THE IMPACT OF HIV-1 ON FERTILITY IN SUB-SAHARAN AFRICA: CAUSES AND CONSEQUENCES

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INTRODUCTION

The mortality impacts of the HIV epidemic in sub-Saharan Africa are profound - adult mortality has doubled or trebled even in communities in which prevalence remains below 10 per cent (Timaeus, 1998; Boerma and others, 1998), and life expectancy in many countries is falling rapidly. The social and demographic consequences of this unprecedented mortality increase have been the main focus of demographic research on the epidemic, but in this paper we turn to a field which has received less attention: the links between HIV and fertility.

Rationale for the study of HIV and fertility

HIV epidemics have become a significant influence on fertility in badly affected areas of sub-Saharan Africa. Population-based surveys in south-western Uganda (Gray and others, 1997) and analyses of data from antenatal clinics in a number of other countries show 25-40 per cent lower fertility in women with HIV (Zaba and Gregson, 1998). While some of this sub-fertility reflects prior disposition to other sexually transmitted infections (STIs) among HIV incident cases (Ross and others, 1999), about half results directly from the infection itself. Furthermore, persuasive evidence for extensive changes in fertility-related behaviour resulting from HIV epidemics and measures taken to control the spread of infection is emerging from community studies. In some cases, behaviour change is concentrated among infected members of the population, but other changes are more widespread. A few of these behaviour modifications are the result of conscious intentions to alter fertility; but most are undertaken without considering the fertility effects or regardless of these consequences. Finally, changes in population composition, due to greater mortality in groups at higher risk of infection, add to the fertility effects of HIV epidemics when viewed at the population level (Zaba and Gregson, 1998).

These inter-actions between HIV and fertility have a number of important consequences for HIV surveillance and for understanding demographic trends in areas of high sero-prevalence. Low fertility in HIV positive women generally means that HIV prevalence data collected at antenatal clinics will understate true levels of infection among women of reproductive age. HIV induced population level reductions in fertility will complicate analyses of fertility transition in countries in the early stages of fertility transition. They will magnify the reduction in population growth caused by increased mortality but offset the latter's effect on population structure. An HIV epidemic will result in fewer orphans than previously anticipated and its effect on early childhood mortality will be smaller.

In many respects, the precise nature of these consequences will depend on the mechanisms through which HIV affects fertility and *vice versa*. For example, sub-fertility in HIV positive women could reduce over time if it is a consequence of STD acquired prior to HIV infection (Garnett and Gregson, 1998), but will persist if it results directly from the HIV infection. Understanding the causes

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of the HIV fertility relationship is therefore an important precursor to understanding the consequences. Understanding sexual behaviour, a field which demographers have studied because of its importance as a fertility determinant, is of paramount importance in the fight against AIDS.

Aims and overview of the paper

In this paper, we begin by reviewing the areas of overlap between the cultural, socio-economic and proximate determinants of HIV epidemics and fertility. These common determinants underpin the mechanisms through which different patterns of fertility-related behaviour are associated with contrasting HIV epidemics. After this, we focus on the changes in fertility likely to occur during the course of a major HIV epidemic, identifying specific mechanisms, examining the current evidence for changes through these mechanisms, and considering their potential fertility effects.

The conceptual framework of the underlying processes of change occurring in an HIV epidemic is incorporated into a mathematical model, and used to study the effects on population-level fertility and broader patterns of long-term demographic change. We illustrate how this model can be applied by showing how epidemics of similar magnitude could have different fertility effects in countries at different stages of development. The mathematical model is also used to examine the implications of the relationship between HIV and fertility for surveillance of an epidemic based on data from antenatal clinics. Finally, we use the model to demonstrate how this relationship has important consequences for the wider demographic and socio-economic impact of HIV epidemics in sub-Saharan Africa by examining the effects on population growth and age-structure.

CAUSES OF THE IMPACT OF HIV ON FERTILITY

Common cultural and socio-economic determinants of HIV and fertility

It has been suggested that the cultural and socio-economic factors that shape marriage and fertility in sub-Saharan Africa might also limit the effectiveness of AIDS control strategies (Bledsoe, 1991). If so, many of the factors that promote high fertility in African societies would be expected to facilitate HIV transmission. Furthermore, some of the processes of change that bring about reductions in fertility will simultaneously influence the course of local HIV epidemics. Evolution of cultural norms and practices resulting from processes such as increased urbanization, migration, education, secularization and technological innovation have been linked to the onset and progression of fertility transition in many societies (Caldwell and others 1992) and is also likely to influence patterns of spread of HIV epidemics.

In table 1, we identify some of the cultural, social and economic factors and processes that influence both fertility change and patterns of spread of HIV infection. Factors are categorized according to whether they are important determinants of fertility level, of HIV prevalence in the female population, or whether they influence both. The high fertility norm in pre-transition societies makes it difficult for couples to maintain consistent condom use even where the risks of HIV transmission are. In these respects, cultural systems that support high levels of fertility are more open to extensive HIV transmission. However, the situation is more complicated than this. Other aspects of cultural systems associated with high fertility, such as early marriage and taboos on pre-marital sexual intercourse, can act to restrict the spread of HIV infection. Features of traditional culture such as prolonged breastfeeding and sexual abstinence, which support prolonged birth spacing and suppress levels of natural fertility, can increase HIV transmission. Polygamy, which tends to reduce birth rates slightly (Pebley and Mbugua, 1989), is often associated with relatively low levels of extra-marital sex and could therefore limit the size of some HIV

epidemics (Frank 1992; Gregson and others 1999a). Equally, there are traditional practices not directly related to fertility outcomes but which affect levels of HIV transmission. Male circumcision, which appears to afford some protection against STIs including HIV (Caldwell and Caldwell 1993; Moses and others 1990), is one such practice.

Processes of social change, driven in part by local economic development, are frequently associated with fertility declines. Typically, more urbanized, better educated, secular populations, with lower child and adult mortality rates, are characterized by low or falling birth rates. Increased spousal separation due to labour migration can also lower fertility. However, these processes will have mixed implications for HIV control. Economic development, urbanization and secularization, and the accompanying increases in spatial mobility and more liberal sexual mores, will generally facilitate the spread of HIV infection. Increases in income differentials may also contribute to greater heterogeneity in rates of sexual partner change and lead to less assortative mixing patterns, which, in combination, can increase the size of HIV epidemics (Garnett and Anderson, 1993). Such patterns are particularly likely where the socio-economic status of women is low in relation to men (Bassett and Mhloyi, 1991). At the same time, higher levels of education should increase the feasibility of AIDS control initiatives especially those aimed at increasing knowledge and awareness of the risks of HIV transmission. Better health facilities should provide a stronger basis for controlling other STIs which act as cofactors in sexual transmission of HIV. Equally, proper screening of blood donations for transfusions and safe practices for the use of sharp instruments in medical settings are more practicable in such conditions and access to regular condom supplies is easier to ensure. If social development can facilitate fertility decline, it may be that cultural and religious resistance to condom use will also dissipate faster in more developed societies.

Relationship between respective proximate determinants of HIV and fertility

The socio-economic factors discussed above influence HIV transmission and fertility through an overlapping set of proximate determinants – table 2. A factor can have a positive effect on fertility but a negative effect on the size of an HIV epidemic - or *vice versa* - because it influences different proximate determinants. For example, urbanization may reduce fertility *inter alia* by reducing coital frequency within stable unions but can facilitate the spread of HIV by increasing rates of partnership formation. As we shall see, the contrast in direction of effects can also reflect opposing directions of influence on a common proximate determinant.

Broadly speaking, proximate factors affecting sexual partnership formation and dissolution and coital frequency within partnerships are important determinants of both HIV prevalence and fertility. However, the pattern of concurrent partnerships, a powerful influence on the prevalence of STIs (Morris and Kretzschmar, 1997; Watts and May, 1992), is not a determinant of fertility level, except if it acts through other factors such as coital frequency. Factors governing non-sexual HIV transmission among adults do not affect fertility. Similarly, biological determinants of fecundity influence HIV prevalence only indirectly (Zaba, 1994).

In a number of instances factors affecting both HIV and fertility act in the same direction. For example, extensive sexual activity and low condom use are associated both with higher fertility and higher HIV prevalence. However, this is by no means universal. High rates of divorce and widowhood reduce fertility - birth rates being lower outside marriage – but, other things being equal, increase HIV prevalence. This is because new regular partners often are already infected and, in some cases, a series of short-term relationships occurs before a new stable relationship is established. In African settings, women entering

second marriages frequently become second wives in polygamous unions (Timaeus and Reynar, 1998) or marry men who have also been separated. Thus, second husbands, typically, have had more previous partners than first husbands and are more likely to be sources of HIV infection.

The effects of short-term spousal separation - e.g.: due to labour migration - and post-partum abstinence also work in opposite directions on HIV and fertility. High levels of spousal separation and long duration of post-partum abstinence both reduce fertility but decreased exposure to HIV infection from the regular partner is offset by the higher risk from casual partners. Husbands are at particular risk if they move to settings where prostitution is common – e.g.: mining areas in South Africa (Campbell 1997). Where post-partum abstinence is practised, the taboo applies most strictly to women (Awusabo-Asare, 1996) and husbands may become infected if they resort to casual partners when their wives are sexually unavailable.

With respect to other STIs, biological and behavioural factors combine to exert opposite pressure on fertility levels and HIV prevalence. STIs - and particularly those that cause genital lesions - can facilitate HIV transmission (Greenblatt and others 1988) while those that cause cervicitis, salpingitis and PID can result in infertility (Arya and others, 1973; World Health Organization, 1988). Childbearing is an important source of status in most African societies and men frequently reject infertile partners. Often this leads to behaviour associated with heightened risk of HIV infection (Boerma 1996). A woman's anxiety to prove her fertility can hasten her participation in a new sexual relationship. Where there is no other means of socio-economic support, she may become involved in commercial sex work. Again, factors, which reduce fertility, increase the likelihood of HIV infection. This is true even for women who are infertile for reasons unrelated to STIs, though, in the African context, STI sequallae are thought to account for most cases of primary infertility (Larsen, 1994).

