordant (i.e. only one positive spouse) (Serwadda et al. 1995). With the addition of unmarried HIV-infected adults, these data are compatible with the observed proportion of households with at least one HIV-positive resident. This finding that a high proportion of households have an HIV-positive adult implies that many families are or will be affected

within the coming decade by HIV-related morbidity and mortality. These effects may be exacerbated by the fact that the HIV-positive adult is often a breadwinner and not infrequently the head of the household.

Within the cohort, 28.4 per cent of households were headed by women, and female household heads had higher rates of HIV infection than their male counterparts. Among the 433 women who reported heading their households, the majority (66.5%) were currently widowed, divorced or separated. Although it has been postulated that marital disruption may lead to increased risk of HIV infection (Nabaitu, Bachengana and Seeley 1994), in this subgroup of women reporting current marital disruption, HIV-prevalence was lower, at 31.3 per cent, than among female heads who reported they were single or married, in whom the prevalence was 57.6 per cent and 53.5 per cent, respectively. A substantial number of single women resided in the trading centres, and our previous data indicate that many of these women work in bars and lodges and are involved in some commercial sex transactions (Serwadda et al. 1992). Married women who head a household are more likely to have a husband who is away for work; mobility and migration have also been found to be associated with HIV risk (Serwadda et al. 1992; Nunn et al. 1995), and married female household heads face higher exposure through their husbands.

The number of children under age 15 years was lower, on average, in households headed by HIV-positive adults than if the household head was HIV-uninfected. The overall dependency ratio was also lower in households with HIV-positive adults. The smaller household size and lower dependency ratio in households with HIV-positive heads was related to four factors: a higher proportion of HIV-positive adults who live alone, in particular women working in the cash economy, including bars; the fact that HIV-positive adults are on average younger than HIV-negative adults, and thus have had less time to have children; lower pregnancy rates among the HIV-positive women which we have reported elsewhere (Sewankambo et al. 1994; Gray et al. 1996; Serwadda et al. 1997); and higher mortality among the infants of HIV-positive mothers, which we have also previously reported (Sewankambo et al. 1994). The lower dependency ratio found in households with HIV-infected adults has implications for projections of the overall effect of adult HIV-related deaths on household and on community coping.

In the follow-up period reported in this paper, a substantial proportion of households experienced adult deaths, and two-thirds of these deaths occurred in HIV-infected individuals. In the economically most active age group, over 80 per cent of the deaths observed in these households were associated with HIV. Households experiencing an HIV-related adult death were more likely to have no adults in the economically most active age group at follow-up, compared to households with no death or the death of an HIV-uninfected adult (Table 4). As would be expected from these data, the total dependency ratio increased in households with an HIV-related adult death (Table 5). HIV-related mortality appears to have deleterious effects on household economic well-being, as suggested by the fact that two-thirds of households affected in this manner reported selling household possessions to cover illness and burial costs, and by a reduction of the proportion of such households with selected possessions at follow-up, as seen in Table 6; it also affects stability, as reflected by the higher proportion of households which left the study communities following the death of an HIV-positive adult, particularly if the original household had only two adult members at baseline. Households which experienced adult deaths also lost other family members, a phenomenon noted as well in a separate study in Tanzania (Ainsworth, Ghosh and Semali 1995). Factors which may contribute to the alterations in economic well-being and stability include loss of income, illness and burial expenditures and, potentially, stigmatization of the remaining spouse, resulting in a move outside the community. Only 40 per cent of households reported receiving help from extended family networks with illness and burial costs, a finding compatible with

observations by Seeley et al.(1993), which suggested that support from the extended family could be limited in this economic and cultural context. The burden of HIV infection on household well-being is likely to be increased by the fact that HIV disproportionately affects better-educated individuals and those who work in the cash economy.

Our overall estimate of 29,000 orphaned children in Rakai suggests that seven per cent of the population of the district is composed of children who have lost one or both parents. Since most parental deaths occurred before the study reported above, it is not possible to determine the cause of mortality in most cases. However, from adult death rates at the time of the cohort study described here, we estimate that approximately half of the surviving orphaned children had parents who had died of HIV infection. Although HIV is the leading cause of adult death at this time, HIV transmission in Rakai was probably not common until approximately the late 1970s (Serwadda et al. 1985), and probably did not begin to contribute substantially to adult mortality until the mid-1980s, so that older orphaned children in the cohort study are likely to have lost parents to other causes: almost 20 per cent of orphans in the study population were 12-14 years of age. In addition, many HIV-related adult deaths occur in relatively young age groups before full family size is achieved; the offspring of HIV-infected persons have higher mortality rates and are less likely to survive and be counted as orphans (Sewankambo et al. 1994); and fertility in HIV-infected women is lower than in the uninfected (Sewankambo et al. 1994; Gray et al. 1996; Serwadda et al. 1997). All these factors tend to reduce somewhat the numbers of 'AIDS orphans', although the overall numbers of children who have lost and continue to lose their parents as a result of the epidemic is nonetheless substantial.

Care for orphans represents a significant burden for the population of Rakai: 19 per cent of households reported caring for at least one child under the age of 15 years who had lost one or both parents. Seventy per cent of these orphans had lost their father, but only 46.6 per cent had lost their mother. The higher rate of children who had lost their father is compatible with data which show higher HIV-related mortality in infected men compared to HIV-positive women and more frequent widowhood among women than men, and with data which indicate that within marital couples, husbands are frequently older (Nalugoda et al. 1997), compatible with earlier death of the male spouse. Also, within the rural villages, husbands tend to be HIV-infected before the wives (Serwadda et al. 1995; Konde-Lule et al. 1997), which also increases the probability that the man will predecease his wife. The higher rate of paternal loss has been reported in Masaka district as well (Kamali et al. 1996).

However, despite the severity of the HIV epidemic in the district, the majority of Rakai cohort households continue to be headed by adults aged 15-49 years: in 1990-1991, only one per cent of households were headed by children under age 15, and another six per cent by adults aged 50 years or more. In addition, our observations and reports by village leaders suggest that the phenomenon of 'homeless orphans' is very rare. Nonetheless, the data suggest that orphaned children are at some disadvantage. Households which house orphans are more likely to be headed by women and to report lower incomes, and the data suggest somewhat lower rates of school attendance by children who have lost a parent.

In conclusion, HIV infection has had a substantial impact on the structure and economic welfare of Rakai households, and these effects would not be captured by examining adult HIV-prevalence alone. A very high proportion of Rakai cohort households have HIV-positive adult members, 3.6 per cent experienced an HIV-related adult death each year, and 19 per cent of households report caring for a child under age 15 who has lost one or both parents, of whom approximately half died as a result of HIV. The present analysis illustrates the importance of defining an appropriate 'population at risk'. Although epidemiological studies tend to focus on individuals, the importance of the family and household as the socially relevant unit should not be underestimated and merits further study.

References

Ainsworth, M., S. Ghosh and I. Semali. 1995. The impact of adult deaths on household composition in Kagera Region, Tanzania. Policy Research Department, World Bank, Washington DC, unpublished paper

de Cock, K.M., B. Barrere, L. Diaby, et al. 1990. AIDS - the leading cause of adult death in the West African city of Abidjan, Ivory Coast. *Science* 249:793-796.

Foster, G., R. Shakespeare, F. Chinemana, H. Jackson, S. Gregson, C. Marange and S. Mashumba. 1995. Orphan prevalence and extended family care in a peri-urban community in Zimbabwe. *AIDS Care* 7:3-17.

Gray, R.H., M.J. Wawer, F. Wabwire-Mangen, et al. 1996. Reduced fertility among HIV-infected women: results of cross-sectional and prospective studies in rural Uganda. Paper presented at 11th International Conference on AIDS, Vancouver, 7-12 July. Abstract Mo.B.541.

Kaheru, Z. (Minister of Health, Republic of Uganda). 1989. Results of the Ugandan National Serosurvey, Kampala, 30 November (Press Conference).

Kamali, A., J.A. Seeley, A.J. Nunn, J.F. Kengeya-Kayondo, A. Ruberantwari and D.W. Mulder. 1996. The orphan problem: experience of a sub-Saharan African rural population in the AIDS epidemic. *AIDS Care* 8:509-515.

Konde-Lule, J.K., M.J. Wawer, N.K. Sewankambo, et al. 1997. Adolescents, sexual behavior and HIV-1 in rural Rakai district, Uganda. *AIDS* 11:791-799.

Mulder, D.W., A.J. Nunn, A. Kamali, J. Nakiyingi, H.U. Wagner and J.F. Kengeya-Kayondo. 1994. Two-year HIV-1 associated mortality in a Ugandan rural population. *Lancet* 343:1021-1023.

Nabaitu, J., C. Bachengana and J. Seeley. 1994. Marital instability in a rural population in south-west Uganda: implications for the spread of HIV-1 infection. *Africa* 64:243-250.

Nunn, A.J., H.-U. Wagner, A. Kamali, J.F. Kengeya-Kayondo and D.W. Mulder. 1995. Migration and HIV-1 seroprevalence in a rural Ugandan population. *AIDS* 9:503-506.

Seeley, J., E. Kajura, C. Bachengana, M. Okongo, H.U. Wagner and D. Mulder. 1993. The extended family and support for people with AIDS in a rural population in south west Uganda: a safety net with holes? *AIDS Care* 5:117-122.

Serwadda, D., R.H. Gray, M.J. Wawer, R.Y. Stallings, N.K. Sewankambo, J.K. Konde-Lule, B. Lainjo and R. Kelly. 1995. The social dynamics of HIV transmission as reflected through discordant couples in rural Uganda. *AIDS* 9:745-750.

Serwadda, D., R.D. Mugerwa, N.K. Sewankambo, et al. 1985. Slim disease: a new disease in Uganda and its association with HTLV III infection. *Lancet* ii:849-852.

Serwadda, D., M.J. Wawer, S.O. Musgrave, N.K. Sewankambo, J.E. Kaplan and R.H. Gray. 1992. HIV risk factors in three geographic strata of rural Rakai District, Uganda. *AIDS* 6:983-989.

Sewankambo, N.K., M.J. Wawer, R.H. Gray, D. Serwadda, C.Li, R.Y. Stallings, S.D. Musgrave and J. Konde-Lule. 1994. Demographic impact of HIV infection in rural Rakai District, Uganda: results of a population-based cohort study. *AIDS* 8:1707-1713.

Todd, J., R. Balira, H. Grosskurth, et al. 1997. HIV-associated adult mortality in a rural Tanzanian population. *AIDS* 11:801-807.

Wawer, M.J., D. Serwadda, S.D. Musgrave, J.K. Konde-Lule, M. Musagara and N. Sewankambo. 1991. Dynamics of the spread of HIV-1 infection in a rural district of Uganda. *British Medical Journal* 303: 1303-1306.

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Orphanhood, child fostering and the AIDS epidemic in rural Tanzania*

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Abstract

The AIDS epidemic has caused an increase in adult mortality and consequently an increase in the numbers of orphaned children. Data were used from the Kisesa Community Study in northwest Tanzania, to assess the prevalence and consequences of orphanhood in the context of existing child care practices in a rural area with moderately high HIV-prevalence. This study was carried out in a ward with about 20,000 people with HIV prevalence of 6.2 per cent among adults 15-44 years and slightly over one-third of adult deaths associated with HIV/AIDS.

Seven point six per cent of children under 15 and 8.9 per cent of children under 18 had lost one or both parents. Child fostering was very common. Virtually all orphans and foster-children were cared for by members of the extended family, often the maternal grandparents: 14 per cent of households had at least one orphan. Such households did not have a lower economic status, but had a less favourable dependency ratio. Households with orphans were also more likely to be female-headed. Follow-up mortality rates were similar among orphans, foster-children and other children, for both sexes. Mobility was much higher among orphans and foster-children, and orphans and foster-children had somewhat lower school attendance rates: lower enrolment and higher dropout rates.

The problem of rapidly increasing numbers of orphans needs to be considered in the context of previously high levels of adult mortality, child-fostering practices and general poverty. The extended family seems to be able to absorb the increase in orphans, because caring for children of other members of the family is widespread, whether the parents are alive or dead. This study yields no evidence that orphans as a group are disadvantaged, although certain subgroups of orphans or orphan households may be more vulnerable and in need of support.

Adult mortality in sub-Saharan Africa is expected to increase rapidly during the 1990s and beyond, owing to the AIDS epidemic. As AIDS mortality primarily affects adults in the reproductive ages, this will lead to a rapid increase in the number of orphaned children. Empirical data from Uganda (Kamali et al. 1996) and Zimbabwe (Foster et al. 1995), and simulations (Gregson, Garnett and Anderson 1994) have indicated the magnitude of the

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problem of orphanhood in association with the AIDS epidemic. Projections showed that there could be as many as one orphan for every two healthy economically active women about ten years after HIV prevalence has reached its peak in areas severely affected by the epidemic (Gregson et al. 1994). In 1995, WHO estimated that about 10 million children had been orphaned through AIDS since the beginning of the epidemic in Africa.

The most detailed studies on orphanhood in association with AIDS have been conducted in Mutare, Zimbabwe, in an area with high HIV-prevalence (Foster et al. 1995, 1996). About half of parental deaths in the five years preceding a survey in 1992 were thought to be associated with HIV/AIDS and 13 per cent of all children under 15 years were orphans. The extended family continued to be the main source of care for orphans. The quality of care for orphans by the extended family was generally considered satisfactory and comparable to care for other children. However, some traditional patterns of care for orphans were changing. Maternal relatives were now the main carers, while in the past the paternal extended family was the main source of orphan care.

Similar conclusions were reached in a study in rural Uganda (Kamali et al. 1996). No significant differences in schooling and mortality could be observed between orphans and other children. It was concluded that the family system was still coping with the more than 40 per cent increase in orphans due to the AIDS epidemic.

This study presents data on orphanhood and child care patterns from a rural area in Tanzania, where HIV prevalence is moderately high, but overall adult mortality is likely to have been high in the decades preceding the AIDS epidemic. The orphan problem is analysed in the context of pre-existing child care patterns, particularly the practice of child fostering.

Data and methods

The study was conducted in Kisesa ward in Mwanza Region, northwest Tanzania. The ward has a population of 20,000 and lies 20 kilometres east of the regional capital Mwanza, along the main road to Kenya. Kisesa ward includes six villages, with about 40 per cent of its population located in a trading centre along the road. More than 90 per cent of the population belong to the Sukuma tribe, which is also the largest ethnic group in Tanzania.

In Tanzania, the national AIDS program defines an orphan as a child under the age of 18 years whose mother (maternal orphan) or father (paternal orphan) or both parents (double orphan) have died. Other children can likewise be divided into children living with both biological parents, children living away from their mother (maternal foster-child), from their father (paternal foster-child) and from both parents (double foster-child). Different definitions of child fostering have been used in studies from mainly West Africa. Some have defined a foster-child as a child not living with the mother (Bledsoe, Ewbank and Isiugo-Abanihe 1988), but others used local definitions rather than location of the biological parents (Renne 1993; Castle 1995). We used demographic rather than socio-cultural criteria to define foster-children. An important difference from all other studies is that we excluded orphans from the category of foster-children. In other words, foster-children in our study have living parents. This distinction was not made because of local concepts, but because we were interested in comparing orphans with children who were brought up by other people than the biological parents.

In this study several data sources were used. Data on the prevalence of orphanhood and fostering were obtained from a baseline census. In 1994 all households in Kisesa ward were visited by local field workers who listed all persons, with their sex, age (and birth date), schooling, and relationship to the head of the household. For each household member it was asked whether their father and mother were still alive and, if alive, whether they were living in the same household. If the latter was the case the line number of the parent had to be recorded. Finally, questions were asked about household sources of income (trade, off-farm

employment), household assets (radio, bicycle and motorized vehicle) and structure of the house (roof, floor). Households with no external source of income, no household assets and the cheapest house structure were classified as very poor.

An in-depth survey on orphans and foster-children was carried out among care-takers during 1995-1996. One rural village was initially selected for interviews using precoded and open-ended questions on child care and support in households with orphans or foster-children under the age of 15 years. In total 169 households were interviewed out of 216 listed households in this village, resulting in data on 300 orphans and foster-children (non-response rate 22%). In addition, 158 interviews were held with care-takers of double orphans and double foster-children in the whole ward. Information was obtained on 21 of 43 listed double orphans (non-response rate 51%) and 137 of 240 listed double foster-children (non-response rate 43%). The main reasons for non-response were that the child had moved since the baseline census or the care-takers were not available for interview. There were also cases of misclassification: children listed as orphan or foster-child during the census had living parents or were living with their parents (see discussion).

During 1994-95 a sero-survey was conducted among 5721 adults 15-44 years. Overall HIV prevalence in the ward was 6.2 per cent, but it was 10.3 per cent in the roadside village and 4.2 per cent in the five rural villages (Kisesa Sero-survey Team 1996).

Data from a demographic surveillance system were used to assess mortality and mobility among orphans and other children. Following the baseline census, four-monthly visits were made to each household and vital events were registered. If a death had occurred, an assistant medical officer visited the household for a verbal autopsy interview with the relative of the deceased. HIV serology was available from the sero-survey for 28 per cent of deaths. These data are used to compare the household conditions among HIV- and non-HIV-associated deaths (for details see Boerma et al. 1997).

