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**Has the HIV epidemic peaked?**

John Bongaarts (Population Council)  
Thomas Buettner (United Nations)  
Gerhard Heilig (United Nations)  
François Pelletier (United Nations)

**Abstract**

This study first reviews the highly diverse regional and country patterns of HIV epidemics and discusses possible causes of the geographic variation in epidemic sizes. Past trends and projections of the epidemics are presented next and the peak years of epidemics are estimated. The potential future impact of new prevention technologies is briefly assessed. A final section summarizes the future impact of the epidemic on key demographic variables. The main finding of this analysis is that the HIV epidemic reached a major turning point over the past decade. The peak years of HIV incidence rates are past for all regions and the peaks of prevalence rates are mostly in the past except in Eastern Europe where it is expected to peak in 2008. But in part due to the life-prolonging effect of ART and population growth, the absolute number of infected individuals is expected to keep growing slowly in sub-Saharan Africa and remain near current levels worldwide, thus posing a continuing challenge to public health programs. No country is expected to see a decline in population size between 2005 and 2050 due to high mortality related to AIDS.

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Over the past quarter century the HIV virus has spread to all corners of the globe, resulting in one of the deadliest epidemics of modern times. In 2007 a total of 2.1 million men, women and children died of AIDS. The death toll will remain high in the future because 33.2 million individuals are currently infected and about 2.5 million new HIV infections occur each year (UNAIDS 2007). Most of these currently and newly infected individuals are likely to die of AIDS eventually despite the increasing availability of treatment.

In response to this unprecedented health threat a massive global effort has been mounted to reduce infections through prevention programs and to extend the lives of infected individuals with antiretroviral therapy (ART). Prevention efforts are partly responsible for a major turning point in the epidemic: After a period of rapid spread the epidemic appears to have stabilized in most countries and the proportion of adults infected with HIV is no longer increasing (Shelton et al. 2006, UNAIDS 2007). In a number of countries the proportion infected is even declining.

The first part of this article reviews the highly diverse regional and country patterns of HIV epidemics and discusses possible causes of the geographic variation in epidemic sizes. Past trends in epidemics are presented and explanations are offered for the recent stabilization and/or decline of the epidemic. Next, projections of the future course of the epidemic are examined and the peak years of the epidemics are estimated. The potential future impact of new prevention technologies is briefly assessed. The study concludes with a summary of the past and projected future impact of the HIV/AIDS epidemic on key demographic variables including the crude death rate, the population age structure and population growth.

### **Current status of the epidemic and causes of geographic variation**

Worldwide approximately 0.8% of adults is infected with HIV. This global average conceals wide variation among regions (UNAIDS 2007). Figure 1 presents regional estimates of HIV prevalence in 2005, measured as the percent of adults aged 15-49 that is currently infected. Prevalence in all world regions outside sub-Saharan Africa is a fraction of one percent. In contrast, prevalence in sub-Saharan Africa is 5.0% -- an order of magnitude larger than in the rest of the world. Even larger differences in epidemics exist within sub-Saharan Africa where prevalence levels range from a fraction of a percent in several countries in Western Africa to above 15% in some countries in Eastern and Southern Africa. The astounding concentration of the epidemic in a small region of the world is clear from the world map in Figure 2. Within-country variation in HIV prevalence can also be large, but this issue will not be addressed here.

It should be noted that estimates of HIV prevalence and incidence are uncertain for many countries. This is especially the case in developing countries that have not had a nationally representative survey to measure prevalence. Our analysis therefore emphasizes regional patterns which are somewhat more robust. Unless otherwise noted all country and regional estimates and projections in this study are taken or calculated from data provided by United Nations (2007) updated with estimates from UNAIDS (2007). See appendix for further details.