Individual and population level effects of HIV on fertility

The extensive areas of overlap between the socio-economic and proximate determinants of HIV epidemics and fertility provide many possible pathways through which an HIV epidemic can lead to changes in the fertility of an affected population. The principle potential mechanisms are listed in table 3. The list includes mechanisms that take effect at the individual and population levels. Individual level changes include both purely behavioural responses and changes that involve alterations in reproductive physiology. Those that primarily affect currently infected women will probably be less significant at the population level. Even in severely affected populations, a minority of women have HIV infection at any one time and an even smaller fraction are at the more advanced stages of infection when effects such as reduced coital frequency and increased amenorrhoea are most pronounced. Other behaviour changes – e.g.: delayed onset of sexual relations and increased condom use - could encompass the majority of the population and endure for a long time after the epidemic passes its peak.

Many of these mechanisms will reduce fertility, but others will offset this effect. The relative importance of the different mechanisms will vary between populations so that their net effect on fertility will also vary. In particular, the likely nature and scale of the net effect of an HIV epidemic of any given size on the fertility of a population will depend *inter alia* on the point in the demographic transition which has been reached. A variety of factors including pre-existing patterns of contraceptive use and sexual union formation will influence the nature of the behavioural response to the epidemic. Added to this is the question of intent. Will there be a conscious effort to raise fertility in response to HIV driven increases in death rates? In high mortality populations, couples may plan large families to "insure" against losing children[1] or decide to speed up their pattern of childbearing when a child dies – i.e.: to consciously

"replace" the child (Ware 1977). In an HIV epidemic, individuals who know they have HIV – and, possibly, others too - could seek to accelerate their childbearing.

In table 4, we re-classify the mechanisms listed in table 3 according to whether they can be regarded as conscious actions taken with the intention of influencing future fertility. A number of the actions take the form of deliberate responses to the epidemic. However, only two of these - the insurance and replacement effects following infant death; and increased contraception to avoid vertical transmission and orphanhood - represent conscious attempts to influence fertility and these act in opposing directions. Anxieties about adverse effects on maternal and child health and the possibility of orphanhood could outweigh desires for increased fertility in many instances. The net aggregate effect of such changes among HIV positive women would be minor because women who know they have HIV form a small minority of the HIV infected, and as noted earlier, those living with HIV are themselves a minority of the total population at any given time. For those who do know they are infected, fertility concerns may be uppermost in the minds of those with very few children. However, by the time they are aware of their infection, most will already have two or more children and, for these women, concerns about the care and wellbeing of existing children may be paramount (Setel, 1995). The insurance effect could be stronger for women who are unsure whether they have HIV (probably the majority) but these women will also be anxious to limit their own risk of becoming infected. How individual women react will clearly depend on the local cultural context and their own personal socio-economic circumstances.

This said, the analysis in table 4 highlights the possibility that a number of unintended changes in the proximate determinants of fertility could result in substantial changes in actual fertility outcomes. These include physiological changes affecting HIV negative women with HIV positive partners (column 4) as well as those that directly affect infected women (column 3). They include deliberate actions taken to avoid HIV transmission which have unintended and unrecognized consequences for fertility. Changes in some fertility determinants could occur in any of these ways. For example, other things being equal, infected women who experience higher levels of foetal loss and infant death will breastfeed and abstain for shorter periods – i.e.: an unconscious natural "replacement effect" will occur. Equally, women worried about passing on HIV infection to their newborn children may adopt shorter periods of breastfeeding even if they are unsure about their own HIV status. Women who fear their partners might "stray" if denied sex for too long might shorten the traditional abstinence period, while others who suspect their partners may be infected already could prolong post-partum abstinence.

Mechanisms and empirical evidence for changes in fertility determinants

The nature and expected scale of changes in birth rates following an HIV epidemic of a given size will reflect the combined effects (including inter-actions) of the various mechanisms identified and will vary according to the local context. In the second part of this paper, we will consider how the relative and combined effects of the different mechanisms could be influenced by the underlying socio-economic and demographic context and will examine the overall significance of changes specific and non-specific to HIV infected women. Before doing so, we will describe the individual mechanisms in more detail and review the current evidence for changes in the proximate determinants of fertility. To date, the full effects of HIV epidemics on mortality have been felt in only a small number of populations and only a few population-based studies of fertility impact have been undertaken. Where substantial and lasting behaviour change takes place only after increasing mortality has been recognized locally, the nature and potential extent of such changes will be obscured in early studies. Nonetheless, there is already sufficient evidence to substantiate a *prima facie* case for HIV epidemics becoming a major influence on fertility in badly affected populations

Changes affecting all women

In reviewing the biosocial and behavioural impact of HIV on fertility, we start by considering changes that may affect women irrespective of their serostatus and then look at changes which predominantly or exclusively affect HIV positive women. In the wider context, the principal biosocial impacts of HIV on fertility are likely to follow from the close inter-relationships between HIV, other STIs, and infertility. Primary and especially secondary sterility remain common in many sub-Saharan African countries (Larsen, 1994) and the link between untreated STIs and sub-fertility is well established (Arya, Nsanzumuhire, and Taber, 1973; Sherris and Fox, 1993; WHO: Rowe, and Vikhlyaeva, 1988). To the extent that HIV increases susceptibility to other STIs or adversely influences the natural history and response to treatment of these infections (Laga, 1992), an epidemic will tend to increase the prevalence of these infections and thereby reduce fecundity. On the other hand, ulcerative and probably non-ulcerative STIs can act as co-factors in sexual transmission of HIV (Greenblatt and others, 1988; Laga, 1992; Latif and others, 1989). Improved STI diagnostic and treatment services can reduce the incidence of new HIV infections (Grosskurth and others, 1995) and are promoted as an effective measure to counter epidemics. Where these services succeed in reducing the prevalence of other STIs, there will be an upward pressure on fertility (Garnett and others, 1992). Indeed, improved STI services are believed to have contributed to the early rise in birth rates seen in the initial stages of fertility transition in many developing countries (Dyson and Murphy, 1985).

Reductions in rates of partner change also would be expected to slow the spread of STIs (Allen and others, 1992) and therefore increase birth rates. Increased use of condoms would also reduce the prevalence of STIs and associated sterility but the resulting upward pressure fertility would be offset by their contraceptive effect. These individual relationships have been demonstrated in empirical studies, but, as yet, there are few data on their net effect at the population level. On balance, HIV control driven STI treatment programmes seem most likely to increase fecundity, particularly amongst the HIV negative, but more studies are needed to test this hypothesis.

Uninfected as well as infected women can have regular partners who are HIV positive, but the likelihood of this is greater among the latter. Where her partner is infected, a woman's fertility can decline due to his reduced fecundity, either because coital frequency is lower (Dublin and Blattner, 1993) or because spermatogenesis is reduced (Krieger and others, 1991; Martin and others, 1991). In the minority of cases where the partner has been diagnosed as HIV positive or is suspected of being infected, condom use may be more likely. Finally, in areas of high HIV prevalence, the incidence of widowhood will also increase. Many of these women will be infected themselves and some will not outlive their partners for very long. Even so, for those who survive and do not remarry, birth rates will fall. Increasingly, widow remarriage is perceived as problematic because of the dangers of further HIV transmission (Gregson and others, 1997b).

The best available evidence on behavioural responses to HIV epidemics which affect fertility comes from a few small-scale quantitative and qualitative studies (Asiimwe-Okiror and others, 1997; Gregson and others, 1997b, Mukiza-Gapere and Ntozi, 1996). Larger surveys of knowledge, attitudes, beliefs and practices (KABP) have also reported changes in behaviour, but are hard to evaluate. Reporting bias is a major difficulty and some questions are insufficiently well specified – e.g.: questions on condom use often fail to draw distinctions between casual and regular partners or consistent and occasional use. The scope of behaviour change investigated in such surveys rarely extends beyond rates of partner acquisition and condom use.

Pre-marital sex and pregnancies are common in sub-Saharan Africa and age at first marriage is young (Gage and Meekers, 1994). It was initially thought that the HIV epidemic would result in higher levels of female sexual activity at young ages, as men would seek younger partners believing they would present a lower risk of infection (PANOS Institute, 1989). However, results from a longitudinal study in urban areas of Uganda point to a rise in ages at first sex and first marriage associated with recent increases in AIDS cases (Asiimwe-Okiror and others, 1997). This association is supported by findings from focus group discussions held with young people in six districts of Uganda. Participants reported fear of marriage because they were unsure of the sero-status of their prospective partner (Mukiza- Gapere and Ntozi, 1996). Another Ugandan study (Carpenter and others, 1997) found declining fertility among the never married; evidence of declining pre-marital sexual activity. Gregson (1977b) found that in two rural areas of Zimbabwe young women with greater awareness of the risks of HIV infection delayed sexual debut. Since significant proportions of women are sexually active before marriage in many sub-Saharan African populations (Carael, 1995), reductions in pre-marital sex or increase in condom use within pre-marital unions because of fear of AIDS will reduce rates of pre-marital pregnancy. Where pre-marital pregnancies precipitate early marriage, their reduction will cause an increase in age at marriage and further reduce teenage fertility.

Anecdotal reports of marital breakdown following diagnosis or suspicion of HIV infection are numerous[2]. Staff at counselling-testing clinics in Uganda recount stories of couples breaking up on the spot upon receiving the HIV test results and there have also been many reports of women and children being abandoned by husbands (Ndinya-Achola and others, 1990). However, the choice for a woman with an infected or unfaithful husband is rarely straightforward. In Zimbabwe, as elsewhere, divorce frequently cuts women off from conventional family and economic resources and increases the chances of their becoming involved in commercial sex. Not surprisingly, HIV prevalence is particularly high among divorced women (Gregson and others, 1995). A number of women interviewed said divorced women could experience difficulties in remarrying and the possibility of HIV infection emerged as a significant new reason why this was problematic (Gregson and others, 1997b). Non-married women already have much lower birth rates than other women and reduced coital frequency and more regular use of condoms because of HIV would suppress extra-marital fertility further.

In the Zimbabwe study, women were asked whether the AIDS epidemic had made them change their minds about how many children they would like or the timing of their next birth. Almost half said they now wanted fewer children and a similar proportion reported a preference for a delay in the timing of the next birth. Very few wanted to increase or accelerate their childbearing (Gregson and others, 1997b). Birth rates are already falling in Zimbabwe, so it is possible that the pattern of response was influenced by a more general and widespread desire to reduce fertility. Given underlying cultural pressures such as the enhanced status associated with motherhood and the importance of survival by living descendants, the AIDS epidemic might have been expected to reverse this trend, at least among younger women. However, there was no evidence of an association between greater awareness of the risks of HIV infection and increased desired family size. Any conscious replacement or insurance effects would so far seem to be weak in this population.