Results

Prevalence of orphanhood and child fostering

Table 1 presents the prevalence of orphanhood by age of the child among 10,015 children in the baseline census. Overall, 8.9 per cent of children under 18 years and 7.6 per cent under 15 were orphans. There was a rapid increase in the proportion orphaned by age: from three per cent under five years to 18.1 per cent at 15-17 years. Of children under 18 years 5.5 per cent were paternal orphans, 2.5 per cent were maternal orphans and 0.9 per cent had no living parents.

Table 1
Orphanhood and child fostering by age of the child (%)

Parents' status	0-4	5-9	10-14	15-17	All
Mother died	0.7	2.1	4.3	4.4	2.5
Father died	2.3	5.1	7.3	11.3	5.5
Both died	0.1	0.5	1.8	2.5	0.9
Mother away	1.8	5.8	7.2	8.1	5.1
Father away	23.4	15.2	13.0	11.1	16.8
Both away	6.9	14.0	15.0	16.4	12.2
Both present	64.9	57.3	51.4	46.2	56.9
Number	3310	2927	2537	1241	10015

Among single-parent orphans it was common for the remaining parent to live elsewhere. About 58 per cent of the fathers of 246 maternal orphans and 32 per cent of the 371 mothers of paternal orphans were not living with their child. Thus, 40 per cent of the single-parent orphans did not have their surviving parent living with them.

Child fostering was very common. Among all children under 18 years 34.2 per cent were not living with one or both biological parents: 5.1 per cent did not live with their mother; 16.8 per cent did not live with their father and 12.2 per cent lived with neither parent. In other words, 17.3 per cent of children under 18 did not live with their mother. There were no differences in fostering and orphan rates between boys and girls.

Reasons for absence of a parent were obtained from the in-depth survey. The main reasons for the father not being with the child were: premarital child (37.1%), divorce (29.1%) and living with another wife (14.8%). The main reasons for the mother not being with the child were divorce (64.5%) and premarital child (11.4%). Work was seldom a reason for the parent not being present. The reasons were similar for single and double foster-children.

Household comparison

In Table 2 three types of household are compared: households with orphans, households with children under 18 but no orphans, and households with no children under 18 years. Among the 3353 households in Kisesa there were 470 households with at least one orphan (14% of all households). In such households there were on average 1.9 orphans. Forty-two per cent of all households had foster-children. One in five of these households with foster-children also had orphans. Orphan households had more children than other households with children (4.4 and 3.5 children respectively), but not more children under five years (1.2 in both).

Orphan households were larger than other households (7.9 and 6.3 persons respectively) and had a less favourable dependency ratio, which was defined as the sum of children under 18 and persons 60 years or older divided by the number of persons 18-59 years. The dependency ratio for all households was 1.33, for households with children 1.43 and for households with orphans 1.64. Only one household was headed by a child under 18 years. There were 110 households with no adult 18-59 years: 11.4 per cent of households without children, 1.3 per cent of households with children and 2.6 per cent of households with orphans had no adults.

Table 2
Composition and economic status of households (HH) with orphans, with children but no orphans and without children under 18, Kisesa, 1994

	HH with orphans	HH with children	HH no children	Total
Number and percentage of households	470 (14%)	2280 (68%)	603 (18%)	3353 (100%)
Mean N of orphans	1.9	0	0	0.3
Mean N of foster-children	1.5	1.2	0	1.0
Mean N of children	4.4	3.5	0	3.0
Mean N of adults 18-59	3.0	2.6	1.6	2.5
Mean N aged 60+	0.5	0.3	0.4	0.3
Total N of people	7.9	6.3	1.9	5.8
Dependency ratio	1.6	1.5	0.3	1.3
Per cent female headed	36.8	15.1	25.2	20.0
Per cent N adults 18-59	2.6	1.3	11.4	3.3

In one in every five households a female was considered the head of the household by its members. Among 36.8 per cent of the orphan households the head was a woman, compared to 15.1 per cent among other households with children. Among maternal orphans 16.3 per cent lived in a female-headed household, among paternal orphans 54.6 per cent and among double orphans 38.3 per cent.

There were no significant differences in economic status according to an index based on off-farm income, household assets, and structure of the house (data not shown in Table 2). Both the mean score and the proportion classified as very poor were similar in all three types of households.

Table 3
Care-takers for orphans and foster-children: percentage distribution by type of orphan/foster-child, Kisesa, 1995

	Other parent	Grand-parents	Aunt/ uncle	Brother/ sister	Other relative	Other	N
Maternal orphan	40.9	40.9	4.6	-	4.1	4.6	22
Paternal orphan	34.2	39.0	2.4	17.1	2.4	4.9	41
Double orphan	-	53.8	23.8	23.8	23.8	4.8	21
Maternal foster-child	77.4	22.6	-	-	-	-	31
Paternal foster-child	57.7	31.7	-	0.8	4.9	4.9	123
Double foster-child	-	71.0	10.8	5.1	11.7	1.4	214

Care-takers

Among the 21 double orphans in the in-depth survey the grandparents, older siblings, aunts, uncles and other relatives were all possible care-takers (Table 3). If the grandparents were the care-takers of double orphans, they were mostly from the mother's side (5/6). Among 63 single-parent orphans the surviving parent or the grandparents were the most frequent care-takers. Most grandparents were from the mother's side (68%). Also among 214 double foster-children (both parents living elsewhere) the grandparents were the most common care-takers (71 per cent). Most grandparents were from the mother's side (52% of all double foster-

children). Even for maternal and paternal foster-children grandparents were an important source of care. More than 95 per cent of all orphans and foster-children were taken care of by relatives.

Orphan care-taker patterns were examined by age and sex of the child, including whether the child was cared for by maternal or paternal family. No differences could be observed in type of child care-taker by age and sex.

A minority of care-takers of orphans received support from the surviving parent (if living elsewhere) or other relatives (Table 4). Foster-children more frequently received support from the parents living elsewhere. In particular, if both parents were living elsewhere, 70 per cent received some support from the parents. This was mostly assistance in obtaining clothes, school support and food, and financial support. No child in Kisesa received support from an organization.

Table 4
Support for orphan and foster-children from outside the household by parents and other relatives

	Number	Parental support (%)	Other support (%)
Maternal orphan	22	25.0[a]	9.1
Paternal orphan	41	20.0[b]	7.3
Double orphan	21	-	9.5
Maternal foster-child	31	22.6	6.5
Paternal foster-child	123	13.0	5.7
Double foster-child	214	69.6	10.3

[a] Among 12 maternal orphans whose father is living elsewhere

[b] Among 20 paternal orphans whose mother is living elsewhere.

Care-takers were asked whether the child had been sick in the last month and what had been done. A high proportion of both orphans (39.3 %) and foster-children (33.7 %) had reportedly been ill, and most children had been taken for some type of treatment. The proportions of children taken for treatment were somewhat higher among foster-children than among orphans (97% and 88% respectively, $p < .05$).

AIDS mortality

To assess the role of HIV/AIDS associated mortality the longitudinal data were used. During two years of follow-up 160 adult deaths occurred in the age group 15-59 years. Verbal autopsy interviews were conducted for 141 deaths and HIV/AIDS was considered a probable cause for 35 per cent of deaths (for details see Boerma et al. 1997). Forty-nine per cent of deaths were of women, and 56 per cent of HIV-associated deaths concerned women.

Table 5
Characteristics of deceased adult and household dependency ratio before death, Kisesa, 1994-95

Sex	HIV	Number	Mean age at death	Dependency ratio
Male death	HIV-related	20	38.7	1.7
	Non-HIV	50	32.4	1.0
Female death	HIV-related	28	*32.6*	1.2
	Non-HIV	43	*39.5*	1.6

Note: numbers in italics - difference significant at 5 per cent level using t-test.

The overall mean age at death for men was 34.2 years and for women 36.8 years. Table 5 compares the age at death and household composition by HIV status and sex of the deceased. Men's HIV-related deaths were at a later age than non-HIV-related deaths: 38.7 and 32.4 years respectively. Among women the opposite was observed: 32.6 and 39.5 years among HIV and non-HIV-related deaths respectively.

There were differences in the household dependency ratio before the adult death. During the baseline census the dependency ratio was higher in households with male HIV deaths than in households with non-HIV deaths (1.7 and 1.0 respectively). In households with female deaths due to HIV the dependency ratio was lower than in the households with deaths not related to HIV (1.2 and 1.6 respectively). These differences appear to reflect age differences of the deceased. Death of an older person, often a head of a household, leaves a household with a higher dependency ratio.

The proportion of new orphans due to HIV/AIDS mortality could not be computed because of the high proportion of children not living with their parents: not all children of the deceased parent can be identified.

Table 6
School attendance by age and sex

Age	Orphans		Foster-children		With both parents		Total	
Boys								
	N	%	N	%	N	%	N	%
6-8	69	8.7	325	7.1	503	5.0	897	6.0
9-11	94	53.2	278	46.8	412	51.0	784	49.7
12-14	107	*71.0*	255	*69.8*	382	83.5	744	77.0
15-17	113	*54.9*	242	*60.3*	306	69.0	661	63.4
Girls								
	N	%	N	%	N	%	N	%
6-8	64	12.5	294	8.8	522	8.2	880	8.8
9-11	86	53.5	254	51.6	423	55.3	763	53.9
12-14	101	78.2	267	*76.0*	362	82.6	730	79.6
15-17	112	44.6	201	*40.8*	267	55.4	580	48.3

Note: percentages in italics - significantly different at 5 per cent level from children living with both parents (chi-square test).

Schooling

Table 6 examines school attendance among orphans, foster-children and children living with their parents, by age and sex. Two indicators were chosen: proportion of children aged 13-17 years who never attended school and proportion who dropped out of school before completing seven years of education. The age at school entry in Tanzania is seven years, although the median age at school entry was nine years in Kisesa. Education is free though costs of uniforms, school materials and sometimes tuition have to borne by the care-takers. The schooling rates were very similar among the three groups. Among boys 13-17 years there were no differences between orphans and foster-children, but both had significantly lower enrolment and higher dropout rates than children living with both parents. Among girls enrolment was lower among orphans and foster-children compared to other children, but no significant differences could be observed for dropout rates.

In the in-depth survey the reasons for not being in school were explored. Among 41 children eight years and over the main reason given by the care-takers was that the child was considered too young (56%). Other reasons were health problems (10%), no place in school (10%), and no money (5%).

Mortality and migration

Table 7 presents mortality rates among orphans, foster-children and other children listed during the baseline census. In general, there were no significant differences in mortality between the three groups of children, but numbers in the orphan group were small.

Table 7
Annual mortality rates among orphans, foster-children and children living with both parents during two-year follow-up by age, Kisesa 1994-96.

	N	Deaths	PYO	Mortality rate
1-4 years				
Orphans	97	1	132	7.6
Foster-children	941	23	1301	17.7
Other	1858	48	2883	16.6
5-9 years				
Orphans	225	1	334	3.0
Foster-children	1025	5	1502	3.3
Other	1677	8	2695	3.0
10-17 years				
Orphans	564	2	833	2.4
Foster-children	1335	7	1932	2.3
Other	1879	4	3007	2.1

Orphans and foster-children were, however, more mobile than other children. During two years of follow-up 29.9 per cent of orphans present at the baseline census had moved to another household; 31.8 per cent of foster-children had done so; and 16.4 per cent of other children (difference of orphans and of foster-children with other children significant; $p < .001$). There were no clear differences in migration rates by age and sex of the child.

Discussion

In this rural population nine per cent of children under 18 years were orphans and 14 per cent of the households had at least one orphan. Child fostering was very common with 12 per cent

of children under 18 not living with either parent, and 17 per cent not living with their biological mother.

Underreporting of orphans and orphan households may be a problem in censuses and surveys (Foster et al. 1995). A comparison of orphan and fostering status during the baseline census and the in-depth interviews allows an assessment of the level of misclassification during the baseline census. Since the in-depth survey focused on children under 15 years the comparison was limited to these children. Among 102 children listed as orphans in the baseline census, 18 per cent were found during the in-depth interviews to have a living parent. All these children were foster-children, that is, at least one of their parents was living elsewhere. Among 396 reported foster-children whose care-takers were interviewed during the in-depth survey, seven per cent were not foster-children. Most of those who were not foster-children were found to be orphans (16 out of 22). If we assume that the baseline census was correct for children with both parents living with them in the households, the underreporting rate for orphans can be estimated from these figures. The orphan rate for children under 15 years was slightly underestimated: instead of 7.6 per cent, eight per cent of all children under 15 were orphans. The foster rate was overestimated: instead of 33.4 per cent of all children under 15, 32.3 per cent were foster-children.

The misclassification may partly be due to the use of a less knowledgeable respondent during the baseline census. Although the effects of misclassification on estimates of prevalence of orphanhood and fostering appear to be limited, the analysis of the consequences of orphanhood and child fostering may have been affected by this bias. Another bias may be caused by the high mobility of orphans which has led to low response rates in the in-depth survey. This may introduce a bias in the data on care-takers and support, as those orphans who move frequently may be in a different position from orphans who are in a more stable environment.

Paternal orphans were more common than maternal orphans. For instance, among children under 15, 4.7 per cent had lost their father, 2.2 per cent had no living mother and 0.7 per cent had lost both parents. These proportions are comparable to data from rural Uganda where 6.3 per cent were paternal orphans, 2.8 per cent maternal and 1.3 per cent double orphans (Kamali et al. 1996). There are more paternal than maternal orphans if mortality of fathers is higher than of mothers; or if fathers die at an age when they have more children under 15 than mothers have at the age when they die. Mothers in societies with high fertility have the highest number of children under 15 in their mid-thirties (first child at about 18-20 years), while fathers have the highest number of children under 15 in their early forties, assuming an age difference between marital partners of about seven years. In Kisesa paternal orphans were more common because of higher male mortality in the past, and not because deceased fathers had more children under 15 years than deceased mothers, as the mean age at death of adult women was 39 years and of adult men 33 years. The AIDS epidemic in Kisesa has reduced the mortality difference between men and women (Boerma et al. 1997) and this may increase the proportion of maternal orphans even more than paternal orphans. The age patterns of deaths due to AIDS pushed the mean age at death of women down and of men up, to the mid-thirties for both (Table 5). This implies that for both sexes more orphans per death can be expected. Two factors can mitigate the effect of AIDS on maternal orphan rates, also relative to paternal orphan rates. Fertility among women with HIV infection may be lower than that of other women (e.g. Boerma, Urassa and Isingo 1996; Gray et al. 1997). Male orphan rates will be less affected because HIV-positive men may have fathered children with women whose parity was not affected. In addition, children of HIV-infected mothers are at higher risk of mortality due to HIV than children of HIV-infected fathers, because of vertical transmission, which is less common among HIV-infected men as their wives may be discordant.

No satisfactory explanation can be given for the discrepancy between maternal and paternal orphan rates. In Uganda underreporting of maternal orphans was considered a possible explanation for the difference between maternal and paternal orphan rates (Kamali et al. 1996). This was thought to be due to divorce or separation causing the mother to return to her natal home without her children and to the tendency of men to remarry after the death of their wives, and subsequent reporting of the new wife as the biological mother. These arguments may only partly explain the difference. The Kisesa data on co-residence of children and biological parents indicate that in fact more fathers are not living with their children than mothers and underreporting of fathers' mortality may thus be at least equally probable. Data from another study in Uganda indeed show that men tend to remarry earlier and more frequently than women after the death of their spouse (Ntozi 1997), but it is still speculative whether the new wife will be reported as the biological mother. The Sukuma people, the dominant ethnic group in our study population, are known for relatively high rates of widow remarriage, although a large proportion still remains unmarried (Kirwen 1979:124). Polygyny may also play a role. Another wife may be regarded as the mother after the death of one of the wives.

The prevalence of orphanhood not only depends on the magnitude of the epidemic but also on the phase of the epidemic (Gregson et al.1994). It appears that the AIDS epidemic in Kisesa is still in a fairly early stage and that adult mortality and, consequently, the prevalence of orphanhood are likely to increase in the next few years. In nearby Mwanza town, however, HIV prevalence has been stable at 10-12 per cent among women attending an antenatal clinic since 1989 (Ministry of Health 1995), while HIV prevalence in Kisesa roadside village is similar to that in the town. In the rural areas of Kisesa HIV prevalence may increase slightly from its current 4.2 per cent, but annual incidence in rural Mwanza Region has been estimated as less than one per cent per year (Grosskurth et al. 1995). In this context it is not likely that the HIV/AIDS epidemic and orphanhood prevalence will reach levels as observed or anticipated in Mutare, Zimbabwe (Foster et al. 1995) or Rakai, Uganda (Konde-Lule et al. 1996). It is more likely that the situation will become very similar to Masaka, Uganda, where adult HIV prevalence was about eight per cent and orphan prevalence 10.4 per cent (Kamali et al. 1996). The relatively low rate of double orphans may also rise as the epidemic progresses.