Large generalized epidemics are devastating but they are also relatively rare. This finding is consistent with the fact that HIV is not a particularly infectious agent in heterosexual relationships which are the dominant mode of transmission in most countries. On average the risk of transmission between an infected man or woman and his or her uninfected heterosexual partner is about 1 in 1000 (Gray et al. 2001; Wawer et al 2005)<sup>1</sup>. This low level of infectiousness apparently is sufficient to keep the epidemic confined to a small portion of the population in most countries. The reasons for the explosive epidemics in parts of Eastern and Southern Africa remain controversial but the following factors are considered critical to the rapid spread of the vi-

rus (Cameron et al. 1989; Caldwell 2000; Halpern and Epstein 2007; Quinn et al. 2000; Shapiro 2002):

*-Multiple and concurrent sexual partners.* Since the HIV virus is spread primarily through sexual contact between men and women in sub-Saharan Africa it is reasonable to assume that variation in sexual behavior is a source of variation in epidemics. But frequency of sex alone is not a useful indicator of risk because a heterosexual epidemic cannot occur in a population in which all sex occurs in monogamous unions. The key factor is not frequency of intercourse but frequency of partner change and the number of concurrent partners<sup>2</sup>. Epidemiological models have demonstrated the critical role of a high rate of new partner acquisition and sexual mixing patterns in the spread of HIV (Anderson 1996, 1999). For uninfected individuals frequent partner change raises the risk of encountering an infected partner, and for infected individuals any new partner implies a new chance to spread the infection. Concurrent partnerships appear to be particularly conducive to the spread of HIV through networks of individuals and these partnerships appear to be more common in sub-Saharan Africa than in other world regions (Epstein 2007; Morris and Kretzmar 1997; Halperin and Epstein 2004, 2007; Orubuloye et al. 1994; Swidler and Watkins 2007). Unfortunately, this topic is difficult to study because nationally representative estimates of sexual behavior have limited data on partner concurrency and the validity and reliability of self reported behavior on this topic is unclear (Cleland et al. 2004).

Indirect evidence supporting the key role of multiple partners is provided by a recent study of the ecological correlation between HIV prevalence and the age at first sex and first marriage among females by Bongaarts (2007). This analysis of aggregate data from 33 sub-Saharan countries found no correlation between HIV prevalence and the average age at first sex, but epidemic size was strongly and significantly correlated to the average age at marriage and to the interval between age at first sex and first marriage.<sup>2</sup> Countries with very large HIV epidemics (Botswana, South Africa and Namibia) have ages at marriage in the mid 20s which is much higher than elsewhere in the continent. Late age at marriage results in a long period of premarital sex because age at first sex in these late marrying populations is not higher than elsewhere. This long interval of premarital sex apparently puts women at elevated risk due to frequent partner change, multiple concurrent partners and high partner infectiousness, thus facilitating the spread of HIV.

*-Absence of male circumcision*

A potential role for a protective effect of male circumcision was first proposed in the late 1980s when ecological and prospective clinical studies in sub-Saharan African populations found a highly significant inverse correlation between levels of male circumcision and HIV prevalence (Bongaarts et al. 1989; Cameron et al. 1989; Moses et al. 1990). Recent reviews of the accumulated evidence confirm the higher risk of HIV infection among uncircumcised males (Bailey et al. 2001; Halperin and Bailey et al. 2001; Weiss et al. 2000; Epstein 2007). These findings have been controversial and proposals to introduce male circumcision as a preventive measure have not been actively considered until very recently. Any doubts about the effects of male circumcision were removed with the conclusion of three recent randomized controlled trials in Kenya, South Africa, and Uganda. (Auvert et al. 2005; Gray et al. 2007; Bailey et al 2007). The reduction in the risk of infection in these three trials was 50- 60%, thus demonstrating conclusively that male circumcision is protective. It is unfortunate that circumcision programs were not implemented earlier.

The main biological explanation for the observed protective effect of male circumcision on HIV acquisition involves the dendritic cells on the inner side of the foreskin. These cells are particularly susceptible to HIV infection, and circumcision removes this vulnerable point of entry for HIV (Vachot et al. 2006). In addition to this direct effect, uncircumcised men are at higher risk of infection with certain other sexually transmitted infections (STIs), such as chancroid and syphilis, which have been associated with increased risk of transmission of HIV (Weiss et al. 2000). Male circumcision is thus likely to have both a direct effect on transmission as well as an indirect effect by reducing the incidence of some ulcerative STIs. It should be emphasized that the protective effect of male circumcision is partial and circumcised men are still at risk although at lower level; they should therefore continue to use condoms.