HIV epidemics could increase contraceptive use and alter the mix of methods used due to the need to protect against infection and through the impetus they give towards more open discussion about sex and reproductive health. Changing patterns in contraceptive method could occur, as some methods are protective against HIV infection while others may act as cofactors in HIV transmission (Daly and others, 1994). New methods, such as the female condom, may provide women with greater protection against

sexually transmitted HIV infection (Ray and others, 1995). The International Planned Parenthood Federation and some Western governments have recommended combinations of contraceptive methods because condoms can be a relatively unreliable method of family planning (Doppenburg, 1993; Gordon and Klouda, 1989). However, many failures result from incorrect use and effectiveness may improve with more intensive HIV led education programmes. In much of sub-Saharan Africa, there are substantial practical and cultural difficulties in achieving a high uptake of condoms[3]. For example, few women are familiar with condom use and condoms are associated with casual rather than long-term childbearing relationships (Mehryar, 1995).

Modern contraceptive methods have become increasingly popular in Zimbabwe over the past decade and account for much of the recent fertility decline (Mandishona, 1989; Parirenyatwa, 1995). Condoms are more widely used as the principal method of contraception but are still employed by only 8 per cent of current users of modern methods. A small minority of rural women (6 per cent) report changing method since hearing about AIDS; almost all of these now use condoms and one third use the pill and condoms in combination (Gregson and others, 1997b). In urban areas, intensive peer-education and condom distribution programmes have led to substantial increases in reported condom use within casual relationships; a finding substantiated by a recent fall in STI cases (Wilson and others, 1994). In Uganda, where the epidemic is more advanced, increases in condom use have been reported among adolescents and adults (Asiimwe-Okiror and others, 1997; Konde-Lule and others, 1997; Kilian and others, 1998). Given the low underlying levels of contraceptive prevalence in Uganda (Kaijuka and others, 1989), these increases also may have reduced unplanned pregnancies.

Breastfeeding is almost universal in sub-Saharan Africa and contributes towards long periods of post-partum insusceptibility to conception. Breastfeeding is also a significant mechanism for vertical HIV transmission (Dunn and others, 1992; Van de Perre and others, 1991), a fact which, until now, has not been publicized in African communities because of the wider benefits of breastfeeding to infant and maternal health (World Health Organization, 1995). However, many women suspect that HIV can be transmitted via breastmilk and some have abandoned the practise (Gregson and others, 1997b). More widespread knowledge of the link between breastfeeding and infant infection will probably reduce breastfeeding further particularly where there are viable alternatives. To the extent that this results from increased provision (and take-up) of voluntary counselling-testing services for pregnant women, the majority, whose test results are negative, may be persuaded to continue breastfeeding. However, overall, there is likely to be some upward pressure on fertility, particularly where contraceptive prevalence is low.

Post-partum abstinence continues to be practised by many African women, albeit for shorter periods than in the past (Caldwell and Caldwell, 1977; Caldwell and others, 1989). Men in rural Zimbabwe cited female post-partum abstinence as a reason for having extra-marital affairs. Women feared HIV infection because they suspected their partners had other relationships and, in some cases, shortened their abstinence periods to reduce this possibility (Gregson and others, 1997b). Male extra-marital relationships are common in many African populations (Carael, 1995) and it seems unlikely that it is only in Zimbabwe that these occur more frequently during periods when wives are abstaining. Where husbands become infected during these periods, wives are at particularly high risk of infection when sexual relations are resumed, because their husbands' infections are recent and highly contagious (Hudson 1993). Equally, if a woman becomes infected while she is breastfeeding, the risk of vertical transmission is heightened. Thus, a period of female post-partum abstinence during which the husband has other relationships followed by resumption of sexual activity before breastfeeding is terminated could result in the infection of both partners and the child. If more women become conscious of these aspects of HIV transmission, further

adjustments in abstinence and breastfeeding practices could occur. These changes could spread rapidly, since the idea that sexual relations during breastfeeding can damage the health of the child because the milk is poisoned is a familiar one in African cultures.

Changes particularly affecting women with HIV infections

Lower fertility among HIV positive women has been recorded in clinical settings (Allen and others, 1993; Batter and others, 1994; Ryder and others, 1991) and, more recently, in population-based studies (Carpenter and others, 1997; Sewankambo and others, 1994). Data from Uganda suggest that pregnancy rates can be 50 per cent lower, even after allowing for the effects of other factors including the presence of other STIs (Gray and others, 1997). The association may be stronger at older ages (Allen and others, 1993; De Cock and others, 1994; Gray and others, 1997; Johnstone and others, 1988; Stephenson and others, 1996), one possible reason being the greater reduction in fertility typically seen at more advanced stages of HIV infection (Ryder and others, 1991).

The mechanisms causing lower fertility in women with HIV are not currently well understood, but probably reflect a combination of biosocial and behavioural factors. Miscarriage, spontaneous abortion and stillbirths appear to be more common in infected women (Gray and others, 1997; Miotti and others, 1991; Ryder and Temmerman, 1991; Brocklehurst and French, 1998). Increased prevalence of amenorrhoea has also been recorded in women at the later stages of HIV infection (Widy-Wirski and others, 1988) and coital frequency is liable to decline. However, if many children of HIV positive women die in infancy, average periods of breastfeeding and abstinence will tend to be shorter and conception rates could increase. Some of the subfertility observed in a cohort of Ugandan women with known infection dates resulted from lower than average gravidity prior to HIV infection (Ross and others 1998).

A number of different forms of behaviour change can occur after a positive HIV diagnosis. In rural Zimbabwe, almost all respondents felt that a woman who found she was infected should stop having children. Reasons given included the risk of vertical transmission (which was thought by many to be inevitable) and concern about orphans (Gregson, 1997b). However, what women do when actually faced with the decision may be different. Some may not wish to inform their partners for fear of violence and abandonment (Keogh and others, 1994; Temmerman and others, 1993) and would therefore experience difficulties negotiating continuous contraceptive use. For others – particularly those with few living children – the wish to bear children may over-ride concerns about HIV transmission. This could contribute to the smaller fertility shortfalls seen at younger ages. While condom use has been reported as being higher and contraceptive pill use lower in a controlled study of HIV positive women, usage was thought to be erratic and probably had little overall effect on fertility (Keogh and others, 1994; Ryder and others, 1991; Setel, 1995). In Western countries, HIV positive women, including some black Africans, have higher rates of induced abortion (Stephenson and others, 1996; Thackway and others, 1997), but it is difficult to tell whether this is the case in Africa. Voluntary terminations are known to be common, especially among young single women but are frequently illegal. As a result, unsafe procedures are often used and abortion is a leading cause of maternal mortality (Mpangile and others, 1993; Rogo, 1993). However data from Zaire suggest that the current excess in induced abortions among infected women in sub-Saharan Africa is small (Ryder and others, 1991).

There are very few direct data on breastfeeding decisions after HIV diagnosis. Infected women living in areas of sub-Saharan Africa areas where diarrhoeal diseases and other potentially fatal infections are common, have generally been advised to breastfeed because of the wider health benefits. Recent WHO guidance proposes disclosure and discussion of the issues with women known to be HIV positive

(World Health Organization, 1996). There are very few such women at present, but greater access to voluntary counselling-testing services – which may follow the advent of more affordable therapies for reducing vertical transmission – could lead to reductions in breastfeeding and foetal loss, possibly offset by increases in induced abortions and subsequent contraceptive use, particularly in urban areas.

Finally, women who are diagnosed as HIV infected and reveal this to their partners – or whose partners suspect they are infected – are more likely to experience divorce and separation, will encounter greater difficulties finding a new husband, and will therefore have lower fertility.

Population level effects

The long-term influence of an HIV epidemic on population level fertility extends beyond the aggregated impact of its individual level effects. This is because, over time, it will significantly alter the composition of the population. In the early years of an epidemic, individuals with high rates of sexual partner acquisition and low levels of condom use become infected and die faster than the population as a whole. If not replaced, their representation in the population will decline. If their fertility differs from that of the general population, overall fertility will be affected wherever they comprised a significant fraction of the initial population. We have already noted that infertile women experience higher rates of STIs and partner change and, thus, higher risks of HIV infection. In a major HIV epidemic, the proportion of women who are infertile will decline and population level fertility will tend to increase.

Other population sub-groups may also be affected disproportionately with contrasting effects. For example, later in an epidemic, if less educated people respond more slowly to education and information campaigns, they may have higher infection rates (Kilian and others, 1998), a phenomenon which would suppress fertility in some populations. Finally, changes in population age and sex-structure can also affect fertility by concentrating the population into more or less fertile age groups and by influencing marriage patterns.

CONSEQUENCES OF THE IMPACT OF HIV ON FERTILITY

Although many of the inter-actions between HIV and fertility have been observed and documented, there are no empirical studies examining the consequences at the level of the whole population. Indeed, such studies would be very difficult to design, since other secular influences on fertility would have to be controlled for. Documenting the consequences for infected and uninfected people would require wide-scale HIV testing which would not be ethical unless assistance is made available to the participants. In these circumstances, approaches based on mathematical models offer the best opportunities for exploring the impacts of HIV on fertility at the population level. In this section, we employ an extended version of the model developed by Zaba (1994). This is a deterministic stable population model based on multiple decrement life table procedures, which allows for the full range of proximate fertility determinants in both the HIV positive and HIV negative populations. The fertility and mortality consequences of an epidemic are integrated within the model. Thus, it is possible to make estimates of the growth and structure of a stable population in which the HIV epidemic has also stabilized at a level consistent with an assumed *per-coitus* infection risk pattern. A mathematical specification of the model can be found in the original paper.

Expected scale of fertility change – more and less developed countries

In this section, we provide some quantitative illustrations of the potential fertility impact of an HIV epidemic of fixed size in populations at the pre/early and mid/late stages of fertility transition. For the purpose of the exercise, we assume that the former are more developed than the latter and are characterized by more extensive contraceptive use and lower non-HIV related mortality. Table 5 shows the assumptions used for each of the proximate determinants of fertility in these two types of population, both before and in the presence of the HIV epidemic.