Care-takers were more commonly from the maternal side. Traditionally, the Sukuma are patrilineal and the children belong to the father's lineage. Anthropological studies in the 1950s and 1960s, however, already showed that the Sukuma maintained considerable flexibility in their marriage system (Lang and Lang 1973; Varkevisser 1973). Several forms of marriage co-existed to allow flexibility in the payment of the brideprice. From our in-depth survey it became clear that payments of brideprice are often postponed and that children only belong to the father's lineage if the payment of the brideprice has been completed. Some respondents mentioned that a payment can be made for each child separately and that payments differ between sons and daughters. Similar findings were reported by Kirwen (1979) in the 1970s. A man may agree to the marriage of his daughter based on the promise of the payment of a brideprice, but if the husband dies before making these payments his lineage will have no legal claim to the children of the marriage.

It is very common to bring up children that are not own children, even though outside support is generally lacking or very limited. Only the majority of double foster-children, with both parents living elsewhere, received support from one or both parents. In general, respondents said that support is only given during funerals (often according to fixed tariffs) and calamities such as fire, a practice called *nzengo*. The concept that children belong to and are reared by the larger extended family still appears to be very important.

Reasons for fostering were only sketchily addressed in our study. Work predominantly from West Africa has shown the great variety of reasons for fostering (Isiugo-Abanihe 1985;

Bledsoe et al. 1988; Renne 1993;Castle 1995). These include marital dissolution, learning a trade or skill, assistance with household tasks, education, support during weaning, and desire for a child in the case of fertility problems. Several studies have shown adverse nutritional consequences of fostering (Oni 1995; Castle 1995),but there was generally considerable variation in health status among fostered children, which can partly be explained by differences in the reasons for fostering (Castle 1995). Children 'pulled' to another household, for example desired by an infertile woman, may be better off than children 'pushed' to another household, for example because of marital instability. Obviously orphans mostly belong to the latter category, and are thus more vulnerable. In the Kisesa study marital instability appears to be an important reason for children not being with their parents.

About one in seven households had orphans. The orphans appear to be added on to a household with children, making the households larger and the dependency ratio less favourable. Even though the economic status did not differ according to economic index, this may make these households more vulnerable. Furthermore, orphan households were much more likely to be headed by women, which may further increase their vulnerability. Further research in Kisesa is investigating the causes and consequences of women-headed households.

Mortality risks did not differ between orphans, foster-children and children living with their parents, but numbers were small for orphans: an increase in mortality at 1-4 years due to vertical transmission of HIV was anticipated but could not be observed. Also in Uganda mortality rates among orphans and other children were similar after excluding HIV-negative children (Kamali et al. 1996).

With regard to schooling, enrolment of boys and girls appeared to be lower among orphans and foster-children than among children living with both parents. School dropout rates were higher for boys, but not for girls. Orphans and foster-children were much more mobile than other children. This is perhaps not surprising, but still is important as it may increase the vulnerability of such children, while at the same time they are much harder to reach than non-mobile children. It is notable that no differences were found between boys and girls in schooling, mortality, use of health services, mobility or other indicators, in contrast with the results of some studies in West Africa (Bledsoe et al. 1988; Oni 1995; Castle 1995).

Discussions about the possible consequences of the millions of orphans in Africa can benefit from a careful consideration of the historical and socio-cultural context. This study indicates that the orphanhood problem in the era of AIDS needs to be considered in the context of previous high adult mortality, child fostering practices and poverty. Adult mortality rates and fertility are likely to have been high in the past and orphans have been a common consequence. The increased mortality due to the AIDS epidemic will now lead to a rapid increase in the numbers of orphans but occur in a context of well-established traditional coping mechanisms. Our study does not show major disadvantages for orphans and is in agreement with reports from Zimbabwe (Foster et al. 1995) and Uganda (Kamali et al. 1996). In all studies the extended family system appears to be able to absorb and care for the orphans with minor adaptations, such as more involvement of the maternal side of the family in a patrilineal system.

One of the main reasons for the relatively smooth absorption of large numbers of orphans by the extended families is the practice of child fostering. About one in three children in the Kisesa population did not live with at least one of their biological parents; 47.5 per cent of households had at least one foster-child or orphan (58% of all households with children). In such a society an orphan child is likely to be less special than in a context where virtually all children live with their biological parents. Almost five times more foster-children than orphans were brought up by grandparents. Schooling rates among older orphans were lower than among children living with their parents but not lower than among foster-children. A significant proportion of orphans were already not living with the deceased parent before the

death of the parent: the consequences of such parental deaths may be limited for the child. The Kisesa data however showed that chances of receiving some external support were lower for orphans than for foster-children.

The orphan problem also needs to be considered in the context of poverty. High dependency ratio and low income were common in many households. Households with orphans may be worse off than other households, but in support programs it will be difficult to focus on all households with orphans (apart from the large number of such households), because a larger number of children in other households will be worse off. For example, in Kisesa of the 3253 children living in households classified as very poor by our economic index only ten per cent were orphans, 37 per cent were foster-children and 53 per cent were children living in the same household as both parents.

Therefore, even though a considerable increase in orphans can be anticipated during the next decade, it is not probable that this population will have to develop radically different coping mechanisms, particularly in most rural areas where peak HIV prevalence among adults remains below ten per cent. The challenge, and probably the only feasible intervention, is to develop community-based support systems which focus on the most vulnerable households and extended families, using only limited external support.

References

Bledsoe, C.H., D.C. Ewbank and U. Isiugo-Abanihe. 1988. The effect of child fostering on feeding practices and access to health services in rural Sierra Leone. *Social Science and Medicine* 27: 627-636.

Boerma, J.T., M. Urassa and R. Isingo. 1996. Infertility and its association with sexual behaviour, STD and HIV infection in Tanzania. Paper presented at IUSSP conference on Innovative Approaches to the Assessment of Reproductive Health, Manila, 24-27 September.

Boerma, J.T., J. Ngalula, R. Isingo, M. Urassa, K. Senkoro, R. Gabone and E.N. Mkumbo. 1997. Levels and causes of adult mortality in rural Tanzania with special reference to HIV/AIDS. This volume.

Castle, S.E. 1995. Child fostering and children's nutritional outcomes in rural Mali: the role of female status in directing child transfers. *Social Science and Medicine* 40:679-693.

Foster, G., C. Makufa, R. Drew, S. Kambeu and K. Saurombe. 1996. Supporting children in need through a community-based orphan visiting program. *AIDS Care* 8:389-403.

Foster, G., R. Shakespeare, F. Chinemana, H. Jackson, S. Gregson, C. Marange and S. Mashumba. 1995. Orphan prevalence and extended family care in a peri-urban community in Zimbabwe. *AIDS Care* 7:3-17.

Gray, R.H., D. Serwadda, M. Wawer, et al. 1997. Reduced fertility in women with HIV infection: a population based study in Uganda. Paper presented at IUSSP Conference on the Socio-Demographic Impact of AIDS in Africa, Durban, 3-6 February.

Gregson, S., G.P. Garnett and R. M. Anderson. 1994. Assessing the potential impact of the HIV-1 epidemic on orphanhood and the demographic structure of populations in sub-Saharan Africa. *Population Studies* 48:435-458.

Grosskurth, H., F. Mosha, J. Todd, et al. 1995. Impact of improved treatment of sexually transmitted diseases on HIV infection in rural Tanzania: a randomized controlled trial. *Lancet* 346, 26 August:530-536.

Isiugo-Abanihe, U. 1985. Child fostering in West Africa. *Population and Development Review* 11: 53-74.

Kamali, A., J.A. Seeley, A.J. Nunn, J.F. Kengeya-Kayondo, A. Ruberantwari and D.W. Mulder. 1996. The orphan problem: experience of a sub-Saharan Africa rural population in the AIDS epidemic. *AIDS Care* 8:509-515.

Kirwen, M.C. 1979. *African Widows.* Maryknoll NY: Orbis Books.

Kisesa Sero-survey Team. 1996. Kisesa sero-survey 1994-1995. Report of basic findings. TANESA Internal Report Series No.8. Mwanza.

Konde-Lule, J.K., N. Sewankambo, M. Wawer and R. Sengonzi. 1996. The impact of AIDS on families in Rakai district, Uganda. Paper presented at 11th International Conference on AIDS, Vancouver. Abstract We.D.363.

Lang, G.O. and M.B. Lang. 1973. The Sukuma of Northern Tanzania. Pp. 224-233 in *Cultural Source Materials for Population Planning in East Africa*, Vol. 3, *Beliefs and Practices*, ed. A. Molnos. Nairobi: East African Publishing House.

Ministry of Health. 1995. *National AIDS Control Program HIV/AIDS.STD Surveillance*. Report No. 10. Dar es Salaam: Epidemiology Unit.

Ntozi, J.P.M. 1997. Widowhood, remarriage, and migration during the HIV/AIDS epidemic in Uganda. Paper presented at IUSSP Conference on Socio-demographic Impact of AIDS in Africa, Durban,3-6 February.

Oni, J.B. 1995. Fostered children's perception of their health care and illness treatment in Ekiti Yoruba households, Nigeria. *Health Transition Review* 5:21-34.

Renne, E.P. 1993. History in the making: an anthropological approach to the demographic analysis of child fostering in South-western Nigeria. Pp. 327-342 in *IUSSP General Population Conference, Montreal*, Volume 3.Liège: Ordina.

Varkevisser, C. 1973. The Sukuma of Northern Tanzania. Pp. 234-251 in *Cultural Source Materials for Population Planning in East Africa*, Vol.3, *Beliefs and Practices*, ed. A. Molnos. Nairobi: East African Publishing House.

World Health Organization (WHO). 1995. The current global situation of the HIV/AIDS epidemic. Global Programme on AIDS Memo 3 July. Geneva.

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Factors leading to the establishment of child-headed households: the case of Zimbabwe

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Abstract

This paper analyses factors associated with the establishment of 43 child- and adolescent-headed households in Manicaland, Zimbabwe. Such households result from the rapid increase in numbers of parental deaths leading to overburdening of the capacity of relatives to fulfil their traditional role of caring for orphans. Most children living in child and adolescent headed households have had both parents die in the preceding five years; many of them receive regular visits and support from relatives. Child-headed households represent a new coping mechanism in response to the impact of AIDS on communities. Community groups can help extended families to cope with the burden of orphans by encouraging the establishment of volunteer-based visiting programs to at-risk households and by channelling essential material support to destitute families.

The number of children being orphaned is rapidly increasing in communities with high rates of HIV infection; by mid-1996, it was estimated that nine million children had lost their mother to AIDS, with over 90 per cent of affected children living in sub-Saharan African countries (UNAIDS 1996). The epidemic is leading to a decreasing proportion of adults in the population and reduced incomes of affected households (Gregson et al. 1994; Leighton 1996:76). As a result of the impact of AIDS on communities, changes are taking place in care-giving arrangements for affected children (Foster et al. 1995); an increasing proportion of orphans are now in the care of the elderly and the very young (Foster et al. 1996; Saoke, Mutemi and Blair 1996:55). The emergence of households headed by children sometimes as young as 10-12 years old is one of the most distressing consequences of the epidemic.

The appearance of child-headed households in communities affected by AIDS is a recent phenomenon with cases noted in the late 1980s in the Rakai district of Uganda (WHO 1990; Alden, Salole and Williamson 1991) and Kagera region of Tanzania (Mukoyogo and Williams 1991). In 1991, such households were observed in Lusaka, Zambia (Ham 1992), Manicaland, Zimbabwe (Foster et al. 1995) and, for the first time, in six villages in the Masaka district of Uganda, where previously no such households had been noted (Naerland 1993). In the United States, cases of teenagers caring for younger siblings after deaths of parents from AIDS were reported in 1993/94 (Levine 1995:194).

In the Rakai district of Uganda, two per cent of orphans were living in households with a carer who was 18 years old or less and 97 per cent of orphan households had an adult of 17 years or more living in the household (UNICEF 1994; Nalugoda et al. 1997). Zambia and Uganda were estimated to have 3.8 and 2.4 per cent respectively of children under 15 years maternally orphaned by AIDS in 1995, increasing to 5.5 per cent and 3.5 per cent by the year 2000 (Michaels 1994). By 1996 in Zimbabwe, it was estimated that eight per cent of children under 15 years were motherless because of AIDS and this was projected to rise to 16-22 per cent by 2001 and 24-40 per cent by 2011 (Gregson et al. 1996).

It seems likely that once households headed by children start to appear in communities affected by AIDS, their numbers and relative proportion will both rise as the cumulative total of orphans continues to increase. Though it is often assumed that the presence of these households in communities implies that extended family methods of support have broken down, this assumption has not been validated since there have been no previous studies of

child-headed households. This study seeks to examine factors surrounding the establishment of such households, and the degree to which they are supported by relatives, and to explore reasons why relatives do not absorb orphans into their own families. Through better understanding of existing coping mechanisms, appropriate methods of support to children living in especially difficult circumstances may be developed.

The extended family and children

Traditional Zimbabwean Shona and Ndebele communities are built around a patrilineal kinship system. Members of the same patrilineage are grouped together, and the residential group is, or used to be, three to four generations. Traditional life is characterized by brotherhood, a sense of belonging to a large family and by groups rather than individuals. The extended family gives security and support and the members share many assets. Bourdillon (1991:26) writes:

> It used to be, and still is, the ambition of a man to gather around him a growing lineage of descendants and dependants who would act as a corporate body for economic purposes and also a united body in times of crisis or tension within the community.

Traditionally, marriage used to be not so much the linking together of two individuals as of two families. When marriage was decided upon, a brideprice in the form of a number of cattle was paid to the bride's family; the payment of brideprice led to the children becoming the responsibility of the father and his family. Brideprice also created a special bond between brothers and sisters. The receipt of brideprice by a family for their daughter's marriage enabled them to pay the brideprice for their son. The son's children thus had a special link with their paternal aunt, who had a unique role in their upbringing. Traditionally, the concept of a 'social' orphan did not exist in Zimbabwean societies. Biologically orphaned children were cared for by members of their extended family, especially by aunts and uncles who took on the care-giving functions of parents. Under certain circumstances, other adults within the extended family besides the biological parents were called 'mother' and 'father' (Arvidson 1996).

The extended family was the traditional social security system and its members were responsible for the protection of the vulnerable, care for the poor and sick and the transmission of traditional social values and education. In recent years, changes such as labour migration, the cash economy, demographic change, formal education and Westernization have occurred and have weakened the extended family. Labour migration and urbanization have led to a reduction in the frequency of contact with relatives and encouraged social and economic dependence; possessions are perceived as personal property and no longer belong to the extended family. Increased life expectancy and family size mean it is now not possible for an extended family of three or four generations to reside together; the diminishing availability of land makes it difficult for large families to be economically independent through subsistence agriculture. Education about social values is likely to be obtained from schools and interactions of children with their peers rather than through traditional mechanisms, which has lessened the ability of older people to exert social control over the younger generation. Brideprice is nowadays often a cash payment earned by the husband-to-be, rather than cattle and other possessions raised by members of his extended family; thus marriage itself has become more a contract between two individuals leading to weaker links between and within extended families.

Some traditional roles of the extended family have been modified whilst others have almost disappeared. In Zimbabwe, brideprice is still commonly practised, though its nature has changed. The imposition of brideprice with high monetary value has led to unions being

established without the payment of brideprice, unrecognized by relatives from either family; such unions are inherently less stable and the children from them may be deemed not to belong to either extended family. Widow inheritance by a brother of the deceased husband is nowadays rare, though the traditional practice may have been replaced by brothers retaining sexual access to widows whilst not customarily inheriting and supporting them as second wives (Drew, Foster and Chitima 1996). The fact that orphans are now being fostered by maternal rather than paternal relatives, especially in peri-urban areas, is symptomatic of the decline of traditional extended family practices (Foster, Makufa and Drew 1995).

In spite of this, it should be emphasized that the extended family remains the predominant caring unit for sick relatives and orphans throughout Africa (Ankrah 1993). In the past, the sense of duty and responsibility of extended families towards other members was almost without limits. Even though a family did not have sufficient resources to care for existing members, orphaned children were taken in. This was the basis of the assertion that traditionally, 'there is no such thing as an orphan in Africa'. Even during the current crisis, and despite the appearance of child-headed households, most orphans are cared for by a member of the extended family. Extended family systems of caring for orphans are adapting to changes taking place within society (Foster, Makufa and Drew 1995). These changes illustrate the strength, resilience and adaptability of extended family coping mechanisms.

Methods

Background

The progression of the HIV epidemic in Zimbabwe has been rapid. Anonymous unlinked surveillance of pregnant women in five urban and 17 rural antenatal clinics during 1992/93 found 21.2 per cent (1205/5679) to be HIV-positive (Ministry of Health 1994). A 1992 enumeration study of orphans in Mutare district found 12.8 per cent of the children in the area studied were orphaned; 50 per cent of recent parental deaths were ascribed to AIDS (Foster et al. 1995). A 1995 enumeration in the area of the current study found 14.7 per cent of children orphaned with one quarter of parental deaths having occurred in the preceding year (Foster et al. 1996).

