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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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)

	Seroprev	alence leve	els	Dates of research reported in 1997 publication		
	Capital (or major city) Dates of publication		Outside capital (or major city) ^a Dates of publication		Capital (major city)	Outside capital (or major city)
	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: ^aMostly 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

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disease, and this book does this with a particular concentration on Uganda, Tanzania, Kenya, Zimbabwe and Malawi.

Map 1 The AIDS Belt

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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 %)

	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:^aUnweighted averages; countries omitted from comparisons if data for both dates not available.

^bUganda, Rwanda, Kenya, Tanzania, Zambia, Malawi (Central African Republic omitted on geographical grounds; Burundi omitted because no new data since 1993a).

[°]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

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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 Kenya¹. 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 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 highrisk 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 diseaster.

² In Nigeria by Eka Esu-Williams in Calabar (cf. Esu-Williams 1995) and I.O. Orubuloye in Ado-Ekiti.

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