In the pre-epidemic phase, important characteristics of the more developed population include a younger age at menarche, lower foetal mortality, and lower levels of sterility, breastfeeding, post-partum and terminal abstinence, all of which would tend to raise its natural fertility levels. However, the more developed population is also assumed to have a later sexual debut, higher contraceptive prevalence, higher rates of sexual union breakdown, and lower rates of re-entry into sexual unions. Sexually active women have the same rates of coital frequency in both populations; in each case, coital frequency declines with duration of current sexual union. Although the two model populations have not been calibrated to represent particular countries, they can be thought of as typifying the pre-transition populations of East Africa (e.g. Tanzania and Uganda) in the less developed case, and the mid- transition populations of Southern Africa (e.g. Zimbabwe and Botswana) for the more developed scenario.

To highlight the contrasting nature of the effects on fertility determinants in these different circumstances, we assumed that HIV reaches a constant endemic prevalence level of around 15 per cent among women of reproductive age in both cases. It is possible, of course, that prior level of development will influence the severity of HIV epidemics but we have not investigated this possibility here. Table 5 shows our assumptions about the changes in fertility determinants occurring among infected and uninfected women in the wake of the epidemic. The differentials include higher foetal loss and a lower mean age at menopause for infected women, who are also assumed to breastfeed for shorter periods, have higher union breakdown rates, lower reformation rates and lower coital frequency within unions. We do not model changes reflecting deliberate attempts to alter fertility in response to knowledge of HIV status or changes in fertility norms resulting from awareness of increased mortality.

Our assumptions about patterns of initiation of sexual activity, onset of secondary sterility and condom use need more detailed explanation. Women who have never been sexually active are, by definition, uninfected. Age at first sex and the build up of sexual activity are thus only defined for the uninfected, who are then assumed to be at risk of acquiring the infection. As those who start sexual activity earlier face a higher lifetime risk of becoming infected, retrospective measures of mean age at sexual debut are lower for HIV positive women. This is a selection effect rather than a determinant of the fertility of infected women.

Women were categorised according to their risks of STI-associated sterility. Since the same pattern of sexual behaviour underlies the risks of STI and HIV infection and each can act as a transmission co-factor for the other, women who acquire HIV infection are concentrated in the group with heightened risk of sterility. The age-patterns (but not the ultimate levels) of onset of secondary sterility and HIV incidence are assumed to be the same.

Contraceptive prevalence rates are assumed equal for HIV infected and uninfected women, but where we test the effects of condom use we assume that uninfected women currently using condoms are protected from infection. HIV positive women are thus initially selected as being non-users of condoms at the time of infection but this assumption is subsequently relaxed – after sero-conversion, condom use rates are taken as being the same in both sub-groups.

The way in which the determinants of fertility in the infected and uninfected sub-groups combine to govern population-level fertility is modified by the size of the HIV epidemic and the severity of its mortality consequences. These factors determine the balance between the proportions of the population that are healthy and sick at each age. In both scenarios, mean survival time was set at 11.5 years from infection for adults and 2 years for infants[4]. The *per coitus* new partner infection risk which resulted in an HIV prevalence of 15 per cent was 0.2 per cent - *per coitus* infection risks were assumed to decline with union duration, down to a level of 0.02 per cent for unions lasting more than 5 years, to represent a selection effect whereby unions in which partners have little sexual involvement with outsiders are presumed to be more stable.

To illustrate the combined effect of possible changes in the different proximate determinants, we used Bongaarts' decomposition model (Bongaarts and Potter, 1983; Gregson, 1994). A summary of the results is given in table 6 and figures 1a and 1b. The overall reduction in total fertility is slight in the less developed scenario (less than 3 per cent overall), but there is a large decline (16 per cent) in fertility levels in the more developed scenario. In the less developed scenario the total fertility of HIV positive women is close to half that of HIV negative women, but this differential is almost absent in the more developed scenario. In both cases, there is a shift in the balance of fertility reduction accounted for by the different proximate determinants. Exposure within sexual unions and contraception become more important while the role of the variables controlling fecundity is diminished. The reason for the latter is partly structural: women with lower risk of secondary sterility form a larger fraction of the total population than previously, as those at high risk have been depleted by high levels of HIV mortality.

Table 6 also shows the potential effect on fertility of each model parameter. Condom use lowers fertility less than other forms of contraception. This is not due to contraceptive inefficiency, which has not been allowed for in this model, but is explained by structural effects. Higher condom use leaves more women free of HIV and other STIs, so they experience higher fertility rates. Higher proportionate use of condoms also reduces the impact of contraception in the HIV infected. This reflects the initial selection of non-condom users into the HIV infected group: the more contraceptors who use condoms, the smaller the proportion of newly infected individuals who use contraception. In both scenarios, age at first sex has a much smaller fertility reducing impact among HIV infected women than among other women. This is because infected women are selected for earlier commencement of sexual activity - even in populations in which sexual activity generally starts late they will be found disproportionately amongst those who become sexually active earliest.

Implications for antenatal surveillance of HIV prevalence

Most national level estimates of HIV prevalence are based on anonymous screening of women attending antenatal clinics, as this procedure is convenient, inexpensive and ethically acceptable (Chin, 1990). Pregnant women are taken as representative of all women, so that these estimates are generally used without adjustment or just standardized for age. However, we have seen that large fertility differentials may arise between infected and uninfected women and that the epidemic itself can affect the fertility of a population.

Figure 2 shows the patterns of age-specific fertility for HIV infected and uninfected women in the two simulations. In both cases, infected women have higher fertility than their uninfected counterparts up to age 20, but substantially lower fertility thereafter. The selection effect at young ages is more evident in the more developed scenario, as sexual activity is assumed to start later in this population, so those who do

become infected at early ages are especially atypical. Differences between the age-specific fertility of infected and uninfected women cause antenatal HIV prevalence estimates to be higher than community levels at ages under 20 but lower above this age (Zaba and Gregson, 1998). The age relationship between antenatal and community prevalence is similar in the two scenarios (figure 3) and should therefore be fairly robust to differences in fertility distributions. Comparison of the crude prevalence measures for pregnant women and all women aged 15-49 over a range of simulations (not shown) indicate that the much lower prevalence in pregnant women at older ages far outweighs the opposite bias at younger ages, especially in simulations with fertility patterns characteristic of less developed populations. Therefore, the crude estimate for pregnant women is likely to be an underestimate of prevalence in all women of childbearing age, more so in less developed than more developed populations. Age-standardization reduces antenatal estimates further, as it gives more weight to older ages than crude estimates, so unstandardized estimates are generally preferable, contrary to what was thought before the extent of lower fertility in HIV infected women was known (Lessner, 1991; Gregson and others, 1995).

For most of the model populations investigated, the extent of the bias (measured as the percentage point difference between prevalence in pregnant women and all women of reproductive age) increases almost linearly with the true HIV prevalence until the true prevalence reaches about 50 per cent and declines thereafter. This simple relationship holds if HIV prevalence is altered without changing the relative values of the fertility determinants for the HIV positive and negative sub-groups. As a proportion of true prevalence, the measurement error declines with increasing HIV prevalence. This is illustrated in figure 4a, which shows the effect of varying levels of *per-coitus* risk of infection in the two populations for which we have developed fertility scenarios. In the less developed scenario, prevalence in pregnant women rises from about 6 per cent to 26 per cent as true prevalence increases from around 8 per cent to 31 per cent. In the more developed scenario, the bias is about 2 per cent lower at the higher levels of prevalence.

The bias in antenatal estimates is very sensitive to the extent of condom use. In figure 4b, we illustrate this by keeping the infection risk per unprotected coitus constant but varying the pattern of condom use. In the "more developed" scenario, this is achieved by fixing overall contraceptive prevalence at 30 per cent but varying the proportion of contraceptors who use condoms between 20 per cent and 100 per cent. In the less developed scenario, we assume that condoms are the only form of contraception used and that usage varies between 1 per cent and 10 per cent. These simulations show that prevalence in pregnant women is more sensitive to the extent of condom use than to changes in community HIV prevalence resulting from condom use. If condoms were used very widely, antenatal HIV prevalence estimates could overstate general population levels.

These findings have important implications for the ability of antenatal surveillance systems to capture real trends in HIV prevalence in the general population. Since it is unrealistic to expect condom use rates to rise to the very high rates shown in figure 4b, we would generally expect antenatal surveillance to under-estimate community prevalence. In any given setting, there will probably a slight increase in the undercount as the epidemic matures and HIV prevalence rises, but, if there is no change in contraceptive use, the direction of the prevalence trend should be captured by ANC surveillance. However, if underlying prevalence falls, due to successful condom promotion, antenatal surveillance could fail to capture the full extent of the trend.

Population growth rate and age-structure effects

In general, fertility changes have a more marked effect on population growth and age-structure than mortality changes. The case of HIV is an exception to this rule. Nonetheless, the fertility effects of HIV epidemics will also be significant in many populations. Figure 5a shows the relationship between the intrinsic growth rate and HIV prevalence for our less developed population scenario. The *per coitus* infection risk is varied to generate alternative levels of HIV prevalence. The measurements of growth presented here would be observed in stable populations in which epidemics have also stabilized. They can thus be interpreted either as the long term consequences of the epidemic or as abstractions showing the tendency of change in these measures independent of initial population structure. Mortality effects on growth were measured by calculating only the direct effects of HIV on female survival, ignoring all behavioural, biosocial and structural changes in fertility. In this population, fertility decrease lowers growth by a third as much as is effected by mortality increase, that is if mortality increase lowers growth by 1.5 percentage points, fertility decrease would lower it by a further 0.5 percentage points. In the more developed scenario, the impact of HIV associated fertility change is even stronger because of the bigger absolute change in total fertility.

Where large-scale HIV epidemics develop, death rates will generally increase and population growth rates will tend to fall. In a number of more developed sub-Saharan African countries, growth rates were falling before any noticeable impact of the epidemic, as fertility declines were outstripping improvements in mortality. In countries such as Zimbabwe and Botswana, the combination of rising death rates due to HIV and continuing declines in birth rates could lead to periods where the rate of natural increase falls slightly below zero (Gregson and others, 1997; U.S. Bureau of the Census, 1997). Whether this will occur will depend significantly on the direction and extent of the impact of HIV epidemics on fertility. At present, it seems most plausible that these epidemics will exert a further downward pressure on fertility, accelerating existing declines in population growth in these countries.