The Families, Orphans and Children Under Stress (FOCUS) program was established in 1993 by Family AIDS Caring Trust (FACT) in Mutare, Zimbabwe. The program is implemented by local churches in four rural areas of Manicaland province with a population of some 35,000 people; orphan households were identified by 88 volunteer women living in villages in program areas and those families most in need were given priority for regular visiting; during 1996, an average of 1398 supportive visits per month were made to 798 needy orphan households. In the city of Mutare, with a population of about 160,000, FACT supervises a church home care program using 30 volunteers with approximately 500 visits a month to some 200 clients and a FOCUS program with 10 volunteer visitors. FOCUS program records were reviewed and households headed by adolescents and children were noted; volunteer visitors identified additional child- and adolescent-headed households for inclusion in the study. A majority of orphan households were identified by one rural program assessed in 1995 and it is likely that a high proportion of households headed by children and adolescents were identified using local knowledge of rural community visitors (Foster et al. 1996). Much underreporting of such households in urban areas was likely since the orphan support program functioning in the city of Mutare was at an early stage in its development.

Definitions

A household is one or more people who share cooking and eating arrangements. The household head is the person primarily responsible for the day-to-day running of the household, including child care, breadwinning and household supervision; if tasks were shared, an attempt was made to determine the person primarily responsible for these tasks. A child is a person under 18 years old. An adolescent is normally defined as a person 13-24 years old; the definition used in this paper for adolescent-headed households is a household headed by a person 18-24 years old who is not the biological parent of children in the household.

The prevalence of child-headed households is still fairly low and some exist only temporarily. Adolescents may leave adolescent-headed households and such households as a result become child-headed. As child household heads reach 18 years old, their households by definition become adolescent-headed. Although this study focused on child-headed households, those headed by adolescents were also studied in view of the associations and similar situations of these two household types. Households headed by adults over 24 years were also studied if they had previously been child- or adolescent-headed. Where households contained adults with little or no responsibility for the day-to-day running of the household, including those with adult dependants who were sick, disabled or elderly, they were termed 'accompanied' child- and adolescent-headed households to distinguish them from households consisting of unaccompanied children and adolescents.

Although difficulties exist in household definition and classification, it was thought important to draw attention to the growing number of vulnerable, unaccompanied children living in especially difficult circumstances by using the terms 'child-' and 'adolescent-headed households'.

Survey instruments

A questionnaire was developed covering basic demographic and socio-economic information. Interviews were conducted in Shona and answers were written in English. Surveys were carried out during a four-week period from February 1997. The oldest available child or adolescent living in the household was interviewed by trained research assistants; occasionally, information was obtained from adults present in the households. Questionnaires were piloted with households outside the program areas and then modified. All households interviewed received material support in the form of food and some received payment of primary school fees for out-of-school children. Further focus-group discussions with household heads and in-depth family studies with other relatives were planned for a later phase of this research project.

Consent

Consent was obtained from respondents who were informed about the nature of the research, assured of confidentiality and given the option to decline to answer any or all of the survey questions. An attempt was made to identify a legal guardian and obtain informed consent for all households where minors were respondents.

Results

A total of 60 households were interviewed; 17 households were excluded because they were found to be headed by a grandparent, mother or aunt. In 43 child- and adolescent-headed households (including 27 child heads and 16 adolescent heads), there were 15 adults over 24 years, 23 adolescents aged 18-24 years and 146 children under 18 years, an average of 0.3 adults, 0.5 adolescents and 3.4 children per household; there were nine children under five

years old of whom three were children of adolescent household heads. The median age of respondents was 16 years (range 10-80 years). In six households, information was obtained from grandparents or aunts. The minimum prevalence of child-headed households in the four rural areas was four per thousand households.

Household classification

Of 43 households, 27 were unaccompanied households consisting of children or adolescents caring for younger children; three were unaccompanied single child or adolescent households; 13 were accompanied child-headed households or adolescent-headed households which contained 15 adults. Seven accompanied households contained grandparents who were too ill or debilitated (4) or blind and old (3) to supervise the households; one household also contained a mentally retarded mother; two households contained aunts, one who was too ill to care for children and one who was living in the same household but was not responsible for the daily supervision of orphaned children; five contained adults 25-29 years old, the spouses of two adolescent household heads, two step-brothers lodging with relatives and a 26-year-old female who had previously been the household's adolescent head.

Four adolescent-headed households were established previously when the household head was under 18 years. One child-headed household was previously headed by an adolescent who later left the household. In the month before the study, one household became adult-headed when children moved temporarily to their grandmother's home after their roof collapsed during a storm; and one became adult-headed after the child head ran away, followed by another sibling, leaving two younger children who moved in with an aunt.

Five households headed by adolescents and none by children were identified in the urban area. One urban household consisted of an adolescent who was left living alone after three younger siblings moved to a relative's rural home. The fact that no child-headed and few adolescent-headed households were identified in the urban area may indicate a low urban prevalence of these households. Living costs are higher in urban areas which leads some to relocate to rural areas where food, accommodation and education costs are lower. Children and adolescents living by themselves in urban areas may be evicted from their property for non-payment of rent or because they are exploited and have difficulty retaining their residence when accommodation is in great demand. There were anecdotal reports of urban child or adolescent-headed households breaking up, with boys becoming ‘street kids’ or leaving to work on rural farms and girls taking up low-paid domestic employment.

Orphanhood

Using a definition of orphans as children under 18 years who have lost either parent, 41 out of 43 (95%) households contained orphans. There were two non- orphan child-headed households: a single mother left home leaving a 16-year-old daughter looking after her younger sister; and a 14-year-old son looked after his mentally retarded mother, three younger children of different, unknown fathers and a blind, old grandmother. There were 44 orphan families since three orphan households contained children from two orphan families. In nine cases the whereabouts of at least one of the parents was not known. Both parents had died among 30 of 35 orphan families; in 18 double-orphan families, the father's death preceded the mother's; in three families, both parents died in the same year; and in eight families, the mother's death preceded the father's death.One case had no information on date of death. Most deaths were recent: 84 per cent of maternal and 74 per cent of paternal deaths occurred in 1993-1996.

Household formation

The household head was female in 25 cases and male in 18 cases. In 40 households containing younger children, the household head was an older sister in 25 cases; an older brother in 14 cases; and in one case an uncle. Children as young as 9 and 11 years old were responsible for looking after younger children and heading accompanied and unaccompanied households (Table 1). Seventy-five per cent of child and adolescent-headed households were established in 1995/1996, possibly reflecting an increasing incidence or the fact that many such households do not last long. Four households were established in the year before the mother's death when children or adolescents assumed headship during the mother's terminal illness. Most households were established in the same year as the last parental death, this being the mother's death in 80 per cent of households.

Table 1
Age of household heads, duration and timing of assumption of headship in 43 households

	Type of household (head)			
	Child, unaccompanied	Child, accompanied	Adolescent	All
N	17	10	16	43
Mean age at assumption of headship (years)	14.1	10.5	19.1	15.5
Mean duration of headship (years)	15.5	3.2	1.8	1.5
Assumed headship same year as parental death (N)	13	7	12	32
Assumed headship at least one year after parental death (N)	1	1	3	5
No data on timing, assumption of headship, or no orphans (N)	3	2	1	6

Reasons why current child or adolescent became the household head

In 21 orphan households, children or adolescents became household heads during the terminal illness or immediately following the death of a parent, this being the death of the mother in 15 cases and of the father in six cases (Table 2). One child assumed headship after the mother deserted. Twenty-one households had a total of 24 household heads other than the parent. Most of these caregivers lived with the children for less than a year before they left, became sick or died. Child or adolescent-headed households were established after illness or death of grandparents, aunts or an unrelated household head in 14 out of 43 households leading to nine unaccompanied and five accompanied households with, in the latter case, grandparents or aunts remaining and children or adolescents taking over household supervision.

Table 2
Reasons for change in headship by characteristics of the previous household head among 43 child and adolescent-headed households (CHH/AHH).

	Mother	Father	Grand-parent	Sibling	Aunt	Other	Total

Death or terminal illness	15	6	3	1	2	1	28
Illness or incapacitation	0	0	4	0	1	0	5
Left because of illness	0	0	3	0	0	0	3
Left for other reasons[a]	1	0	0	8	1	0	10
Total	16	6	10	9	4	1	46[b]

[a] Other reasons include employment, education, marriage; deserted; moved in temporarily; replaced by older household head.

[b] Three CHH/AHH had two previous household heads.

Respondents were asked why relatives left children or adolescents living by themselves (Table 3). In 13 households, there was no known relative in the family able to care for the children. In one household, relatives left children alone because this was the mother's dying wish. In the remaining 29 households, a relative was available who might have been able to provide direct care for the children; but in 88 per cent of these households, the relative did not want to care for the children while in 32 per cent of households, the children did not want to move to the relative's household or the relative to move in with them. Relatives did not want to have the children living with them because the relatives had their own life to live, they had no space, they were in need of care themselves or they had no love for the children. In three cases, children did not move in with relatives because the children lived close to a relative who arranged to regularly visit them.

Table 3
Reasons why relatives left children or adolescents living by themselves (n=43, more than one response possible).

Reason	Number of responses
Relatives did not want children to move in with them	13
Relatives did not want to move in with children	11
Children did not want to move away from their home	8
Children wanted to stay together	3
Children did not want relative to move in with them	2
Mother's dying wish for children to stay by themselves together	1
No known relative	13
Not known or not answered	4

Extended family support

During the two years before the study, eight children under five years old, five children 5-9 years and four children 10-14 years left 13 households to stay with nine aunts, four grandparents, two sisters and one unspecified relative. One 14-year-old household head and his 13-year-old sister ran away from their household.

Sixteen households headed by children or adolescents received regular visits (more than once a month) by aunts (8), grandparents (2), sisters (1) or unspecified relatives (5); 11 received irregular visits (less than once a month) by relatives; three were known to have living relatives but received no visits from them. In those receiving irregular or no visits, relatives lived far away in 27 per cent of households. Relatives were unable to visit because they were too old in two cases. In the year before the study, 14 households received material support from relatives in the form of money (9), clothing (7), food (6) and school fees (5). Ten households known to have living relatives received no material support whilst there was no information on six households with living relatives.

Discussion

Although AIDS is only one of several factors leading to the changes being observed in traditional patterns of child care (Foster, Makufa and Drew 1995), it is undoubtedly the main factor predisposing to the establishment of child-or adolescent-headed households in this study. The AIDS epidemic is leading to a rapid increase in the number of double and maternal orphans. Previous studies have shown AIDS to be the underlying cause of most recent parental deaths in one area where this study was carried out (Foster et al. 1995). In a majority of these households, their establishment was associated with the second parental death which was usually that of the mother; 82 per cent of households contained children who had lost both mother and father and 89 per cent of parental deaths occurred in the preceding five years. In view of this characteristic pattern of parental deaths, it is likely that AIDS was the cause of death in a majority of study households. A number of other factors also predispose to the establishment of child-headed households: rapid increase in the number of parental deaths; death of one or both parents; reluctance of relatives to foster orphans; lack of contact of

relatives with children; death or sickness of a relative; presence of adolescents or older children able to care for younger children; preference of children to live in child-headed households; last wish of dying parent; death of single mother; and inheritance of residence by surviving children .

Uncles' and aunts' reluctance

There were 30 study households where a relative was known to be alive and in most (88%) of these households, it was stated that the relative did not want to care for the children. The preference of many parents should they be incapacitated is that their children be looked after by a same-generation relative such as an aunt or uncle (Foster et al. 1996). But many uncles and aunts are reluctant to foster relatives' children, possibly because of their concern that fostering relatives' orphans would result in a reduction of their own children's standard of living. If forced by economic circumstances to choose between their own and fostered children, they would tend to show preference towards the former. This course of action might lead to accusations against them of neglecting fostered children in their care by community members. Rather than risk such censure and in order to protect their own children, relatives may refuse to accept orphaned children into their family.

However, in 30 per cent of the households studied, relatives, mostly aunts, had taken at least one child from families in the study into their own households; most children under five years were fostered by relatives though older children were often not taken in. The main reason for refusal to take in relatives' children was probably economic. In many cases, aunts and uncles provided material support or regularly visited child-headed households; in some cases it was stated that relatives did not foster children in their own families because they lived nearby and instead had chosen to regularly visit. These are indications that in many cases, households headed by children or adolescents are a new expression of the extended family's coping mechanism rather than the result of children slipping through the extended-family safety net.

In some cases, relatives may consider themselves free of responsibilities towards orphans, even though they are closely related to the children. Relatives may not recognize the legitimacy of orphaned children, if, for example, a sister had children but was never married or if brideprice was never paid to her brothers; in such circumstances, they may feel justified in not providing support to her orphaned children after her death. Some relations have had little contact with a relative's family before the parent's death. A Kenyan study found that whereas families living below the poverty line tended to foster children, wealthier relatives, whom one might expect to be more able to foster relatives' children, maintained minimal links with orphans (Saoke et al. 1996:51). Some relatives may be concerned about fostering orphans when they suspect that the parent died from AIDS. They may fear contracting HIV infection from the children, or are afraid that bringing such children into their home may lead to stigmatization.

Distance and lack of knowledge of relatives about situation of children

There were 13 households in this study where respondents reported that they did not know any living relative. However the actual number of such households without living relatives is probably smaller, given the youth of the respondents in this survey. With families sometimes being separated by large distances, regular communication between family members may be difficult; as a result, close ties that formerly existed between family members have become weaker. Lack of assistance by relatives to child-headed households may be due to poor communication, with relatives simply not knowing about the desperate situations being faced by orphaned children living in difficult circumstances. Households which are separated by

large distances from their relatives such as migrant families and foreigners who have infrequent contact with their extended families are especially vulnerable in this regard (SafAIDS 1996). Barriers such as national borders make it especially difficult for extended families to fill their traditional roles of providing social support in times of difficulty.

Death or sickness of a relative

In this study, ten households were established following the death or illness of a grandparent and five followed death or illness of an aunt or unrelated household head. Three household heads who became ill moved out to be cared for by another relative while five continued living in the households.

When a parent, especially a mother, dies of AIDS, orphaned children often go to live with a grandmother, a practice referred to as 'skip-generation parenting' (Levine 1995:193). Orphans are often cared for by grandparents because there is no other relative willing or able to look after the children. Grandparent-headed orphan households are becoming increasingly common as a result of AIDS. In Zimbabwe, 125/292 orphan households (43%) were headed by grandparents; in Kenya, 41/152 (27%) were grandparent-headed whilst in New York, 25 out of 43 maternal orphans (58%) lived with a grandmother or an aunt (Foster et al. 1996; Saoke et al. 1996:55; Working Committee 1996:23). Grandparents are likely to be older and be more incapacitated than aunts and uncles. In Zimbabwe, 82/256 orphan caregivers (32%) were 60 years or older. During one year, in a population in Zimbabwe of some 11,000 people, three sibling-headed households became established following the death of a grandparent (Foster et al. 1996). Increasing numbers of child-headed households are likely to occur in the future through the death or sickness of relatives.

Adolescents learn child care during caregiver's illness

In four cases the households were established in the year before the last parental death. In such cases, households may be left by relatives to continue after the death of the parent because of the demonstrated ability of the adolescent to care for children appropriately during the parent's terminal illness. It is common for older children to take over parenting roles during prolonged parental illness due to AIDS. Adolescents learn responsibility, effective coping mechanisms and nurturing skills in this situation (Grodney 1994:140; Levine 1995:194). Were parents to die suddenly and unexpectedly, adolescents would have had no opportunity to take over care-giving responsibilities for younger children, forcing relatives to foster children. Similarly, if there were no adolescent caregivers in the household, relatives might feel forced to foster a relation's orphans.

Children's preferences and parent's dying wish

In this study, eight orphan families stated that they wanted to stay together and either declined to move to a relative's home or refused to have an unwelcome relative move in with them. In some cases, siblings in a family may choose to form child or adolescent-headed households. The children may desire to stay together as a family group rather than be split up between various relatives, or wish to stay living at their own residence in familiar surroundings, rather than change school, friends, home and neighbourhood. They may resist attempts of relatives to foster them in the relative's household, fearing maltreatment or because the relative only agrees to foster younger siblings. Orphaned children may be concerned about losing their inheritance rights to property and land if they are fostered. They may also be concerned about the possibility of neglect, abuse and exploitation by certain relatives. Urban children in particular may be concerned about their schooling being discontinued or a deterioration in

their standard of living should they be fostered by a poor rural relative. So instead, children may actively choose to stay living together in their own household rather than relocate to a poor, reluctant or abusive relative's home.

In one case, a child-headed household became established to fulfil promises made to a dying mother. Sometimes, adolescents have to make a deathbed promise to take care of young children and keep them together (Levine 1995:194; Foster et al. 1996). As a result of such promises, adolescents who might otherwise prefer to see the family fostered resist reasonable strategies for fostering suggested by relatives or child welfare authorities.

Death of a single mother

Child-headed households may be particularly likely to result from single-mother households. In this study, four such households resulted from the death of a single mother whose partner had left or deserted, and one resulted from a single mother leaving her teenage daughters to care for themselves. Increasing numbers of children in developing countries are being cared for in single-parent-headed households. In Zimbabwe, nearly one million children were born to divorced, widowed and never-married women (Central Statistical Office 1994). Some of the most vulnerable orphans are children of single mothers, especially if the mother was a prostitute. When a single mother becomes sick or dies, her children may be left in the care of grandparents. Because such orphans are from single-parent households, they may be neglected by other relatives who refuse to provide any support to the children because they consider them illegitimate.