*-Other sexually transmitted diseases.* Certain STIs (e.g. chancroid, genital herpes and syphilis) raise the risk of transmission of HIV directly by causing genital ulcers (Auvert et al. 2001; Bailey et al. 2001; Brown et al. 2007; Cameron et al. 1989; Fleming and Wasserheit 1999). In addition, STIs may have an indirect effect on HIV transmission by raising virus levels in the blood and other bodily secretions (Quinn et al. 2000). STIs are more common in sub-Saharan Africa than in other regions of the world (Chin 2007, WHO 1999).

*-Absence of condom use.* If used consistently condoms provide a high degree of protection against HIV infection as well as against infection with other STIs. Condom use is rare (about 1%) among married couples in sub-Saharan Africa (United Nations 2006) and even among men having commercial sex, on average only half report using a condom at the last sexual intercourse (DHS 2007). In generalized heterosexual epidemics compliance is below the level needed to have a significant impact on the epidemic (Hearst and Chen 2004).

It is beyond the scope of this study to quantify the role of each of these factors in determining the size of HIV heterosexual epidemics. Such an analysis is difficult due to the lack of reliable measures of these determinants. It should be noted, however, that apparently all four of these factors are present in the largest epidemics. The presence of one or two alone is not sufficient to cause substantial epidemics. For example, male circumcision is rare in much of Western Europe, marriage is late and most men and women have multiple partners during their sexually active years. Yet HIV prevalence is very low among heterosexuals in Western Europe. The likely explanation is the protective effects of condom use, the near absence of other STIs and the lower prevalence of concurrent partnerships. In contrast, Southern Africa's epidemics are very large because multiple and concurrent partnerships are relatively common, male circumcision and condom use are relatively rare and other STIs are highly prevalent.

### **The evolution of the epidemic**

Trends in HIV epidemics vary between countries and regions but a general pattern consisting of a slow start in the early 1980s or 1990s, followed by a period of rapid expansion and ending with an endemic plateau, is apparent. HIV prevalence levels (adults 15-49) by region from 1980 to 2005 are plotted in Figure 3. (The vertical line in this and other figures separates estimates up to 2005 and projections from 2005 onwards.) Similar patterns are observed for individual countries. For example, Figure 4 presents prevalence trends for sub-Saharan countries. Epidemics vary in their starting date and size, but the general shape of the prevalence pattern is

similar. It is notable that nearly all these epidemics reached their peak in the 1990s or the early 2000s.

The appearance of recent plateaus is one of the epidemic's most interesting and important features. A stable prevalence level implies that the virus is present in a small proportion of the population but it is nevertheless not spreading further. An explanation of this phenomenon is that every population is a heterogeneous mixture of subgroups with widely varying infection risks. At the high end of the risk spectrum are sex workers and their clients, needle sharing intravenous drug users (IDUs) and homosexual men, and at the low end of the spectrum are men and women living in monogamous unions or without sexual partners. The epidemic is often self-sustaining in the former but not in the latter groups. At the onset of an epidemic the virus quickly invades the highest risk groups but it then runs into resistance when the pools of high risk individuals become infected or die out. The overall size of the epidemic depends on the sizes of the different risk groups. In the US and Europe, for example, parts of the homosexual and IDU groups are at relatively high risk, but infection risks are very low for the vast majority of heterosexuals. As a result, HIV prevalence among heterosexuals in these regions is a fraction of one percent. In contrast, Southern African populations have substantial high risk groups of sex workers and their partners, and the virus spreads more readily in the general population through diffuse networks of multiple and concurrent partners. An epidemic reaches a plateau when the virus has achieved maximum penetration of the vulnerable subgroups. This point seems to have been reached in most countries in the world by the early 2000s.