The relationship between population age-structure and mortality is fairly complex. Decreased survivorship is associated with a younger age-structure but reduced population growth, also a consequence of increased mortality, limits this tendency. HIV mortality is heavily concentrated at young adult ages so the direct effect is stronger than the indirect effect that acts through the growth rate. The results shown in figure 5b, derived from our "less developed" model with varying *per coitus* infectivity, illustrate how the mortality effects of high HIV prevalence produce a younger age-structure. However, lower birth rates yield older age-structures because population growth is reduced. Where an HIV epidemic reduces fertility, this ageing effect could counterbalance the tendency of increased mortality to produce a younger age-structure. In this example, the epidemic has little net effect on the mean age of the population.

Implications for the broader demographic effects of HIV epidemics

Fertility decline can also influence other demographic effects of HIV, and it is important to take account of fertility differentials between infected and uninfected women. The case of maternal orphanhood is a good example. Declining fertility would tend to increase the proportion of children under 15 who are orphaned, because the child population would have an older age structure, and older children have a higher cumulative risk of parental death. Falling fertility in the context of an HIV epidemic, would thus tend to further increase the proportion orphaned. However, if women with HIV have lower fertility, the overall proportion of orphaned children will increase more slowly. There will also be fewer children who are orphaned at very young ages, particularly if the fertility effect of HIV on infected women is greatest at more advanced stages of infection. Of course, the degree of change in the absolute number of orphans in a population will also depend on the population age-structure and growth rate, which, as we

have seen, are themselves affected by the fertility impact of an HIV epidemic. These effects are discussed in more detail elsewhere (Gregson and others, 1999b).

Lower overall birth rates will reduce the numbers of babies infected and dying from HIV related causes, in absolute terms. Lower birth rates in women with HIV will also reduce the impact of epidemics on early childhood mortality rates. Fewer children will be born to infected women so the proportion of all babies who are born with HIV infections will be smaller and the impact of the HIV epidemic on infant and childhood mortality will be less severe than would otherwise be expected. In particular, young orphans are less likely to be infected, so that death rates among orphaned children may not be quite as bad as was first feared. Lower orphan mortality will result in a slight ageing in the orphan population as a whole.

CONCLUSIONS

The relationship between HIV and fertility is bi-directional and highly complex and empirical study of the relationship at the population level probably impossible. Nonetheless, the consequences are profound and, therefore, it is important that an understanding of the intricacies of the relationship is developed. In this paper, we have attempted to advance current understanding by: (a) reviewing the areas of overlap between the socio-economic and proximate determinants of HIV epidemics and fertility; (b) using this review as a basis for identifying possible mechanisms for interaction between HIV epidemics and fertility; (c) examining existing evidence for the effects of these mechanisms; and (d) applying a mathematical model to study the population level effects of HIV on fertility and the implications of fertility differentials between infected and uninfected women for HIV surveillance based on data from antenatal clinics.

The review of common determinants highlighted the fact that individual factors and processes of change can have contrasting effects on HIV epidemics and levels of fertility. Some aspects of traditional culture that support high fertility can serve to suppress levels of HIV prevalence. Equally, aspects of socio-economic development generally associated with declining fertility may facilitate the spread of HIV epidemics. Thus, it is not altogether surprising that southern African populations where levels of socio-economic development are relatively high and fertility is falling are experiencing the most severe HIV epidemics (U.S. Bureau of the Census, 1997).

Population-based data suitable for quantifying the effects of possible mechanisms of interaction between HIV and fertility have been slow to emerge. However, the few studies conducted show that significant changes are taking place in the populations studied. In particular, HIV infection is strongly associated with sub-fertility and changes in reproductive behaviour have been recorded. Current evidence on conscious fertility decisions taken in response to the epidemic in general or to personal knowledge of infection status is inconclusive. Moves are underway to introduce voluntary counselling and testing services for pregnant women, as a pre-requisite for providing anti-retroviral therapy to reduce mother to child transmission of the virus. If these are successful, they should improve fertility outcomes for infected women but may also prompt further changes including reduction in breastfeeding and increase in contraception and induced abortion.

For the purposes of generating the illustrations based on the mathematical model, we assumed that the scale of the HIV epidemic would be the same in more and less developed societies. Under this assumption, HIV reduces total fertility substantially in the more developed scenario but has little net effect in the less developed population. If epidemics are more severe in more developed countries the differential between the effects on fertility will be greater still. The simulation results illustrate how surveillance systems which rely on data from antenatal clinics can fail to capture the true extent of HIV epidemics

within the population as a whole and could conceal improvements resulting from increases in condom use. They emphasize the need for a straightforward procedure for adjusting antenatal estimates.

Finally, the model simulations show that a change in fertility caused by an HIV epidemic would significantly affect the overall impact of the epidemic on key demographic indicators including population growth and age-structure. In some cases, fertility change tends to re-enforce the impact of increased mortality, but, in others, it can alleviate these effects. Fertility differentials between infected and uninfected women are important in determining the effects of an epidemic on demographic features such as orphanhood and early childhood mortality.

TABLE 1. CULTURAL, SOCIAL AND ECONOMIC FACTORS AND PROCESSES WHICH AFFECT FERTILITY LEVELS AND MAY ALSO INFLUENCE HIV PREVALENCE

Effect of factor on fertility		
Positive effect	*Negative effect*	*No known effect*
	A. Factors which tend to increase HIV prevalence	
High fertility norms	Cultural norms supporting prolonged birth spacing	Absence of male circumcision
Cultural or religious opposition to contraception		
Cultural norms supporting universal marriage		Testing out of potential marital partners
Monogamous marriage system		
Low status of women relative to men	Extensive urbanization	Liberal sexual mores
Low contraceptive (condom) availability	Extensive labour migration	Substantial income differentials
	B. Factors which may influence HIV prevalence, but direction of association is uncertain	
High infant and child mortality	High per capita income	
Taboo on induced abortion	High female education levels *	

* Initially more educated individuals appear to be at higher risk of HIV infection. However, as epidemics progress and more information becomes available through schools and media-based education programmes, this differential may be reversed (Kilian, et al 1998).

TABLE 2. PROXIMATE DETERMINANTS OF FERTILITY AND ADULT HIV PREVALENCE

Effect of factor on fertility		
Positive effect	*Negative effect*	*No known effect*
A. Determinants which tend to increase HIV prevalence		
Higher proportion ever entering sexual relations	Longer duration of post-partum abstinence	Extensive concurrent partnerships
Younger age at onset of sexual relations	Extensive temporary separations	Contaminated blood transfusion
Higher coital frequency	Higher incidence of divorce	Needle sharing
Lower prevalence of barrier contraception	Higher widowhood incidence	
Older age at terminal abstinence	Greater STI induced infertility	
Higher union reformation rate after separation/widowhood		
B. Determinants which may influence HIV prevalence, but direction of association is uncertain		
Young age at menarche		
Low induced abortion		
Low use of non-barrier contraception		
Shorter duration of breastfeeding amenorrhoea *		
Older age at menopause		

*Affects pediatric but not adult HIV infection.

TABLE 3. HYPOTHESIZED DIRECTION OF EFFECTS OF AN HIV EPIDEMIC ON FERTILITY

Bio-social change		*All * women*	*HIV+ only*	*Overall effect*
	A. Individual level			
BS1	Increased widowhood	-	-	-
BS2	Reduced coital frequency due to increased morbidity	-	-	-
BS3	Increased spontaneous abortions and stillbirths	-		-
BS4	Increased amenorrhoea	-		-
BS5	Lower fecundity due to poor nutrition & low spermatogenesis	-	-	-
BS6	Reductions in other sexually transmitted infections		+	+
Behaviour change				
BH1	Delayed onset of sexual relations		-	-
BH2	Reduction in pre-and extra-marital sexual relations		-	-
BH3	Delayed marriage – possibly resulting in increased celibacy		-	-
BH4	Reduced polygyny		+	+
BH5	Increased divorce	-	-	-
BH6	Reduced remarriage	-	-	-
BH7	Switching of family planning method from pill to condom	+	+	+
BH8	Reduction in breastfeeding	+	+	+
BH9	Reduction in post-partum abstinence		+	+
BH10	Condom use among previous non-contraceptive users	-	-	-
BH11	Less contraception due to insurance and replacement effects	+	+	+
BH12	More contraception and abortion to avoid infant infection and orphanhood	-	-	-
	B. Population level			
P1	Change in population age and sex-structure			+/-
P2	Excess mortality among infertile women			+
P3	Excess mortality among hormonal contraceptive users			+
P4	Excess mortality among women with multiple partners			+

* may affect HIV+ women disproportionately.

TABLE 4. INDIVIDUAL LEVEL CHANGES ARISING FROM AN HIV EPIDEMIC WHICH COULD AFFECT FERTILITY: DELIBERATE AND UNINTENDED CHANGES

	Nature of HIV response			
	Intended		*Unintended*	
Nature of fertility consequences	Possibility of HIV transmission	Due to woman being HIV+	Due to woman being HIV+	Due to wider HIV epidemic
Intended to change fertility	BH11-BH12	BH11-BH12		
Unintended fertility consequences	BH1-BH10*, BS6	BH6-BH8, BH10	BH5-BH6, BH8, BS2-BS5	BS1-BS2, BS5-BS6

* in the case of BH8 (reduction in breastfeeding), the change would be to prevent possible mother-to-child transmission. In each of the other cases, the change would be to prevent the woman's own infection

Mechanisms can fall into more than one category. For example, divorce may occur following diagnosis of HIV infection in either partner. Where the woman is infected, her partner may divorce her, which would be an unintended event from the woman's point of view (column 3). In the reverse situation she might divorce the husband to reduce her own chances of becoming infected, which would be an intended event (column 1).