Inheritance of residence by surviving children

The right of children to remain in their home after the death of their parents influences whether or not the family will continue to stay living together in their residence. In rural areas in Zimbabwe, the issue of land alienation by relatives seems not to be pronounced. However in a study of single-mother households in a rural area of Zimbabwe, whereas a majority of married and widowed women lived in their own accommodation, most divorced and never-married women rented accommodation or shared their house with relatives (Juliusdottir 1995); children of single mothers may be forced out of their accommodation after maternal death. In other African countries, the right of surviving children to continue living in their rural residence appears to be an important factor in determining whether child or adolescent-headed households become established. In urban areas in Zimbabwe where there is shortage of accommodation, it is difficult for children to hold on to their accommodation after parental death. Most people lived in rented accommodation in urban areas where this study was carried out. However, three out of five urban child-headed households lived in their own accommodation, suggesting that if children were living in rented accommodation before the parental death, it may be less likely for child-headed households to become established.

Conclusion

The new phenomenon of child-headed households appearing in communities affected by AIDS is an indication of saturation of traditional extended-family orphan coping mechanisms. Some communities may have better preservation of traditional coping mechanisms, such as those in remote rural areas with little urban migration and with lower life expectancy; the more traditional the community, the more capable it may be to cope with increasing numbers of orphans. Thus in an area of rural Mwanza, Tanzania, with an HIV prevalence of six per cent, eight per cent of children were orphaned but 32 per cent of non-orphaned children were fostered, not living with their biological parents, an indication that extended family coping

mechanisms were relatively intact; of 3353 households surveyed, only one child-headed household was identified (Urassa et al. 1997). High numbers of such households may indicate weakened traditional coping mechanisms or overwhelming of coping mechanisms by large numbers of orphaned children.

HIV prevalence rates in urban areas are higher than in rural areas in Zimbabwe. It is somewhat surprising that child-headed households in this study appeared to be less prevalent than in rural areas, notwithstanding differences in sampling methodology. In urban areas, a combination of social and economic factors hinders the establishment of such households. Prospective studies of households with risk factors predisposing to their being headed by children or adolescents might clarify whether child-headed households are being established in urban areas following parental death and the ways in which such households terminate.

When these households first occur in communities, they may be transient. Families take time to organize coping strategies in response to unaccompanied children. Child-headed households may disintegrate when children integrate into a relation's household, sometimes after a crisis when relatives who were previously equivocal agree to take in the children. Projections for Zimbabwe suggest that the current high orphan incidence rate of 2-3 per cent per annum has occurred since the early 1990s and is likely to continue until at least 2010 (Gregson et al. 1997). As the numbers of orphaned children multiply, it is likely that there will be increasing numbers of child-headed households; they will become less transient, existing for longer periods; and household heads will be younger.

The appearance of child-headed households does not necessarily mean that extended families are abandoning their responsibility to care for relations' children. This study demonstrates that among households with known relatives, most were receiving regular supportive visits and small amounts of material support from their extended family. In some cases, members of the extended family refused to take unaccompanied children into their households because they knew that a relative was living nearby who could provide support and supervision. A number of orphans were taken into a relative's household where they were being cared for, especially when the children were under five. In a minority of child-headed households it seemed that extended-family methods of support had broken down since such children received little or no support from relatives and appeared to be particularly vulnerable to exploitation as a result of destitution and lack of adult supervision. The numbers of such unsupported households are likely to increase dramatically in the future in the face of poverty as the number of new orphans increases and as grandparent-caregivers or aunts and uncles become sick and die.

Households headed by children or adolescents thus represent a new coping mechanism in response to the impact of AIDS on communities. Where these households exist, there are often relatives living nearby who are providing material support, supervision and regular visits; such relatives are often also struggling to bring up their own families. Community groups can help extended families to cope with the burden of orphans and prevent the breakdown of the extended-family safety net by encouraging the establishment of volunteer-based visiting programs to at-risk families, and by channelling essential material support to destitute families (Foster et al. 1997).

References

Alden, J.S., G.M. Salole and J. Williamson. 1991. Managing Uganda's orphan crisis. Kampala: Technologies for Primary Health Care (PRITECH) Project.

Ankrah, E.M. 1993. The impact of HIV/AIDS on the family and other significant relationships: the African clan revisited. *AIDS Care* 5:5-22

Arvidson, M.1996. Caring for children: orphans in the AIDS-affected in Zimbabwe. Program on Population and Development, Report No. 13. Lund, Sweden: University of Lund.

Bourdillon, M.F.C. 1991. *The Shona Peoples*. Gweru, Zimbabwe: Mambo Press.

Central Statistical Office. 1994. *National Report*. Harare.

Drew, R.S., G. Foster and J. Chitima. 1996. Cultural practices of orphaned families in the north Nyanga district of Zimbabwe. *Journal of Social Development in Africa* 11:79-86.

Foster, G., C. Makufa and R. Drew. 1995. Am I my brother's keeper? Orphans, AIDS and the extended family's choice of caregiver. Sociétés d'Afrique and SIDA Newsletter, October. Bordeaux.

Foster, G., C. Makufa, R. Drew, S. Kambeu and K. Saurombe. 1996. Supporting children in need through a community-based orphan visiting program. *AIDS Care* 8:389-403.

Foster, G., C. Makufa, R. Drew, S. Mashumba and S. Kambeu. 1997. Perceptions of children and community members concerning the circumstances of orphans in rural Zimbabwe. *AIDS Care* 9: 393-407.

Foster, G., R. Shakespeare, F. Chinemana, C. Makufa and R. Drew. 1995. Orphan prevalence and extended family care in a peri-urban community in Zimbabwe. *AIDS Care* 7:3-17.

Gregson, S., G.P. Garnett, R. Shakespeare, G. Foster and R. Anderson. 1994. Determinants of the demographic impact of HIV-1 in sub-Saharan Africa: the effect of a shorter mean adult incubation period on trends in orphanhood. Pp. 65-92 in *AIDS Impact and Prevention in the Developing World: Demographic and Social Science Perspectives*, ed. J. Cleland and P. Way. Supplement to *Health Transition Review* 4. Canberra: Australian National University.

Gregson, S., B. Zaba, G. Garnett and R. Anderson. 1997. Projecting the HIV/AIDS epidemic in southern Africa. Paper presented at IUSSP/University of Durban Conference on the Socio-demographic Impact of AIDS in Africa, 3-6 February, Durban.

Gregson, S., T. Zhuwau, R.M. Anderson and S. Chandiwana. 1996. *The Early Socio-demographic Impact of the HIV-1 Epidemic in Rural Zimbabwe*. Harare: Blair Research Institute.

Grodney, D. 1994. Programs for children and adolescents. In *AIDS and the New Orphans*, ed. B. O. Dane and C. Levine. Westport: Auburn House.

Ham, M. 1992. Children learning to be strong. *Africa South Magazine*, June:40-41.

Juliusdottir, M. 1995. Female-headed households in Zimbabwe: demographic characteristics and livelihood strategies. Program on Population and Development, Report No. 9. Lund: University of Lund.

Leighton, C. 1996. The direct and indirect costs of HIV/AIDS. Pp. 65-87 in *AIDS in Kenya: Socioeconomic Impact and Policy Implications*, ed. S. Forsythe and B. Rau. Arlington: Family Health International/AIDSCAP.

Levine, C. 1995. Today's challenges, tomorrow's dilemmas. In *Forgotten Children of the AIDS Epidemic*, ed. S.Geballe, J. Gruendel and W. Andiman. New Haven: Yale University Press.

Michaels, D. 1994. Projections of motherless AIDS orphans under 15 in six countries. In *Action for Children Affected by AIDS - Programme Profiles and Lessons Learned*. New York: World Health Organization/UNICEF.

Ministry of Health and Child Welfare. 1994. *Report of an HIV / STD and AIDS Surveillance Workshop, 7-8 July, 1994*. Harare.

Mukoyogo, M.C. and G. Williams. 1991. AIDS orphans, a community perspective from Tanzania. Strategies for Hope No. 5. London: Action Aid/AMREF/World in Need.

Naerland, V. 1993. AIDS-Learning to be more helpful. Kampala: Redd Barna.

Nalugoda, F., M.J. Wawer, J.K Konde-Lule, R. Menon, R. H. Gray, D. Serwadda, N. K. Sewankambo and C. Li. 1997. HIV infection in rural households, Rakai District, Uganda. This volume.

SafAIDS. 1996. Orphans on farms: who cares? An exploratory study into foster care for orphaned children on commercial farms in Zimbabwe. Harare: Southern Africa AIDS Information Dissemination Service.

Saoke, P., R. Mutemi and C. Blair. 1996. Another song begins: children orphaned by AIDS. Pp. 45-64 in *AIDS in Kenya: Socioeconomic Impact and Policy Implications*, ed. S. Forsythe and B. Rau. Arlington: Family Health International/ AIDSCAP.

UNAIDS. 1996. UNAIDS and UNICEF launch the 'Children in a World with AIDS' Initiative. Factsheet. 28 August. Stockholm.

UNICEF. 1994. *Action for Children Affected by AIDS - Programme Profiles and Lessons Learned*. New York: World Health Organization/UNICEF.

Urassa, M., J.T. Boerma, J. Z. L. Ng'weshemi, R. Isingo, D. Schapink and Y. Kumogola. 1997. Orphanhood, child fostering and the AIDS epidemic in rural Tanzania. This volume.

World Health Organization (WHO). 1990. Draft discussion paper on the care and support of children of HIV infected parents. 14 September. Geneva.

Working Committee on HIV, Children and Families. 1996. *Families in Crisis*. New York: Federation of Protestant Welfare Agencies.

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The impact of the African AIDS epidemic*

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The contemporary AIDS epidemic can be compared with the other major visitations of pestilence. In Europe 20 million or more people probably died during the Black Death in 1347-1351, and globally perhaps 20 million died during the 1917-1919 influenza epidemic. By the end of 1996 the world estimates for the AIDS epidemic were over 6 million dead and a further 23 million seropositive and nearly all certain of death. In numbers dying, the AIDS epidemic will certainly far exceed both other historic epidemics, although no-one knows the total mortality in Europe and Asia for the Black Death. The reason for the inevitability of greater mortality from AIDS is the open-endedness of the present epidemic. Both the previous epidemics just cited were over in three or four years, and it is this relatively short duration which was thought to characterize epidemics. In contrast, the first AIDS cases were identified in 1981, the result of infection mostly over the previous decade. Thus, the AIDS epidemic is already a quarter of a century old, and the level of infection is still climbing both globally and in the Third World. There is no evidence as yet about its likely duration or even whether it will become endemic in some parts of the world.

There are, however, contrasting aspects of the disease. World population is now three times its level in the early twentieth century and ten times that of the fourteenth century. Population growth rates in most of the Third World are now so high that even a huge rise in mortality may not cancel them out, and we are not yet certain that any country will experience a decline in population size because of AIDS. In comparison, the Black Death ravaged a near-stationary population, reducing Europe's numbers by perhaps one-third. The high population growth rates of the developing world have come about because of continuing high fertility together with declining mortality which has raised life expectancy even in sub-Saharan Africa to almost 50 years. Thus, the most pessimistic projection of the present epidemic does not show the expectation of life at birth falling to as low a level as 30 years in any sub-Saharan African country, while the influenza epidemic reduced India's expectation of life for the whole intercensal decade, 1911-1921, to 18.5 years.

Nevertheless, there is in Africa a contemporary AIDS epidemic which in its intensity and in its impact on the population can be likened to the plague. In its intensity it is quite unlike anything experienced by national populations outside sub-Saharan Africa, although some sectors of other populations, such as homosexuals in the United States, may have comparable experiences. This severe epidemic is identified in Table 1 and Map 1. The affected population is found in a long belt stretching from the Central African Republic and southern Sudan through Uganda, Rwanda, Burundi, Kenya and Tanzania to Malawi, Zambia, Zimbabwe, Botswana, South Africa and Namibia. The map, but not the table, identifies southern Sudan, which has been omitted from the table both because it is not a national population and because HIV testing is so poor as to be suggestive rather than definitive. In South Africa, KwaZulu-Natal has been disproportionately affected (and the major city in the table is not Johannesburg

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* In this concluding chapter, other chapters are referenced by providing only the author's name and no date. All other publications are referenced with the year of publication. Assistance has been provided by Jeff Marck, Wendy Cosford and Pat Goodall of the Australian National University's Health Transition Centre.

but Durban), while the rural populations of Kenya and Tanzania have the highest HIV levels in the western parts of those countries. Parts of central and northern Mozambique also appear to have HIV levels similar to those found in the contiguous parts of the main AIDS belt (*AIDS Analysis Africa* 1997:7). Some of the measures, especially rural ones, may be affected by the location of the studies. Thus the Tanzanian rural estimate comes from Mbeya, one of the worst affected parts of the country. The World Health Organization's (1995:356) estimate for the whole country implies a rural level under 10 per cent. M. Carael (personal communication 1997) challenges the Rwanda figures and reports that Ethiopia and Djibouti probably now qualify for inclusion in the main AIDS belt. He reports that Addis Ababa now records the seroprevalence level among pregnant women as nearly 15 per cent (the rural rate reported by Health Studies Branch 1997 for Jimma, a rural area, was almost nine per cent).

Table 1
Countries with the world's highest HIV seroprevalence levels in the general (low-risk) population (percentage of adult population)

	Seroprevalence levels				Dates of research reported in 1997 publication	
	Capital (or major city)		Outside capital (or major city)[a]		Capital (major city)	Outside capital (or major city)
	Dates of publication		Dates of publication			
	1993	1997	1993	1997		
Malawi	31.6	32.8	na	11.8	1995	1995
Botswana	8.8	32.4	4.1	16.0	1995	1994
Zimbabwe	18.0	32.0	12.8	16.0	1995	1993
Zambia	24.5	27.9	16.0	12.7	1994	1994
Rwanda	33.4	25.4	9.8	na	1995	-
Swaziland	2.3	21.9	na	na	1993	-
Burundi	19.9	20.0	1.6	1.8	1992	1992
Uganda	29.5	18.5	5.0	6.5	1995	1994
South Africa	1.7	18.2	na	6.4	1994	1994
Kenya	15.0	18.1	6.3	10.3	1996	1995
Namibia	2.5	17.6	na	10.3	1996	1996
Central African Republic	7.4	16.0	8.5	6.5	1993	1993
Tanzania	11.5	13.7	10.2	15.0	1995/6	1994

Source: Health Studies Branch 1993a, 1997

Note: [a]Mostly rural, but also including provincial towns.

The population belt shown on the map is the home of about 180 million people, or three per cent of the world's population, but is afflicted with the majority (around 55 per cent) of the world's HIV/AIDS. This situation may change as the epidemic spreads in Asia, or it may not if the methods of containment employed in Thailand are copied elsewhere in that continent, and if AIDS continues to intensify in the main AIDS belt of Africa. In any case, the main AIDS belt is clearly the proper locus for a contemporary study of the impact of the

disease, and this book does this with a particular concentration on Uganda, Tanzania, Kenya, Zimbabwe and Malawi.

Map 1
The AIDS Belt

No other part of sub-Saharan Africa is as much affected by the epidemic. Abidjan and Bobo Dioulasso in West Africa most closely resemble the major cities of the AIDS belt, but in 1995/96 each recorded a lower seroprevalence level than any of the cities listed in Table 1; in addition, there was some evidence that rural seroprevalence in Côte d'Ivoire was well below East and Southern African levels, while no data were available for rural Burkina. In much of West Africa seroprevalence was as low as one per cent in rural populations and three per cent or less in the cities. Nevertheless, sub-Saharan Africa as a whole probably accounted for almost two-thirds of the world's HIV/AIDS.

It is clear from Table 1 that there has been quite dramatic change within the AIDS belt over the last few years. The most unanticipated change is summarized in Table 2. In East Africa, which recorded the highest seroprevalence until 1993, the levels appear to have stabilized or fallen slightly in the cities, admittedly at the very high proportion of almost a quarter of the population. Seroprevalence is still slowly increasing elsewhere in these countries, but is only half the city level. In contrast, seroprevalence in Southern Africa has soared, overtaking East Africa. In the cities it has quadrupled to about one-quarter of the population, which may turn out to be the level of stability, while in provincial areas it has reached 16 per cent, a rural level that had not been anticipated on the basis of the earlier East African experience.

Table 2
Regional change between 1993 and 1997 seroprevalence publication (unweighted averages %)[a]

	East Africa[b]		Southern Africa[c]	
	Capital (or major city)	Outside capital (or major city)	Capital (or major city)	Outside capital (or major city)
1993	24.3	9.4	6.7	8.5
1997	22.7	11.1	24.4	16.0
Change	-1.6	+1.7	+17.7	+7.5

Notes: [a]Unweighted averages; countries omitted from comparisons if data for both dates not available.

[b]Uganda, Rwanda, Kenya, Tanzania, Zambia, Malawi (Central African Republic omitted on geographical grounds; Burundi omitted because no new data since 1993a).

[c]Zimbabwe, Botswana, Namibia, South Africa, Swaziland.

Sources: Table 1.

The levels in the cities are almost unbelievably high at one-quarter of the adult population. Given that the average period from infection to death is probably less than nine years in Africa, and that the adult age span covered by most of the testing is four times as long, the implication is that, if the epidemic maintains its present level over the next few decades, the majority of city people will face death from AIDS. The truth may be more complex than this, for the city may contain a substantial, if minority, population unlikely to be infected, while many of those infected may return to rural areas. Nevertheless, cities will continue to grow because the balance of rural-urban migration will continue to be towards the urban areas; indeed epidemic deaths are already opening up the urban job market. There is no certain evidence that the rural AIDS epidemic is self-sustaining rather than depending on continued reinfection from urban areas. In the Masaka longitudinal study in Uganda a high proportion of the persons identified for the first time as being seropositive had recently returned to the area, mostly from the towns (Nakiyingi 1995; Nunn et al. 1995). The experience of the TANESA project is that seropositive persons are unusually highly mobile and were probably also so before infection (Boerma et al.). In a 1990 study in Rakai, Uganda,

it was shown that not only were rural HIV levels lower than urban ones, but those of small rural agricultural villages were much lower than those of trading centres, villages, or small towns with a bus stop, a few shops and access to commercial sex (Health Studies Branch 1993b, Uganda section). This pattern looks like diffusion and the continued growth of the epidemic, but in Uganda and elsewhere it could be a stable situation with infection continuing to trickle from the larger to the smaller residential areas. The Ugandan situation is repeated in Tanzania where the TANESA project found seroprevalence levels of only 4.2 per cent in genuinely rural villages but ones of 10.3 per cent in the more commercial centres along the main road (Urassa et al.). The position is somewhat blurred by Boerma et al. reporting little difference in the death rates of the two populations, but this may be a time-lag phenomenon. Alternatively, AIDS may have negated a pre-existing mortality differential in that earlier the more accessible roadside population had benefited more from medical and public health services, as well as social change, and so had lower death rates.

The reason that the sub-Saharan African epidemic has such a potential for changing the whole of society is that it is the world's only almost exclusively heterosexual epidemic. While homosexuality, bisexuality and intravenous drug use are estimated to be the causes of 87 per cent of HIV infection in the United States, 80 per cent in Europe, and 65 per cent in Latin America, they account for no more than one per cent of the sub-Saharan African epidemic (Mann et al. 1992:117ff; Center for International Research 1994:7). In sub-Saharan African anal sexual intercourse is suppressed as being associated with witchcraft, and, while drug use is common, injected drugs are too expensive for nearly everyone. At least as many women as men are seropositive, and this also means massive vertical transmission to infants. In terms of heterosexual transmission, there are low levels of infection among girls from about 12 years of age, and among boys from some years later. The peak ages at death are in the twenties and thirties, thus removing the families' major earners and orphaning many young children. This is aggravated by the fact that, if one parent is infected, the other is also likely to become seropositive. The African epidemic is making a major assault on the family and society, and this has been the theme of this book. As Gregson and colleagues report, there will be all kinds of demographic ramifications. These will now be addressed.

Mortality

The obvious impact of the disease, and the reason that it is so feared, is that nearly everyone who is infected is doomed to die. There is a period of grace, or the latency period, but, although this postpones mortality, it also condemns those who know their condition to years of being figuratively on Death Row. Two modifying points should be made with regard to sub-Saharan Africa. The first is that few people know they are infected until they are in the final symptomatic stage, perhaps less than five per cent. Some never know because they die as the first symptoms develop. This may lead to greater peace of mind, and is a situation apparently preferred by most of the population, but it radically reduces the possibility of intervening to reduce the level of transmission. The second is that the duration of the latency and symptomatic periods is closer to the situation in the West of the 1980s - that is, before large-scale use of medication - than was once feared. Anzala and colleagues (1995) concluded that the latency period among a sample of Nairobi prostitutes was around four years. Modelling of the early Masaka longitudinal data, noted in an Annual Report, suggested a latency period of only 4.5 years, but, with more years of survival evidence, the estimate has now been raised to seven or eight years. This may seem surprising given the other assaults on the African immune system. Similarly, the early estimate of a symptomatic period of 4.5 months, compared with 12 months in the West in the 1980s, has now been superseded by a new figure of 8-9 months. This is supported in this book by a figure of 8.6 months from the TANESA project in Tanzania (Boerma et al.). If the latency and symptomatic periods had

been atypically short, this would have meant an unusually high death rate for a given prevalence level compared with experience elsewhere.

Not everyone has the same chance of infection. The situation is radically different from that of bubonic plague, influenza or smallpox. Indeed the sexually abstinent or those in what has come to be called a monogamous relationship (a misnomer because it is a description of a type of marriage and not a sexual regime) have no chance at all of being infected. We have already noted that it is more an urban than a rural disease. This book presents other findings on differentials. Carpenter et al. show the greater risk to the young, with half the seropositive females in the Masaka study being infected before reaching 25 years of age. Marriage, in spite of the fact that most wives cannot control their husbands' extramarital sexual adventures, provides some protection. Carpenter and colleagues report of the Ugandan women in the Masaka study that, although the great majority were married, wives accounted for only 53 per cent of the seropositive, compared with 35 per cent of those never married, 12 per cent of the divorced, and four per cent of the widowed. Indeed the divorced exhibited well over double the seroprevalence level of the whole group, perhaps because some resorted to commercial sex for support. Among pregnant women at antenatal clinics in Manicaland, Zimbabwe, single women were three times as likely to be seropositive as married women, and divorced women were five times as likely (Gregson et al. 1996:13ff.). These figures are, however, subject to a selection effect, for some married women became widows because their husbands died of AIDS, while others were separated or divorced because either they or their husbands had AIDS. In all these cases the women themselves were likely to be seropositive.

The implied AIDS mortality in the main AIDS belt is staggering. Blacker and Zaba show that in Kenya, even present HIV levels mean that one-third of those reaching adulthood in this population will die of AIDS. They show that the chance of dying of AIDS is related to both the seroprevalence level and the life expectancy prior to the epidemic. Kenya, with a life expectancy of 54 years, compared with an East African average of 47 years (United Nations 1966), has relatively low mortality from other causes at each age. Where seroprevalence is higher, in the Honde Valley of Zimbabwe, Gregson et al. (1996:13ff) calculate that 50 per cent of each cohort of women will die of AIDS before 50 years of age.

Such high mortality is also recorded by the rare vital registration systems. In the Honde Valley registered deaths have doubled since 1996 (Gregson et al. 1996:17ff), and the earlier decline in infant mortality has now been reversed. These mortality and HIV levels and trends will, if unchanged, result by 2006 in male and female life expectancies of 30 and 32 years respectively instead of the 55 and 59 years anticipated in the absence of AIDS (Gregson et al. 1996:32). Change before 2006 is hardly to be expected because the period of a decade covered by the projection is little longer than the latency period of those just infected when the projection was constructed.

There must remain a possibility that Manicaland is worse affected than Zimbabwe as a whole, and that Zimbabwe's experience will not be identical with that of Southern Africa as a whole. This may be a slim hope in the case of Botswana. Nevertheless, in the Mwanza district of Tanzania, Boerma and colleagues record a rise in mortality of one-third in the TANESA project area. It is greater in the high-risk age groups, and the mortality levels they report from males 35-44 and females 25-34 are those which might be anticipated in populations with a life expectancy of around 33 years (Coale and Demeny 1966, North Model Life Tables). The rise in morbidity and mortality may all be ascribable to AIDS even if the cause of death is recorded otherwise. Glynn et al. record that between 1986 and 1994 the incidence of tuberculosis in rural Malawi doubled because the HIV-positive were seven times as likely to develop tuberculosis as the HIV-negative.

It should be noted that the seroprevalence data in Table 1 are not nationally representative. The figures for the cities are likely to be closer to the truth than those for the

rest of the country which may represent only one or two areas. Of the eleven countries in the table with new seroprevalence data since 1993 for general populations outside the major city, only three carried out national studies, two more chose 4-6 districts across the country, one chose two districts, while for five countries the information is from a single district or location. Mortality data are no better, for vital registration is almost non-existent in sub-Saharan Africa. For national data we depend on surveys which may suffer distortions in the case of AIDS deaths because the epidemic may also have killed the relative who acts as an informant or have resulted in the break-up of the family or household which serves as the information-supplying unit in such studies. As a result there is probably a substantial underestimate of under-five years mortality in AIDS-affected sub-Saharan Africa (Bicego et al. 1997:54-57) and it appears that child mortality is now rising in Zambia, Zimbabwe, Kenya and Namibia. Timaeus and Nunn are more sanguine about the estimation of adult mortality, and conclude that the orphanhood method can be adjusted to provide reasonably accurate adult figures even in the AIDS belt.

The TANESA project has provided further information on how AIDS is interpreted and treated. Without being questioned specifically on the matter, 46 per cent of persons with symptomatic AIDS and 32 per cent of those suffering from other illnesses blamed witchcraft (Boerma et al.). Doubtless many of those who did not volunteer this information also held the same belief. This helps explain why three-quarters of all AIDS sufferers sought help from traditional healers, who specialize in the occult as well as in organic malfunctions, compared with a lower proportion of those with other disorders. Nevertheless, before death half had also visited hospitals, although only one-eighth died there. Experience from elsewhere in Africa suggests that many of these visits were made not because the patients anticipated the underlying cause would be eradicated but because they wanted relief from distressing symptoms and pain. The attempt to secure such alleviation impoverishes AIDS victims' families throughout the continent as they buy ever more medicines and analgesics. The proportion visiting hospitals is too low and varies too much through the region, to allow any estimate of seroprevalence levels, although hospitals are often the first institutions to warn of the arrival of the epidemic.

Fertility

The important new information is that HIV infection almost certainly lowers fertility through biological mechanisms. The lower fertility of the seropositive women compared with the seronegative ones had been reported for a cohort of women in Kinshasa, Zaire (Ryder et al. 1991; Batter et al. 1994), with fertility being about 25 per cent lower among seropositive women and with that gap increasing with age. But these women were told their serostatus. Those HIV-infected were encouraged not to become pregnant again and were assisted to practise family planning. Their level of contraceptive use was significantly above that of the seronegative although probably not enough to explain the full fertility differential. Nevertheless, it appears that the researchers were satisfied that the intervention had succeeded and that fertility had been curbed by contraception. Similarly, Gregson et al. (1996:27ff) reported that fertility had declined by one child per woman in Zimbabwe's Honde Valley which they ascribed at least partly to greater use of contraception, especially condoms, employed increasingly in an attempt to prevent HIV infection. Nevertheless, they appear to have suspected that HIV itself might be part of the explanation for fertility decline.

By 1997 Gregson et al. concluded that it was likely in Zimbabwe that HIV was causing fertility decline beyond that which could be ascribed to condom use and behavioural change. In this book, Carpenter and colleagues report similar findings from the Masaka longitudinal study in Masaka. From the 10,000 population in 15 villages, the experience of women 15-49 years of age has been followed for six years yielding 11,000 women-years of observation. The

fertility of seropositive women was, as a crude rate, 8.2 per cent below that of seronegative women. But, when the rate was adjusted for age and marital status, the difference widened to 25 per cent. Furthermore, the rate reached by seropositive women was boosted by relatively high fertility among 15-19-year-old women arising from their higher level of sexual activity. Ntozi and colleagues (1997:150) reported to a 1995 workshop in Mbarara that 1992 and 1995 surveys in six districts of Uganda showed total fertility rates 15 and 16 per cent lower in households where AIDS deaths had occurred than in ones where they had not.

Similar evidence was also presented to the January 1997 Durban conference by Serwadda and colleagues for Rakai, Uganda. There, their 1989-92 study showed lower fertility among seropositive women. However, they were dissatisfied by the limited range of controlling factors they had investigated, and so from 1994/5 they have conducted another study focusing on pregnancy levels. This revealed pregnancy levels among women with neither HIV nor syphilis of 21.4 per cent, merely with no HIV of 20.8 per cent, with HIV alone of 13.4 per cent, and with both HIV and syphilis of 8.5 per cent. The pregnancy level of seropositive women was 36 per cent below that of seronegative women. But, when the researchers adjusted for other relevant factors, this gap widened to 55 per cent, with asymptomatic seropositive women exhibiting 51 per cent lower fertility and symptomatic women 77 per cent lower.

These findings are likely to change radically our interpretation of the AIDS epidemic. The obvious change is that AIDS will almost certainly reduce population growth rates further than has been hitherto predicted. Population projections have so far been confined to the rise in mortality, but now will almost certainly have to incorporate declining fertility. The two factors combined will probably lead to estimates of very low rates of population growth in many East and Southern African countries. In fact, Gregson and colleagues believe that Zimbabwe, under the influence not only of HIV-induced rises in mortality and falls in fertility but also of a voluntary fertility transition achieved by contraception, may well experience declining population numbers in the coming decade. The same analysis could yet apply to Botswana, Namibia, Swaziland and KwaZulu-Natal. By April 1997, the Health Studies Branch of the US Bureau of the Census believed that declining population would by 2010 characterize Botswana and Zimbabwe (they did not cover Swaziland and KwaZulu-Natal) and, beyond Africa, Guyana (Stanecki and Way 1997:4).

A second implication is that some countries where the beginning of a voluntary fertility transition has been suspected may instead merely be the victims of an HIV-led fertility decline. This is especially the case where contraceptive use levels are low. A possible example is Zambia. Ongoing work by Gregson and Zaba suggests that fertility is likely to fall about one per cent for every two per cent rise in seroprevalence. In Zambia this would probably mean a decline in the total fertility rate of about seven per cent or half a birth per woman, a substantial part of the apparent fertility decline.

A third, and disconcerting, implication is that our knowledge and understanding of the African AIDS epidemic may be disastrously defective. To ascertain HIV levels, persons are required who represent the major society and who provide blood for testing as a matter of course. For the general or low-risk population in sub-Saharan Africa that population is now almost always pregnant women in antenatal clinics. Every one of the figures in Table 1 comes from this source (Health Studies Branch 1997:38-39). The women provide the blood for more general testing and are usually not told that HIV testing will be carried out on a sample. That sample is not linked to the women's records and consequently neither the women nor the clinic staff know the results for individuals. The use of these women to provide representative figures for the whole community is based upon the finding that the roughly equal numbers of women and men are seropositive in those African communities where general testing has been done. During the whole period of the Masaka project, the seropositive level of everyone over

13 years of age has been eight per cent while that of women 15-49 has been twelve per cent (Carpenter et al.). What we now have is a situation which suggests that pregnant women may not be representative of all women. If pregnancy levels among seropositive women are one-third below those of seronegative women, then the former will be unrepresented in antenatal clinics compared with the situation in the whole community. Where clinic samples show seropositive levels around 30 per cent, as they do in Blantyre (Malawi), Gaborone (Botswana), Harare (Zimbabwe) and Lusaka (Zambia), then the true level among the adults of those cities might well be 40 per cent. The figures for populations outside the city may not be 16 per cent, as in Botswana and Zimbabwe, but over 20 per cent.

Marriage

The AIDS epidemic might be expected to have some impact on marriage. Indeed, Gregson and colleagues emphasize that all demographic parameters are likely to change. Change in age at first marriage and proportions marrying should also affect fertility levels. That impact might be muted compared with societies outside the region but it should, nevertheless, be considerable; Gray and colleagues (1997) found a pregnancy level among unmarried women of reproductive age of only half that found among married women, 10.7 per cent compared with 21.5 per cent. Mukiza-Gapere and Ntozi (1995) reported that first marriage rates were down in Uganda as increasing numbers of young women put off marriage because of fear of AIDS. It may be that they believe they have greater control over the extra-union sexual activities of a boyfriend than of a husband, or it may be that some are now remaining virgins until marriage.

Widow remarriage is more likely to be affected. The inheritance of widows by the deceased man's brothers, and sometimes sons, has been widely practised in sub-Saharan Africa. The institution has been in decline for decades, but seems to have moved towards extinction in East Africa as a result of the AIDS epidemic. There is still, nevertheless, a strong belief that widows of reproductive age should remarry. Gregson and colleagues report of Zimbabwe that widows felt that they are less likely to remarry .Many who have been widowed by AIDS now, because men fear AIDS, die of the disease subsequent to their husbands' deaths. Of living women who had been widowed in the five years before the study, only 20 per cent remarried within three years of widowhood (the same level as found among divorcees). Ntozi (1997) reported that the majority of widows under 30 years of age leave their deceased husbands' homes on the deaths of the husbands, usually under pressure from the husbands' relatives, and that around one-third had remarried up until the time of his study. It is this enforced mobility that may result in spreading the epidemic. Widows who do not remarry may well spread the infection more than women who do, because they are probably more likely to have to resort to commercial sex for support.