TABLE 5. PLAUSIBLE VALUES OF PROXIMATE DETERMINANT PARAMETERS FOR FERTILITY PATTERNS IN LESS AND MORE DEVELOPED SUB-SAHARAN AFRICAN COUNTRIES FACING HIV EPIDEMICS

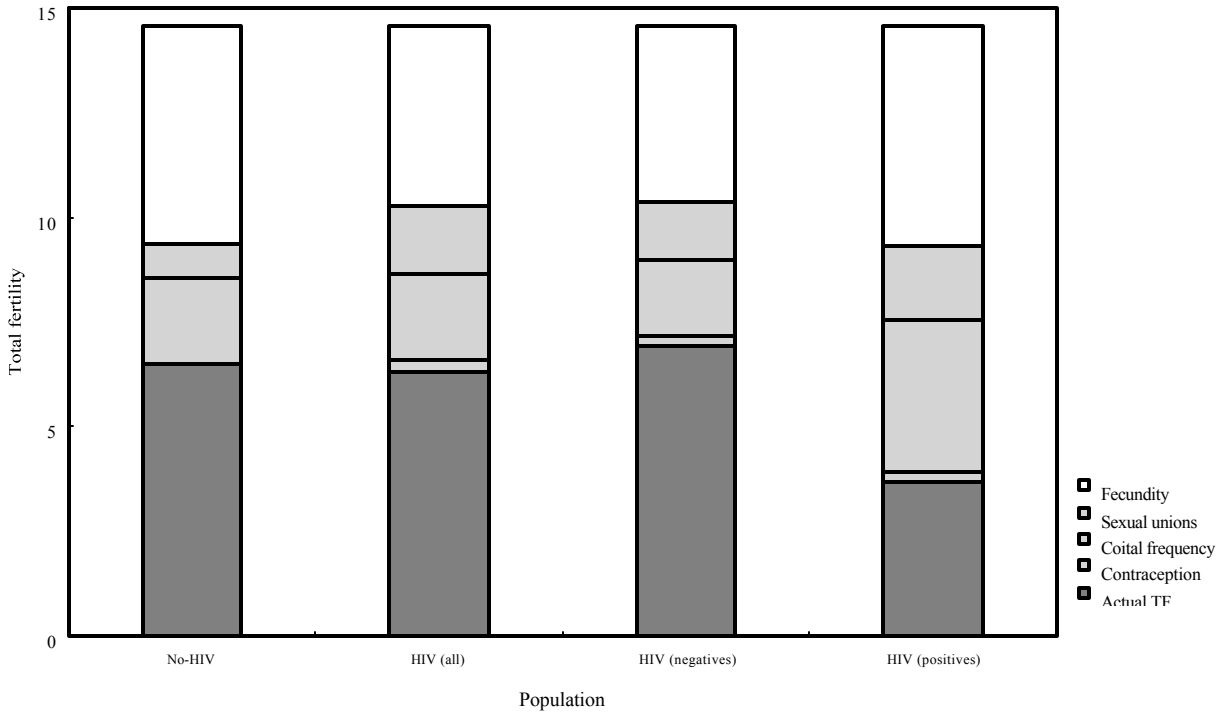
	Theoretical maximum fertility	*Less developed country*			*More developed country*		
		Pre-HIV	*HIV-*	*HIV+*	*Pre-HIV*	*HIV-*	*HIV+*
Fecundity							
Age-range: [menarche, menopause]	[10,55]	[14,50]	[14,50]	[14,45]	[12,50]	[12,50]	[12,45]
Foetal wastage (x US standard)	1.00	1.10	1.10	1.40	1.05	1.05	1.30
Secondary sterility (%) [low, high]	[10, -]	[30,80]	[30, -]	[- ,80]	[20,50]	[20, -]	[- ,50]
Lactational amenorrhoea (months)	1.5	12	9	6	9	6	3
Sexual unions							
Age at first sex: [earliest, mean]	[12,16]	[12,17]	[13,18]	-	[14,19]	[15,20]	-
Divorce & separation - annual risk (%)	0	5	5	10	10	10	20
Widowhood – male life expectancy	85	50	40	25	60	45	30
Remarriage - maximum annual rate (%)	100	100	80	40	70	50	20
Remarriage - peak age (years)	30	25	25	20	25	25	20
Coital frequency							
Post-partum abstinence (months)	1.5	4	2	6	3	2	4
Terminal abstinence (starting age)	60	50	45	40	55	50	45
Coital frequency - [min, max]	[.1,.5]	[.06,.3]	[.06,.3]	[.03,.3]	[.06,.3]	[.06,.3]	[.03,.3]
Contraception							
Peak age-specific usage level (%)	0	0	2	2	55	65	65
Age at peak usage (yrs)	-	-	25	25	35	35	35
Condom use as % of contraception	0	0	100	100	2	20	20

TABLE 6. BONGAARTS' INDICES AND TOTAL FERTILITY FOR LESS AND MORE DEVELOPED SUB-SAHARAN AFRICAN COUNTRIES FACING HIV EPIDEMICS

	Less developed country				*More developed country*			
	Before HIV	*During HIV epidemic*			*Before HIV*	*During HIV epidemic*		
		All	*HIV-*	*HIV+*		*All*	*HIV-*	*HIV+*
Fecundity	**0.52**	**0.56**	**0.60**	**0.39**	**0.63**	**0.67**	**0.68**	**0.51**
Menarche-menopause	0.85	0.82	0.85	0.67	0.88	0.84	0.87	0.71
Foetal wastage	0.93	0.88	0.92	0.72	0.97	0.92	0.96	0.78
Infertility	0.87	0.90	0.93	0.78	0.93	0.94	0.96	0.87
Lactational amenorrhoea	0.71	0.78	0.77	0.82	0.77	0.86	0.85	0.91
Sexual unions	**0.93**	**0.83**	**0.87**	**0.80**	**0.80**	**0.65**	**0.71**	**0.60**
Age at first sex	0.97	0.92	0.92	0.98	0.91	0.86	0.85	0.97
Divorce & seperation	0.95	0.92	0.95	0.88	0.90	0.86	0.90	0.78
Widowhood	0.99	0.98	0.98	0.97	1.00	0.98	0.99	0.97
Remarriage rate	1.00	0.96	0.98	0.96	1.00	0.95	0.97	0.88
Coital frequency	**0.81**	**0.78**	**0.82**	**0.58**	**0.84**	**0.83**	**0.87**	**0.67**
Post-partum abstinence	0.92	0.94	0.97	0.84	0.95	0.95	0.97	0.90
Terminal abstinence	0.98	0.91	0.93	0.82	0.99	0.96	0.98	0.92
Coital frequency	0.89	0.87	0.90	0.76	0.89	0.87	0.90	0.76
Contraception	**1.00**	**0.97**	**0.98**	**0.97**	**0.62**	**0.57**	**0.54**	**0.67**
Contraceptive use: level	1.00	0.97	0.98	0.96	0.68	0.61	0.61	0.63
Contraceptive use: age pattern	1.00	0.97	0.98	0.96	0.62	0.54	0.54	0.57
Condom use	1.00	0.97	0.98	0.97	0.62	0.57	0.54	0.67
Total effect, no interactions	**0.39**	**0.35**	**0.42**	**0.18**	**0.26**	**0.21**	**0.23**	**0.14**
Total effect with interactions	0.45	0.43	0.48	0.23	0.32	0.27	0.29	0.26
Total Fertility - theoretical max	14.6	14.6	14.4	15.9	14.6	14.6	14.4	15.9
Total Fertility - actual	6.5	6.3	6.9	3.7	4.6	3.9	4.1	4.1
Fertility reduction: # children		**-0.2**				**-0.7**		
Fertility reduction: %		**-3%**				**-16%**		

Figure 1. Effect of HIV on the proximate determinants of fertility

a. Less developed country



b. More developed country

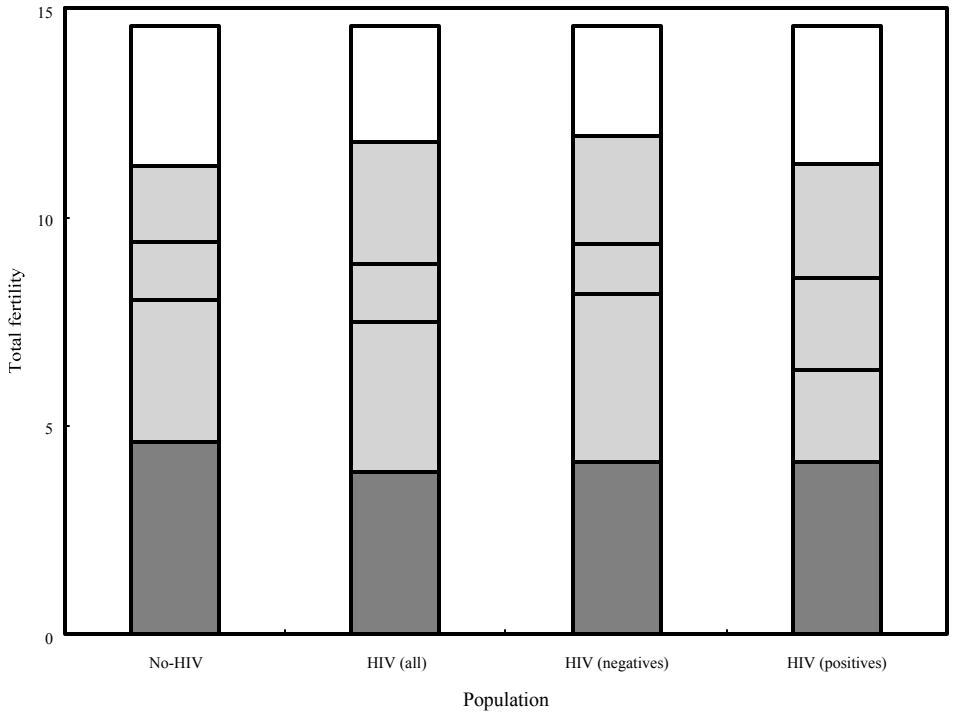
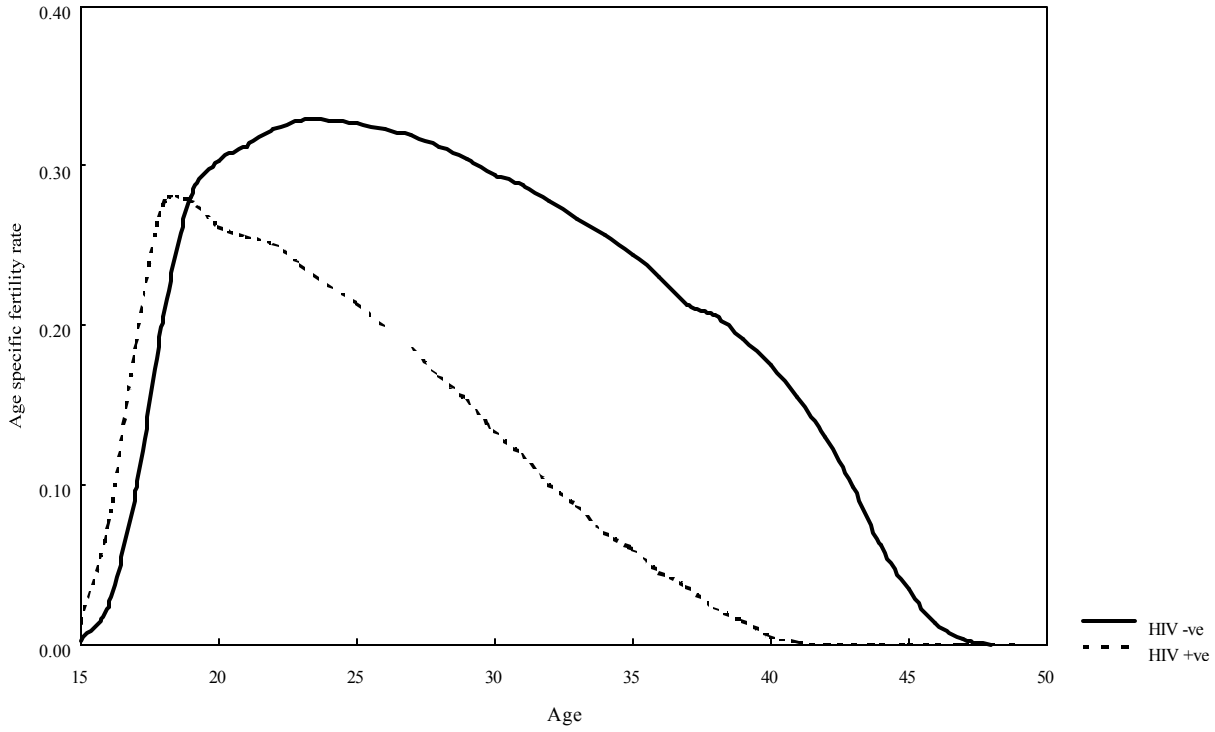