However, the most certain conclusion that can be drawn about the impact of the AIDS epidemic on East African marriage is how few studies have been reported. We know little about resultant changes in ages at marriage or proportions marrying, and the same is true of non-marital unions. We know little about the remarriage of widows, distinguishing between AIDS-widows and other widows. The near-absence of this information is surprising and unfortunate, as well as omitting a necessary parameter for adequately modelling the effect of the epidemic. One of the problems is that even before the epidemic the nature of marriage was changing fast and the line between marriage and more temporary unions was becoming blurred (Parkin and Nyamwaya 1987).

Orphans

Much more attention has been devoted to orphans, although the situation is far from clear. The reason for this concern is that most adults dying of AIDS do so under 40 years of age when they are likely to have young dependent children. AIDS is not the only cause of parental death but for the early 1990s Foster and colleagues (1995) in Zimbabwe and Kamali and colleagues (1996:511) in Uganda calculated that around half of all parental deaths were due to AIDS, which is in line with what we know about changes in mortality in the age groups where young parents are found. The epidemic has doubled the rate of orphanhood. And that situation may not represent a ceiling, for Gregson et al. (1994:448-449) report that the ceiling is reached 20-30 years after the onset of the epidemic, when children under 15 years with deceased mothers may reach 11-18 per cent of the total population (with the new evidence on lower fertility among seropositive women, he is currently revising this figure downward). Caring for AIDS orphans can be a heart-rending task, in a region where one-third of these orphans are themselves likely to die from the disease during their first years of life. Indeed, by 1999 one-third of the under five years of age mortality in the main AIDS belt is likely to be attributable to AIDS (Preble 1990:672), but the proportion varies greatly between countries for it depends not only on the level of the AIDS epidemic but also on the pre-existing level of child mortality.

Such levels of orphanhood will place great stress on the society, although perhaps less than on any other major society in the world. The reason for this is the dominance of the extended family over the nuclear one in the sense that children are expected to make little distinction between their parents and their grandparents, their mothers and their aunts, or their fathers and their uncles. A child sent to live with grandparents or aunts and uncles (as children frequently are) who regarded this as unnatural, unfair or reason for complaint has traditionally been regarded as unnatural and certainly as having no grounds for complaint. Indeed, parents will rarely listen to complaints because they place a higher priority on remaining on good terms with the relatives who have fostered the children. The system remains intact because the fostered children usually expect to work hard and the fostering parents usually demand this.

Most fostering is to relatives. Determining its national scale has depended on the World Fertility Survey (WFS) and the Demographic and Health Survey (DHS). Most of their data are little affected by the AIDS epidemic. Page (1989:414ff) showed from the relevant six WFSs that 21 per cent of Lesotho children under 15 years of age were not living with their mothers and that regional levels ranged between 21 and 22 per cent in Côte d'Ivoire, 13 and 28 per cent in Ghana, 14 and 24 per cent in Cameroon, 9 and 17 per cent in Kenya and 9 and 14 per cent in Nigeria. With additional data from the DHSs, McDaniel and Zulu (1996) showed that 20-30 per cent of children were living away from at least one of their parents in Namibia, Botswana and Liberia at one extreme, less than 10 per cent in Kenya and Mali at the other, while the rest of the surveyed countries fell between 10 and 20 per cent. Those living away from both parents numbered 20-30 per cent in Namibia (and almost certainly in Botswana where the relevant question was not asked), under 10 per cent in Kenya, and 10-20 per cent everywhere else. In Namibia only 37 per cent of child-years were spent with both parents (and Botswana was probably not much higher) compared with 62 per cent in Malawi, 69 per cent in Tanzania and 71 per cent in Zambia.

The new data throw light on the determinants of fostering. Fostering is much more likely when the mother is young or unmarried or has few children. These categories overlap but a middle-aged grandmother often feels she is more capable of raising the family's grandchild than is her young daughter. The children of a woman whose current marital status is divorced are 40 per cent more likely to have been fostered than those of a currently married woman (McDaniel and Zulu 1996:20). That is not the whole story, for many more children are subsequently fostered when divorced persons remarry. The extended family takes the children

rather than let them jeopardize the new marriage or let the children be ill-treated by an unrelated step-parent. WFS and DHS data do not allow this phenomenon to be investigated. Children are more likely to be fostered as they grow older and are of more use to the foster parents, and somewhat more likely to be fostered if they are boys. They are more likely to be fostered out by urban residents, probably mostly rural-urban migrants who have left the children or the youngest ones behind, and by educated mothers who may well have a paid urban job. In short there are many reasons for fostering and the orphaning of children is only one of them. The institution is ancient, robust and founded on the concept of the real family being the extended one. Of 90 million children in the main AIDS belt, around 33 million at any one time are not living with both parents and around 15 million are living with neither. Of those not living with both parents, and if the Mwanza region of Tanzania is typical of the whole belt, perhaps four million are children with one parent dead from AIDS and under one million with both killed by the epidemic. These numbers may well double or even treble, and indeed would reach 11 and four million respectively if the pattern approximates that found in Manicaland, Zimbabwe by Foster and colleagues (1995:8). Nevertheless, they will still remain only a fraction of all fostered children.

Who are the foster-parents? Using WFS and DHS data, McDaniel and Zulu (1996:21) showed, averaging six countries, that just over half were grandparents, one-third other relatives, and one-seventh non-relatives. Two East African countries differed substantially from this pattern. In both Tanzania and Zambia around 80 per cent of foster-parents were grandparents while other relatives and non-relatives each accounted for about 10 per cent. Part of the explanation is a lower fostering rate in these two countries: one-third of the Namibia or Botswana levels and two-thirds of that in Malawi. In Zimbabwe, excluding the parent in the case of orphans with only one parent dead, Foster et al. (1996:395) found that 72 per cent of orphans were living with grandparents, 13 per cent with aunts and uncles, 7 per cent with siblings and 8 per cent with other relatives. This resembles the Tanzanian-Zambian pattern.

In this book, the pattern revealed by the TANESA project in Mwanza fits in with that found for the whole of Tanzania by DHS, with two-thirds or more being cared for by grandparents, and most of the balance being accounted for by other relatives. Less than one in twenty are with non-relatives (Urassa et al.). Even so, of households with children, only one-sixth have taken in any orphans, although the households average 1.5 orphans or one-third of all children in the household.

The Mwanza study finds that foster-children and orphans are less likely than children living with both biological parents to be at school, but the differences are not great (Urassa et al.). The mortality rate for orphans and foster-children is no higher than for other children. Foster et al. (1995:8) came to a similar conclusion about Zimbabwe, as did Kamali et al. (1996:511-512) about Uganda. This contrasts with reports from West African studies of less care and higher mortality among fostered children (Bledsoe et al. 1988; Oni 1995). The failure to detect the impact of vertical transmission probably shows that most deaths of this type occurred while both parents were still alive, partly because most occur at a very young age.

Extraordinarily, the evidence up to now is that the fostering system will probably accommodate the very great numbers of AIDS orphans. There will be exceptions, but the extensive networks of orphanages envisaged by some Western observers will not be needed. This conclusion was reached for Uganda by Hunter (1990:681). Nor is fostering a haphazard process. It is often organized by family meetings and by decisions allocating children to those relatives with the resources or household capacity to take them in. Foster reports other reasons emerging in Zimbabwe, but the child carers he reports are up to 18 years of age and not necessarily relatives. There are cases of households consisting only of children, with teenage heads, but in many cases the explanation is not a failure to find foster-parents, but an often desperate attempt to retain their father's land in the face of the attempts or desires of the dead

man's relatives to acquire it: sometimes those offering to foster. Foster and colleagues (1995:9) found that most fostering is now done by maternal relatives, and, apparently because the society is patrilineal, they concluded that this must be a new pattern brought about by the AIDS epidemic. This may well be wrong; among the patrilineal Yoruba of Nigeria most fostered children have for generations ended up with the mother's relatives.

The social effect

Perhaps the most extraordinary aspect of the African AIDS epidemic is its limited social and political effect. This is a disease which in a number of countries will be the cause of death of half the population. It will lower Zimbabwe's life expectancy by 2000 by 20 years to the level of half a century ago, and according to Stanecki and Way (1997:6) by 2010 to 35 years. It has increased the mortality level of adults in their prime, 20-40 years, to pre-modern levels. At any one time one-third of the people one meets in cities like Harare or Blantyre are infected and have at the most only a few years to live. The situation in the cities approximates that of European cities during the Black Death or Plague. The additional death rate because of the epidemic, up to ten per thousand annually in some countries, is of a similar magnitude to the experience of France during the First World War, an experience that traumatized the French. Yet East and Southern Africa are not traumatized. Governments are not threatened by accusations of mishandling the epidemic. Not a single protest demonstration has occurred. Life goes on in a surprisingly normal way. There has not even been any very marked change in sexual behaviour, and society is not dominated by government demands that there should be. There is no paranoia and little in the way of new religious or death cults. In some ways it is very impressive.

But, to the outsider it is also unnerving and some explanation is needed. The explanation is not that the epidemic mostly assails marginal groups. On the contrary, it is most intense in the cities, where the upper classes are attacked at least as much as the poor and uneducated. It is not that Africans are accustomed to disaster, and have until recently suffered from similar mortality levels so that the experience is not particularly startling. The majority of countries in the main AIDS belt have not suffered war or civil strife, and young adults have not experienced such mortality rates in living memory.

Some years ago the writer was one of the authors of a paper which addressed this puzzle, 'Underreaction to AIDS in sub-Saharan Africa' (Caldwell et al. 1992). The puzzle remains, and the suggested reasons still largely stand. Accordingly, the AIDS epidemic will only slowly be curbed by behavioural changes and may have decades yet to run. Some of the elements are a belief that death is not the end (Mbiti 1989:152), a product of both traditional religion and fervent Christianity or Islam. Furthermore, death will come at its proper time or when destined; a view that Moore (1968:57) claimed was the most persistent imagery in African poetry. It may be preordained or it may be caused by unnatural forces, usually harnessed to someone bearing malice towards the one who dies. In any case there is little that can be done to avoid death, and worrying about it or restraining sexual activity may undermine one's confidence and weaken one's resistance to ill-fortune and disease. Politicians remain somewhat sceptical about foreigners' statistics and projections, with some justification because neither is as secure as could be wished. They are also often ambivalent about whether sexual behaviour can or should be changed. Much of society, moulded by the institution of polygyny, does not believe that a man can be sexually satisfied by only one woman (cf. Orubuloye et al. 1997). The low level of demand by African governments for help has allowed the international community to respond very half-heartedly to the epidemic.

The future

The main AIDS belt is probably not typical of the whole of sub-Saharan Africa, and not a foretaste of the situation likely to develop in the whole region. A solely heterosexual epidemic is not easy to sustain, and indeed no such epidemic exists outside sub-Saharan Africa. The chance of transmission in one sexual act between two otherwise healthy persons except that one is HIV-positive is too low. The sub-Saharan African epidemic is explained by an unusual conjunction of circumstances. It depends on: (1) a considerable level of premarital and extramarital sexual relations, often with parallel partners, arising from the identification in traditional religion of female virtue with fecundity rather than virginity, and a belief, arising from the traditional practice of polygyny, that a man cannot be sexually satisfied by one woman over a lifetime; (2) a significant proportion of the non-marital male sexual activity being with prostitutes, partly because there is a widespread economic component in sexual relations and partly because of the substantial level of male migration; and (3) a poor health service that leaves untreated many sexually transmitted diseases (STDs) that act as cofactors or catalysts.

There is little evidence that these factors vary significantly across sub-Saharan Africa. Yet the intense epidemic has been confined to the East and Southern African main AIDS belt. This is not explained by the happenstance of earlier infection in East and Southern Africa than West and Middle Africa. There was self-sustaining HIV infection among high-risk groups (chiefly, in sub-Saharan Africa, prostitutes) across the continent at an early stage (Mann et al. 1992:896-898). Before 1985, infection had been recorded only in Rwanda, Tanzania and South Africa (predominantly in the latter in the white homosexual community). By 1986 it had been identified not only in Nairobi, Blantyre and Kampala, but also in Abidjan, Dakar, Accra and Cotonou. It was not until 1987 that it was identified in Lagos, but that was also the year when it was first found in Harare. The fact is that in most of West and Middle Africa the epidemic has just failed to take off in the general population. In the early 1990s the HIV level among the general population of Ghana, Nigeria and Senegal was probably around one per cent (and possibly much of that because of the unwitting inclusion of high-risk persons) and it was still at that level in the mid-1990s (Health Studies Branch 1993a, 1997). In Ghana this had occurred in spite of a quite massive return of infected prostitutes from Abidjan, Côte d'Ivoire, in the late 1980s and early 1990s (Anarfi 1993). In Zaire the level has probably remained under three per cent in spite of common borders with the Central African Republic, southern Sudan, Uganda, Rwanda, Burundi, Tanzania and Zambia.

The evidence is mounting that the extra factor, not very strong in itself but sufficient when added to the first three factors to make the decisive difference, is (4) that the main AIDS belt is populated by almost 200 million people in contiguous ethnic groups where males traditionally and generally still remain uncircumcised (Murdoch 1967; Bongaarts et al. 1989; Moses et al. 1990; Caldwell and Caldwell 1993, 1996; Caldwell et al. 1997). The epidemiological evidence linking seroprevalence levels to ethnic groups not practising male circumcision is convincingly strong. In the early 1990s it was only a prediction from this hypothesis that southern Sudan, northern Mozambique, Botswana and Namibia would witness a major rise in seroprevalence. That has now happened and it has occurred nowhere else. It seems likely that the main AIDS belt is now fully constituted (see Map 1).

Predicting the future course of the African and global epidemic is fraught with risk, but the following are a few hopefully informed guesses. The main AIDS belt will expand little or no further, and national seroprevalence levels as high as ten per cent will not develop elsewhere in Africa or the world. The epidemic may well intensify in the main AIDS belt, but in Uganda stability has probably been reached, at a moderate level for the AIDS belt, partly because some ethnic groups in the country practise male circumcision. Nothing comparable with the main AIDS belt will develop elsewhere in the world, even though elsewhere all

epidemics are catalysed by, as well as heterosexual transmission, much more infectious homosexual transmission and/or intravenous drug use. Thailand has contained its epidemic with a general population seropositive level under three per cent in Bangkok and probably under two per cent nationally. If faced with real crisis, most other Asian countries could probably follow Thailand's lead in achieving relatively low-risk sex in their brothels. There is a kind of minor AIDS belt outside Africa, constituted of sub-national populations around the Golden Triangle and practising high levels of intravenous drug use: far northern Thailand, northern Burma, the Hill States of northeastern India and possibly parts of southwest Yunnan in China. The evidence is sketchy and most cited figures are of high-risk groups.

The most pessimistic conclusion is that the far side of the African epidemic may be decades away. The epidemic is already at least two decades sold, and, as can be seen in Table 1, seroprevalence levels in most countries of the main AIDS belt are still rising. There is some evidence that HIV prevalence levels may at last be slowly declining in Uganda, but only among people under 25 years of age (Konde-Lule 1995).

As the epidemic stabilizes its full effect will be clearer. For much of the main AIDS belt the majority of deaths will be caused by the disease. One set of projections suggests that by the year 2010 Zimbabwe, Uganda, Zambia and Malawi will have a life expectancy of 35-36 years instead of 70 years in Zimbabwe and somewhat lower in the other countries, and a crude death rate of 29 per thousand population instead of five (Stanecki and Way 1997:2). This means that the worst is still to come. So far AIDS has killed over four million people in the main AIDS belt. Within a decade that number may die every two years, at the rate of 10 million per decade, a grim product of the continued rise in seroprevalence levels during the 1990s.

Just how far sub-Saharan Africa, in spite of its poverty, malnutrition and disease, is from a Malthusian situation is shown by the fact that even these massive additions to its mortality were not projected by the Center for International Research (1994) to bring population growth to a halt. This is explained by the fact that, while AIDS was projected to add up to 13 points per thousand to some countries' death rates in 2010, the rates of population increase in the first half of the 1990s were at least double that level in 10 of the 13 countries in the main AIDS belt (United Nations 1996). This in turn is explained by high birth rates, with a crude birth rate above 40 per thousand in the majority of AIDS belt countries and a total fertility rate above five in 10 of them.

It is possible that this picture has to be modified. First, fertility transition, explained by greater use of contraception, has begun in at least six of sub-Saharan Africa's 30 nations, and curiously the main AIDS belt contains all of them: South Africa, Zimbabwe, Botswana, Namibia, Swaziland and Kenya[1]. It is possible, but perhaps not likely, that a reaction to the intensification of the AIDS epidemic will halt this transition. Secondly, the new evidence that the AIDS virus itself reduces fertility alters the situation quite radically. Gregson and colleagues now believe that these two factors could result in a modest population decline in Zimbabwe within a decade. There are clearly also other subsequent candidates for such a possibility, Botswana, Namibia, Swaziland and perhaps Lesotho and KwaZulu-Natal. By April 1997 the US Bureau of Census's Health Studies Branch agreed with regard to Botswana, and did not consider the rest (Stanecki and Way 1997:4). Only one country outside the sub-Saharan African main AIDS belt was in the same category, Guyana, and that was not because its HIV levels were as high as those of the main AIDS belt but because fertility transition had left it little above replacement fertility.

Even if population growth continues, high mortality among the prime working age population would be expected to have a major economic impact. Many firms in Southern

[1] It is likely that transition has also begun in Lesotho and Ghana.

Africa are already training a second person for all skilled essential positions. But it is not clear that this is true in the agricultural sector, where people of all ages work. Barnett and Blaikie (1992) anticipated disaster in Rakai, Uganda, but this district is now prospering. The AIDS belt is, in terms of economic growth, performing better than the sub-Saharan African average, and where this is not so, as in Rwanda, Burundi and the Central African Republic, AIDS is not the explanation. Gregson (1997) suspects that the higher HIV levels in Zimbabwe, and by extension all of Southern Africa, arise from greater economic development which fosters more movement, more buses and trucks, greater urbanization and more husbands away from wives on business.

Given the importance of grandparents as foster-parents, especially in such parts of East Africa as Tanzania and Zambia, a question of critical importance would seem to be what happens when today's young and middle-aged adults, eroded by AIDS, move into old age. Population projections by Gregson et al. (1994:444) suggest that there may be no insuperable problem. Certainly the proportion of population 40-65 years of age will decline. But this is partly offset by a decline in numbers under 15 years of age resulting from the epidemic, and wholly offset by an increase in the proportion 15-40 years of age. Gregson et al. comment that there is a much greater apparent problem of dependency if we regard all population under 19 years of age as being economically and emotionally dependent than if we confine this group to those under 16 years (Gregson et al. 1994:441). What mitigates the problem is first the fact that the great majority of 16-18 year olds are now economically active and this will probably remain the case over the next two decades, and secondly that there is the possibility, or perhaps necessity, for less fostering to be done by grandparents and more by uncles and aunts. Given the way African extended family decision-making works, this transition will almost certainly take place gradually without anyone but social scientists even noting the change.

When will the epidemic begin to pass? There has been less change in sexual behaviour than most of us anticipated a decade ago, and there have been no massive national efforts to achieve such change. There have been educational programs, although modest in terms of the challenge and often focused most strongly on captive school children. Governments have been cautious about confronting young and middle-aged men. They suspect, as we found to be the case in Nigeria (Orubuloye et al. 1991), that many ordinary people regard governments and churches as being only too willing to use the AIDS epidemic to destroy their simple pleasures. It is probable that the main forces likely to change sexual behaviour are not government information programs, but acquaintance with AIDS deaths, especially personally but also through the media. Gregson and colleagues (Table 4) present evidence for Zimbabwe supporting the view that deaths from AIDS among a person's friends and relatives influence sexual behaviour. In Nigeria there has been a good government educational program, but research among prostitutes in Lagos showed they had little acquaintance with AIDS deaths, possibly because sick young women went home; and they had little fear of the disease (Orubuloye et al. 1994:113).

There is, for the first time, some evidence of changed sexual behaviour. The decline in seroprevalence among population under 25 years of age in Uganda suggests that something is happening among the young (Konde-Lule 1995). Certainly, adolescents and young adults in Uganda are increasingly worried about AIDS and the implications of sexual behaviour. A study of 1252 letters written between 1991 and 1994 to the health section of a Kampala newspaper showed that more than half focused on this concern (Asera et al. 1996:173). It is believed that young people in Uganda are keeping to a more limited number of partners, are increasingly apprehensive of relations with persons thought to have many partners, and are possibly, especially in the case of girls, starting their sex lives later. Pool and colleagues (1996) studied 752 male workers in a Tanzanian factory where there had been an AIDS intervention program. They found that most of the workers feared death and impoverishment

from AIDS and had accordingly limited their number of sexual partners, often to one, and tried to avoid commercial and other high-risk relations. They also found that the great majority were deeply suspicious of condoms and very reluctant to use them. The researchers attributed the change to the TANESA clinic's program in the factory and not to broader national programs. They concluded that the program had done nothing to reduce the men's strong desire to be promiscuous but had achieved behavioural change by intensifying their fears about the consequences (pp. 219-220). However, the workers were mostly married and in their thirties or older; it is possible that younger men would have been more willing to accept condoms.

The other possible weapon against AIDS is greater use of condoms, especially in high-risk situations. Pool and colleagues believe that aversion to them in Africa is too strong for their use to make any appreciable difference. The writer came to a less pessimistic conclusion, partly because of persistent rises in their level of use, admittedly from a very low base, shown by East African DHS studies, and partly from acquaintance with social marketing success in southern Tanzania (Caldwell 1995).

One might conclude that there are gradual behavioural changes which will probably eventually contain the epidemic. The most decisive will almost certainly be a growth of cautious behaviour in sexual relations, but this will probably be supplemented by an increasing use of condoms, especially outside marriage and other unions with some stability (cf. P. Caldwell 1995). Some experimental efforts are being made to follow the Thai model in encouraging their use in commercial sex.[2] Behaviour is changing partly because of the impact of informational programs and news reports in the media, although the latter are on a smaller scale than might be anticipated. The main cause, however, is undoubtedly prolonged association with AIDS deaths through the loss of friends and relatives and attendance at funerals. There will probably be some lasting effect on sexual mores, but the epidemic is unlikely to bring about widespread monogamous, sexually exclusive and companionate marriage.

If the epidemic is to be contained solely by behavioural change it will probably last for several more decades and kill in the main AIDS belt alone 50 million or more people. There may be a very different scenario if there is a major biomedical breakthrough, probably in the form of an affordable and effective vaccine. This is by no means certain. In the meantime more limited interventions may help to limit the number of deaths. Some attack is being made on sexually transmitted diseases which act as cofactors, although here again the scale of the effort is more modest than might have been anticipated. Programs have begun in Africa on the use of drugs to reduce vertical transmission from mother to foetus. There is much more doubt as to whether the drug 'cocktails' becoming available in the West to prolong the latency period will be cheap enough or able to fit in sufficiently with ordinary Africans' ways of life to have any sizeable impact.

This book, written primarily by demographers, contributes to our knowledge of the demographic and social impact of the epidemic. It brings into focus the recent development of the highest seroprevalence levels in Southern Africa. It shows how Africa has coped with an appalling onslaught of disease which threatens to continue for years. Perhaps most importantly, it has shifted our attention from an almost exclusive concern with mortality to the new concern with the impact of the disease on fertility and population growth rates. It has demonstrated the impact of the disease on orphans and their relatives and shown a society with an amazing ability to soldier on amid horrendous disaster. Probably that ability has also had the effect of increasing and prolonging the scale of the disaster.

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[2] In Nigeria by Eka Esu-Williams in Calabar (cf. Esu-Williams 1995) and I.O. Orubuloye in Ado-Ekiti.

References

Anarfi, John K. 1993. Sexuality, migration and AIDS in Ghana. Pp. 45-67 in Caldwell et al. 1993.

Anzala, Omu A., Nico J.D. Nagelkerke, Job J. Bwayo, Donna Holton, Stephen Moses, Elizabeth N. Ngugi, Jackoniah O. Ndinya-Achola and Francis A. Plummer. 1995. Rapid progression to disease in African sex workers with Human Immunodeficiency Virus type 1 infection. *Journal of Infectious Diseases* 171:686-689.

Asera, Rose, Henry Bagarukayo, Dean Shuey and Thomas Barton. 1996. Searching for solutions: health concerns expressed in letters to an East African newspaper column. *Health Transition Review* 6,3:169-178.

Barnett, Tony and Piers Blaikie. 1992. *AIDS in Africa: Its Present and Future Impact.* London: Bellhaven Press.

Batter, Véronique, Baangi Matela, Malanda Nsuami, et al. 1994. High HIV-1 incidence in young women masked by stable overall seroprevalence among childbearing women in Kinshasa, Zaire: estimating incidence from serial seroprevalence data. *AIDS* 8,6:811-817.

Bicego, George, Siân Curtis, Hendrik Raggers, Saidi Kapiga and Sylvester Ngallaba. 1997. *Sumve Survey on Adult and Child Mortality, Tanzania, 1995: In-Depth Study on Estimating Adult and Childhood Mortality in Settings of High Adult Mortality*. Calverton MD: Macro International.

Bledsoe, Caroline H., Douglas Ewbank and Uche Isiugo-Abanihe. 1988. The effect of child fostering on feeding practices and access to health services in Sierra Leone. *Social Science and Medicine* 27:627-636.

Bongaarts, John, Priscilla Reining, Peter Way and Francis Conant. 1989. The relationship between male circumcision and HIV infection in African populations. *AIDS* 3:373-377.

Caldwell, John C. 1995. Understanding the AIDS epidemic and reacting sensibly to it. *Social Science and Medicine* 41,3:299-302.

Caldwell, John C. and Pat Caldwell. 1993. The nature and limits of the sub-Saharan African AIDS epidemic: evidence from geographic and other patterns. *Population and Development Review* 19,4:817-848.

Caldwell, John C. and Pat Caldwell. 1996. The African AIDS epidemic. *Scientific American* 274, 3:40-46.

Caldwell, John C., I.O. Orubuloye and Pat Caldwell. 1992. Underreaction to AIDS in sub-Saharan Africa. *Social Science and Medicine* 34,1:1169-1182.

Caldwell, John C., I.O. Orubuloye and Pat Caldwell. 1997. Male and female circumcision in Africa: from a regional to a specific Nigerian examination. *Social Science and Medicine* 44,8:1181-1193.

Caldwell, John C., Gigi Santow, I.O. Orubuloye, Pat Caldwell and John Anarfi (eds). 1993. *Sexual Networking and HIV/AIDS in West Africa*. Supplement to *Health Transition Review* 3. Canberra: Australian National University.

Caldwell, Pat. 1995. Prostitution and the risk of STDs and AIDS in Nigeria and Thailand. Pp. 167-172 in Orubuloye et al. 1995.

Center for International Research, US Bureau of the Census. 1994. *The Impact of HIV/AIDS on World Population*. Washington DC.

Coale, Ansley J. and Paul Demeny. 1966. *Regional Model Life Tables and Stable Populations*. Princeton: Princeton University Press.

Esu-Williams, Eka. 1995. Sexually transmitted diseases and condom interventions among prostitutes and their clients in Cross River State. Pp. 223-228 in Orubuloye et al. 1995.

Foster, G., C. Makufa, R. Drew, S. Kambeu and K. Saurombe. 1996. Supporting children in need through a community-based orphan visiting programme. *AIDS Care* 8,4:389-403.

Foster, G., R. Shakespeare, F. Chinemana, H. Jackson, S. Gregson, C. Marange and S. Mashumba. 1995. Orphan prevalence and extended family care in a peri-urban community in Zimbabwe. *AIDS Care* 7,1:3-17.

Gregson, Simon, Geoffrey P. Garnett and Roy M. Anderson. 1994. Assessing the potential impact of the HIV-1 epidemic on orphanhood and the demographic structure of populations in sub-Saharan Africa. *Population Studies* 48,3:435-458.

Gregson, Simon, Tom Zhuwau, Roy M. Anderson and Stephen Chandiwana. 1996. The early socio-demographic impact of the HIV-1 epidemic in rural Zimbabwe: summary report of findings from the Manicaland study of HIV-1 and fertility. Harare: Blair Research Institute.

Gregson, Simon. 1997. Statement at Conference on the Sociodemographic Impact of AIDS in Africa, Durban, 3-6 February.

Health Studies Branch. 1993a. Recent HIV seroprevalence levels by country: December 1993. Research Note 11. Washington DC: Center for International Research, US Bureau of the Census.

Health Studies Branch. 1993b. Trends and patterns of HIV/AIDS infection in selected developing countries. Research Note 12. Washington DC: Center for International Research, US Bureau of the Census.

Health Studies Branch. 1997. Recent HIV seroprevalence levels by country: January 1997. Research Note 23. Washington DC: International Programs Center, Population Division, US Bureau of the Census.

Hunter, Susan S. 1990. Orphans as a window on the AIDS epidemic in sub-Saharan Africa: initial results and implications of a study in Uganda. *Social Science and Medicine* 31,6:681-690.

Kamali, A., J.A. Seeley, A.J. Nunn, J.F. Kengeya-Kayondo, A. Ruberantwari and D.W. Mulder. 1996. The orphan problem: experience of a sub-Saharan Africa rural population in the AIDS epidemic. *AIDS Care* 8, 5:509-515.

Konde-Lule, Joseph K. 1995. The declining HIV seroprevalence in Uganda: what is the evidence? Pp. 27-33 in Orubuloye et al. 1995.

Mann, Jonathan M., Daniel J.M. Tarantola and Thomas W. Netter (eds). 1992. *AIDS in the World.* Cambridge MA: Harvard University Press.

Mbiti, John S. 1989. *African Religions and Philosophy.* 2nd edition. Oxford: Heinemann.

McDaniel, Antonio and Eliya Zulu. 1996. Mothers, fathers and children: regional patterns in child-parent residence in sub-Saharan Africa. *African Population Studies* 11:1-28.

Moore, Gerald. 1968. The imagery of death in African poetry. *Africa* 38,1:57-70.

Moses, Stephen, Janet E. Bradley, Nico J.D. Nagelkerke, Allan R. Ronald, J.O. Ndinya-Achola and Francis A. Plummer. 1990. Geographical patterns of male circumcision practices in Africa: association with HIV prevalence. *International Journal of Epidemiology* 19:693-697.

Mukiza-Gapere, Jackson and James P.M. Ntozi. 1995. Impact of AIDS on marriage patterns, customs and practices in Uganda. Pp. 201-208 in Orubuloye et al. 1995.

Murdock, George P. 1967. *Ethnographic Atlas.* Pittsburgh: University of Pittsburgh Press.

Nakiyingi, Jessica. 1995. Analysis carried out in graduate program, Health Transition Centre, National Centre for Epidemiology and Population Health, Australian National University, Canberra.

Ntozi, James P.M. 1997. Widowhood, remarriage and migration during the HIV/AIDS epidemic in Uganda. Paper presented to the Conference on the Sociodemographic Impact of AIDS in Africa, Durban, 3-6 February.

Ntozi, James P.M., Immaculate M. Nakanaabi and Yovanni A.M. Lubaale, 1997. Fertility levels and trends in the face of the AIDS epidemic in Uganda. In *Vulnerability to HIV Infection and Effects of AIDS in Africa and Asia/India*, ed. J.P.M. Ntozi, J.K. Anarfi, J.C. Caldwell and S.K. Jain. Supplement to *Health Transition Review* 7. Canberra: Australian National University.

Nunn, A.J., H.-U. Wagner, A. Kamali, J.F. Kengeya-Kayondo and D.W. Mulder. 1995. Migration and HIV-1 seroprevalence in a rural Uganda population. *AIDS* 9:503-506.

Oni, Jacob Bamidele. 1995. Fostered children's perception of their health care and illness treatment in Ekiti Yoruba households, Nigeria. *Health Transition Review* 5,1:21-34.

Orubuloye, I.O., John C. Caldwell and Pat Caldwell. 1991. Sexual networking in the Ekiti District of Nigeria. *Studies in Family Planning* 22,2:61-73.

Orubuloye, I.O., John C. Caldwell and Pat Caldwell. 1997. Perceived male sexual needs and male sexual behaviour in southwest Nigeria. *Social Science and Medicine* 44,8:1195-1207.

Orubuloye, I.O., John C. Caldwell, Pat Caldwell and Shail Jain (eds). 1995. *The Third World AIDS Epidemic*. Supplement to *Health Transition Review* 5. Canberra: Australian National University.

Orubuloye, I.O., Pat Caldwell and John C. Caldwell. 1994. Commercial sex workers in Nigeria in the shadow of AIDS. Pp. 101-116 in *Sexual Networking and AIDS in Sub-Saharan Africa: Behavioural Research and the Social Context*, ed. I.O. Orubuloye, John C. Caldwell, Pat Caldwell and Gigi Santow. Canberra: Australian National University.

Page, Hilary. 1989. Childrearing versus childbearing: coresidence of mother and child in sub-Saharan Africa. Pp.401-441 in *Reproduction and Social Organization in Sub-Saharan Africa*, ed. Ron J. Lesthaeghe. Berkeley:University of California Press.

Parkin, David J. and David Nyamwaya (eds). 1987. *Transformations of African Marriage*. Manchester: Manchester University Press.

Pool, Robert, Mary Maswe, J. Ties Boerma and Soori Nnko. 1996. The price of promiscuity: why urban males in Tanzania are changing their sexual behaviour. *Health Transition Review* 6,2:203-221.

Preble, Elizabeth A. 1990. Impact of HIV/AIDS on African children. *Social Science and Medicine* 31,6:671-680.

Ryder, Robert W., Véronique L. Batter, Malanda Nsuami, Nsanga Badi, Lubaki Mundele, Baangi Matela, Mulenda Utshudi and William L. Heyward. 1991. Fertility rates in 238 HIV1-seropositive women in Zaire followed for 3 years post-partum. *AIDS* 5,12:1521-1527.

Stanecki, Karen A. and Peter O. Way. 1997. The demographic impacts of HIV/AIDS: perspectives from the *World Population Profile: 1996*. IPC Staff Paper 86. Washington DC: International Programs Center, US Bureau of the Census.

United Nations. 1996. *World Population 1996*. Data sheet. New York.

World Health Organization. 1995. *Weekly Epidemiological Record*. 70, 50, 15 December.