Figure 2. Age specific fertility rates by HIV infection status

a. Less developed country



b. More developed country

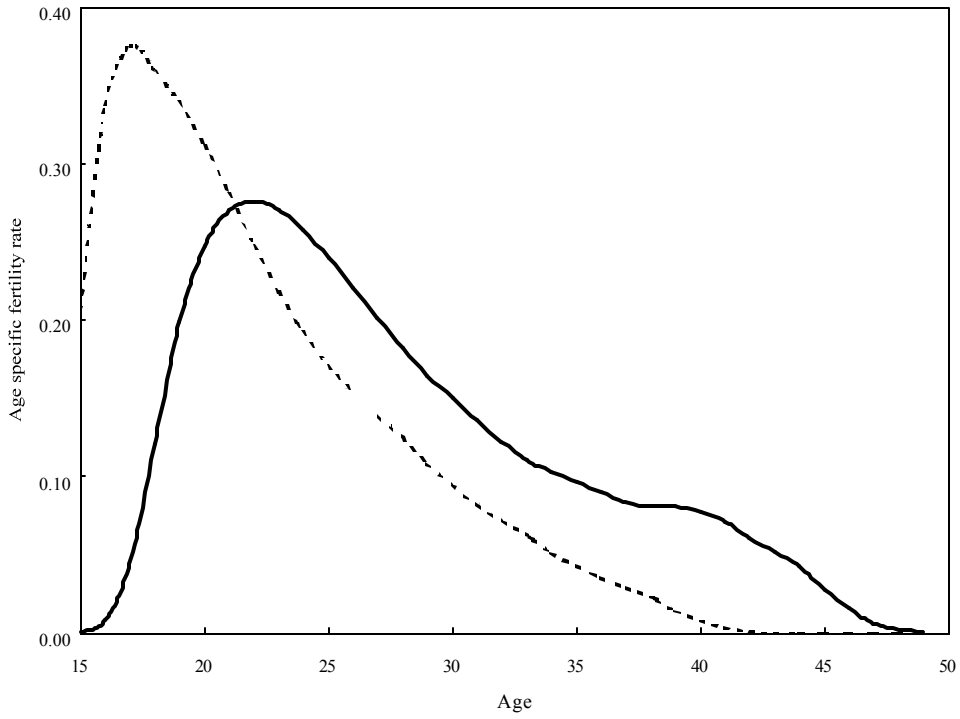
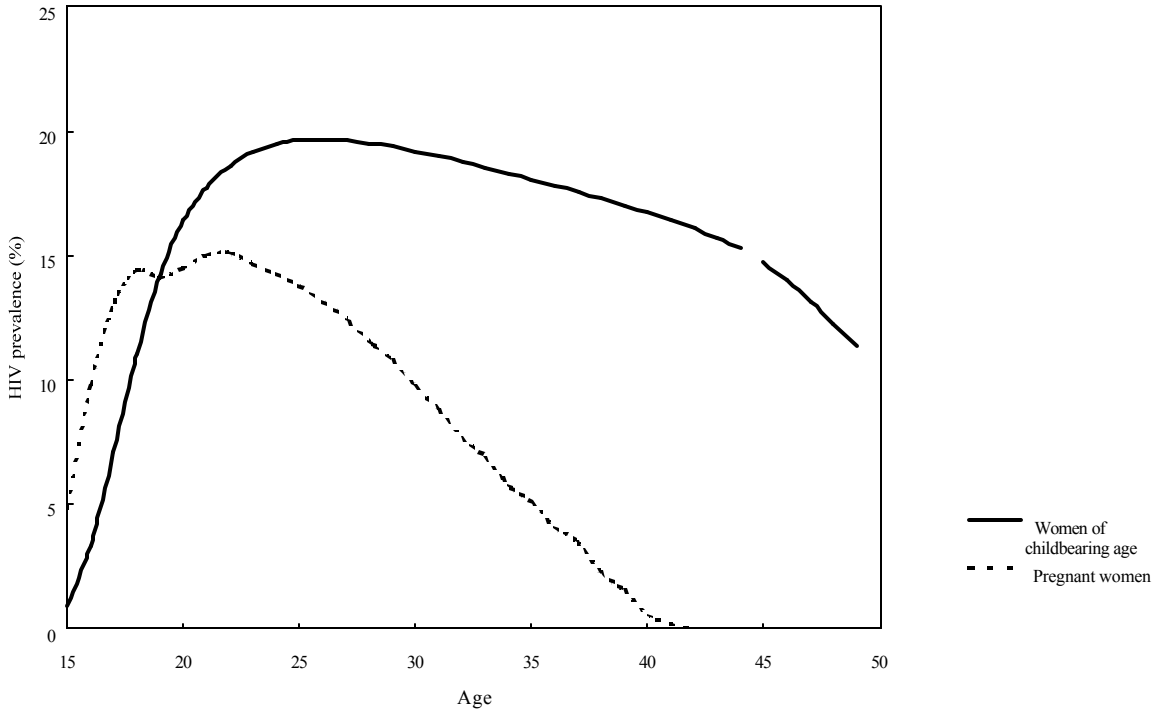


Figure 3. HIV prevalence in the general female population and in pregnant women

a. Less developed country



b. More developed country

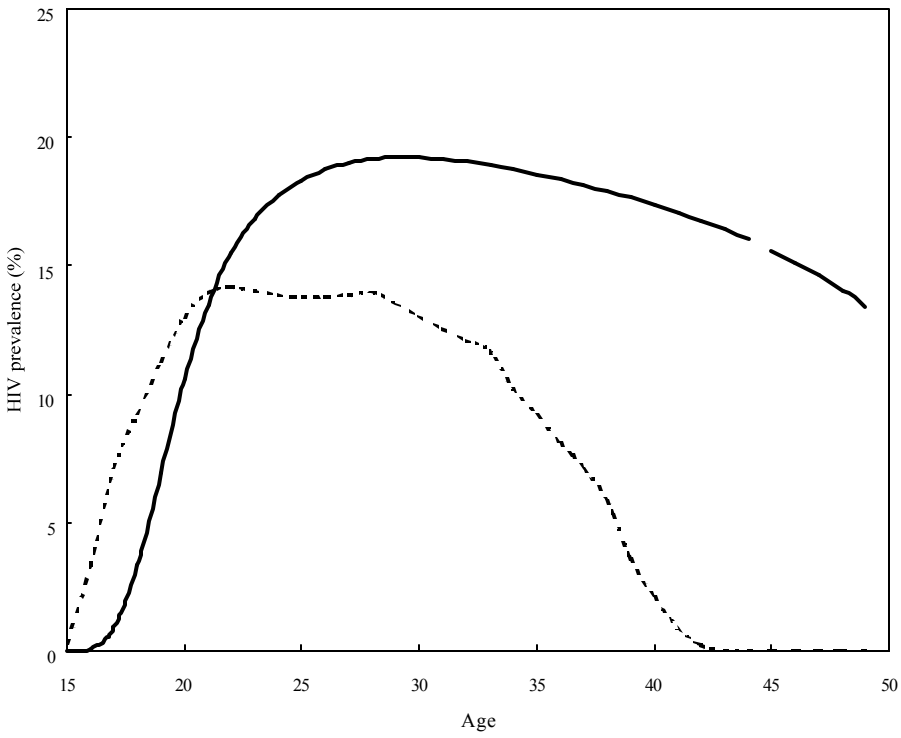
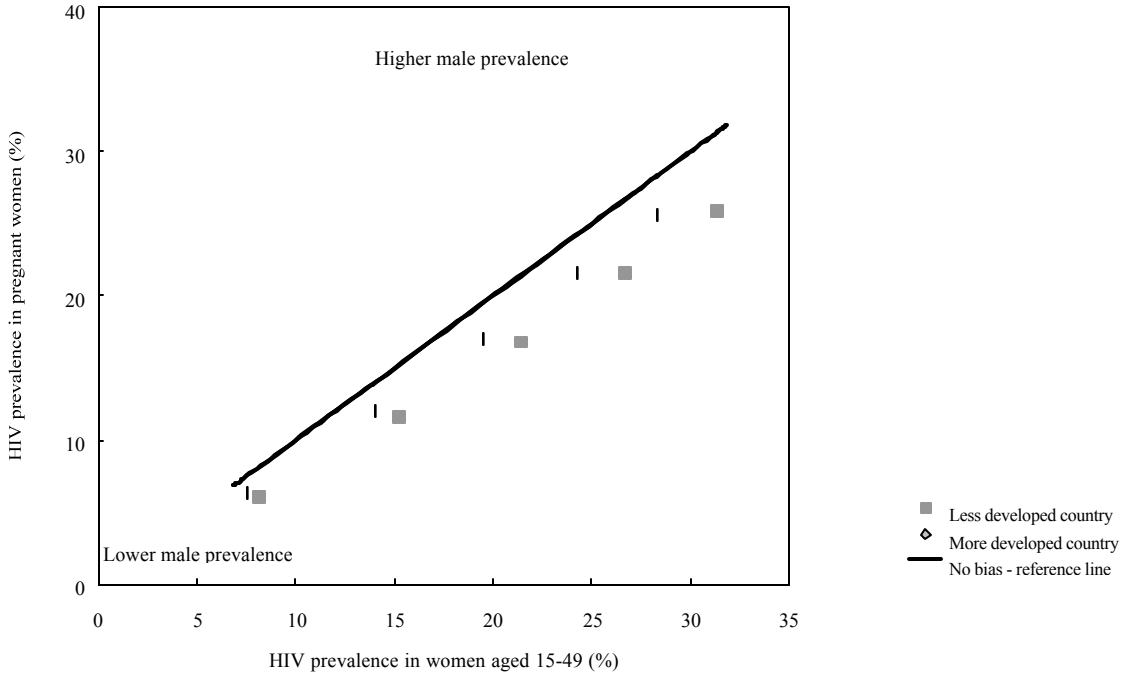


Figure 4. Bias in HIV prevalence estimates based on reports of pregnant women

a. Varying male HIV prevalence



b. Varying extent of condom use

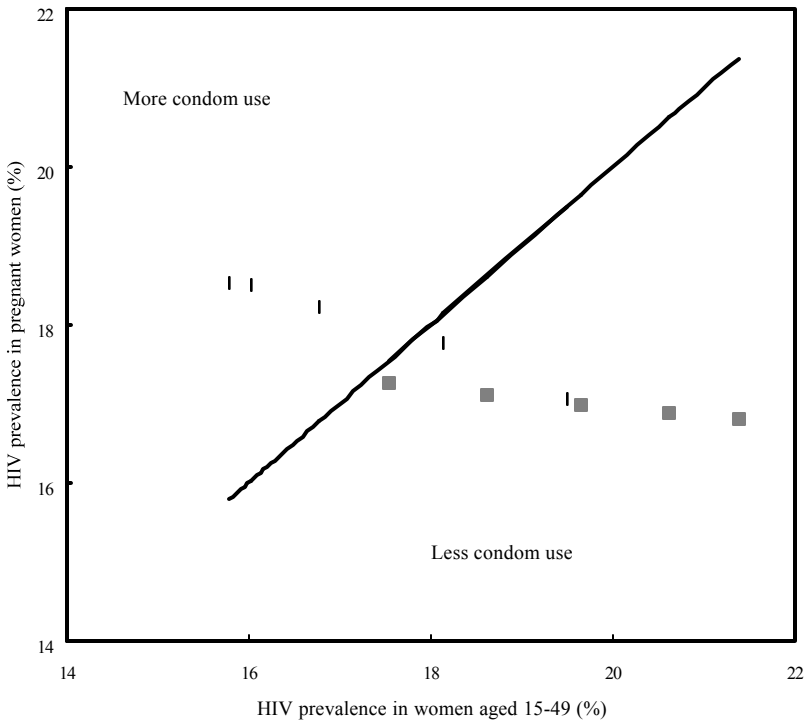
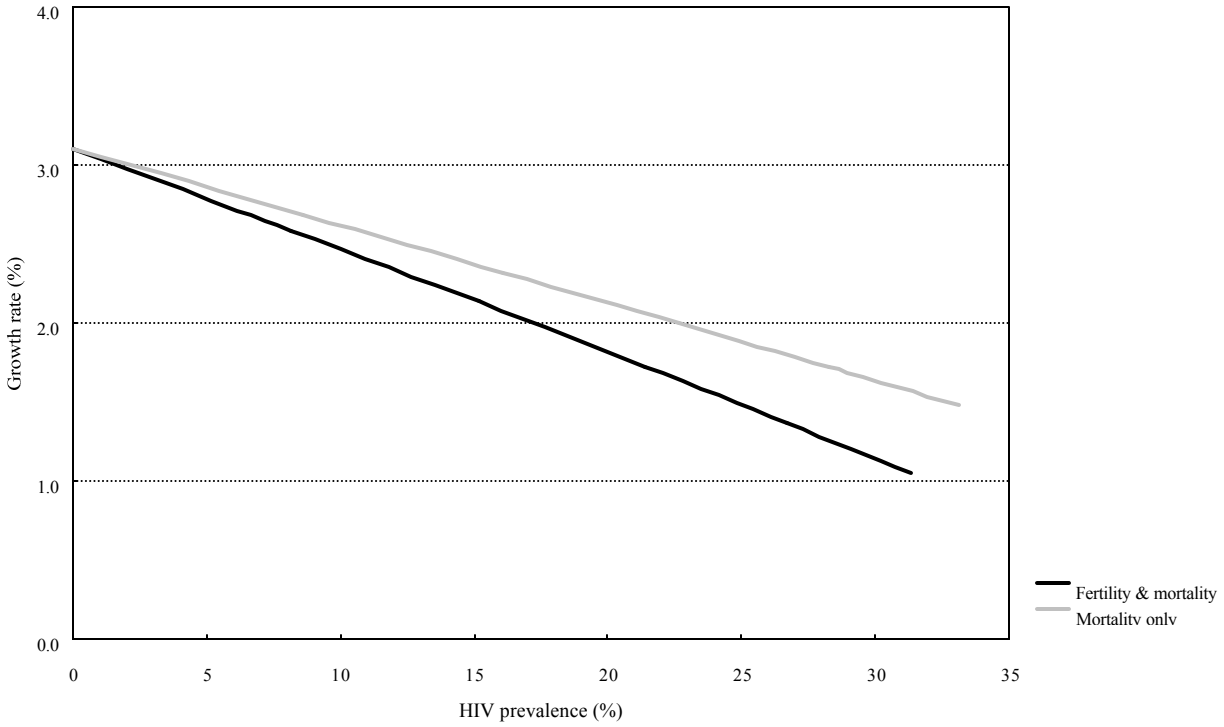
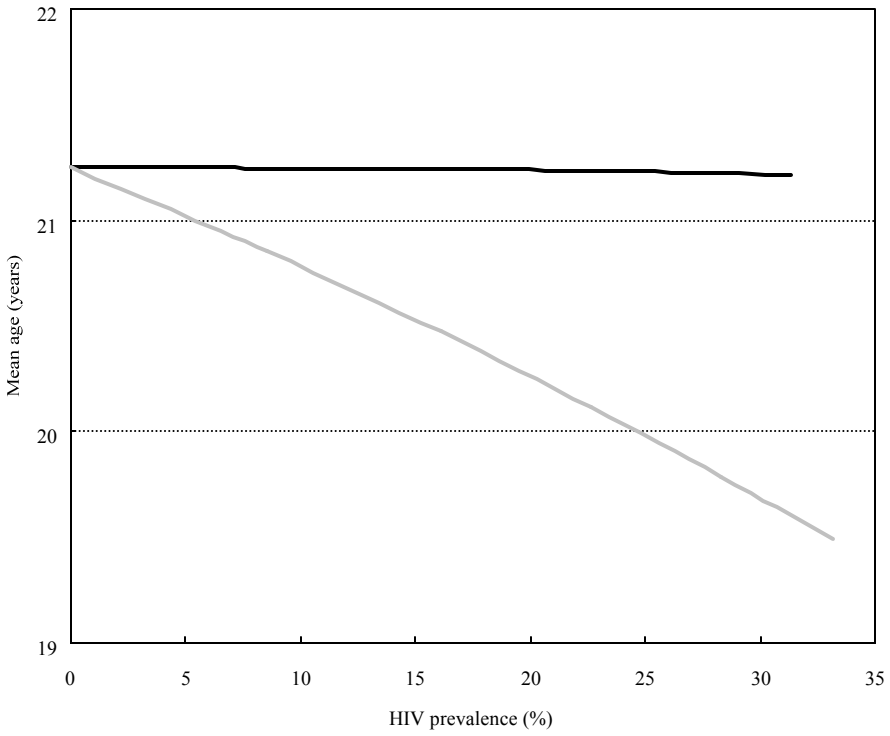


Figure 5. Demographic effects of HIV associated with fertility and mortality changes

a. Population growth (rate of natural increase)



b. Age structure (mean age)



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NOTES

[1]For example, Ankrah suggests that “women may have more pregnancies to offset the perceived threat of infant mortality and ensure the survival of at least some offspring” (PANOS Institute 1992).

[2]Participants in Mukiza-Gapere and Ntozi's focus groups reported instances of both increases and reductions in marital stability. Some stated that unfaithfulness is more likely to result in divorce than in the past while others maintained that the epidemic had reduced episodes of unfaithfulness, so that divorce was now less common (Mukiza-Gapere and Ntozi 1996).

[3]Many authors have noted the cultural obstacles to achieving widespread condom use in African societies (Ankrah, 1991). Others have pointed to the potential for more active education on condoms (Mbizvo and Adamchak, 1989) and the desirability of confronting men directly as "fully rational humane beings who understand what is vital for the protection and assurance of his own survival and that of his wife and posterity" on the need for a change in attitudes and behaviour, particularly in the context of severe HIV epidemics.

[4]These mean survival times correspond to median survival times of around 8 and 1.5 years, respectively.