It is of interest that HIV prevalence is actually declining in a number of countries. Such declines could be attributable to an increase in the number of AIDS related deaths, but behavior change provides an alternative partial explanation. For example, in Uganda prevalence was cut almost in half from 11.8 % to 6.7% between 1991 and 2005 (see Figure 4). The causes of this decline are somewhat controversial but the evidence for behavior change is strong. Both men and women report a decline in reported sex with non-regular partners and a rise in condom use (Bessinger et al. 2003; Stoneburner and Low-Beer 2004, Kilian et al. 1999; Singh et al 2003). Similar trends are observed in a prospective study in Eastern Zimbabwe which documented a decline in HIV prevalence associated with changes in high risk sexual behavior (Gregson et al. 2006). Substantial declines in high risk behavior and infection rates have also occurred among homosexual men in North America and Europe and among heterosexuals in Thailand during the 1990s (UNAIDS 2006). These examples document that substantial changes in behavior can take place when individuals become aware of the health risks of their behavior. Unfortunately, evidence of such behavior changes is not clear-cut for many other countries in sub-Saharan Africa (Bessinger et al. 2003; United Nations 2005). There is little evidence of behavior change in some of the countries with the largest epidemics in Eastern and Southern Africa despite widespread knowledge of the AIDS epidemic and its causes, but even here the epidemics are no longer growing.

Figures 3 and 4 also include UN projections of HIV prevalence rates from 2005 to 2025. These projections are based on a model developed by the UNAIDS Reference Group on Estimates, Modelling and Projections (UNAIDS Reference Group 2002) that was adopted and enhanced by the Population Division for its own prerequisites. This model is fitted to past estimates of HIV prevalence provided by UNAIDS for each of the affected countries and then extrapolated. In the large majority of countries the projections assume prevalence to continue its slow decline observed before 2005. In the few countries where prevalence is still rising the model predicts a peak and a subsequent decline based on the epidemic pattern observed before 2005. Further details on the projection methodology are provided in an appendix.

### **Has the HIV epidemic peaked?**

The preceding analysis relied on HIV prevalence rates to assess trends in the sizes of epidemics. HIV prevalence is the most widely available indicator of epidemic size and it is readily interpreted. But there are three concerns about this indicator's ability to track the epidemics ups and downs accurately.

The first is that prevalence is a lagging indicator of HIV infections. The numerator of the HIV prevalence rate consists of all currently infected adults (15-49), regardless of the time they were infected. Since individuals survive about a decade after infection, the prevalence estimate in a given year is determined by the number of infections that occurred in the past decade or longer.

The second concern is that HIV prevalence is affected by the treatment of AIDS patients. Contemporary antiretroviral therapy (ART) can be highly effective in keeping AIDS patient alive for years beyond the time associated with the natural course of the disease. This keeps the pool of infected individuals (and hence the HIV prevalence rate) larger than would be the case without treatment.<sup>4</sup>

The third concern is that HIV prevalence is affected by a rise in the AIDS death rate. As the epidemic matures, the death rate tends to rise as infected individuals age and every AIDS death reduces the number of infected people.

For these reasons many epidemiologists prefer another indicator to track epidemic trends: the HIV incidence rate. It is defined as the annual rate of new infections among adults aged 15-49. The total infected population at a point in time is the net result of past additions to the infected population through new infections and subtraction through deaths. The rate of new additions to the pool of infected individuals is measured by the incidence rate.

Figure 5 presents UN estimates of incidence rates by region. The overall pattern over time is broadly the same in the different regions: a rapid rise to a peak followed by a slower decline. As expected, the incidence peak in sub-Saharan Africa (0.8% per year) is far higher than in other continents where the peaks are all less than 0.1 % per year. The timing of the incidence peaks also varies significantly. The first occurred in the 1980s in North America (1983) and Latin America (1987). Sub-Saharan Africa (1991) and Asia (1995). Europe has two peaks, the first in 1983 (in Western Europe) and the second in 2001 in Eastern Europe. The global peak in incidence occurred in 1995 at a rate of 0.1% per year. The ongoing declines in incidence are caused by the same factors noted above for prevalence trends: saturation of high risk groups and changes in behavior, but there is an additional third factor: a decline in the average infectiousness of infected individuals. This decline is a result of a rising average duration since the time of infection as the epidemic matures, combined with a reduction in individual infectiousness with time since infection.

A comparison of Figures 4 and 5 shows that the peaks in incidence precede the peaks in prevalence by nearly a decade. This implies that prevalence continues to rise for a number of years after the incidence rate has begun to decline. This is possible because the prevalence rate is calculated from the number of individuals who are currently infected regardless when in the past the infection occurred. For example in the hypothetical case in which all infected individuals survive exactly 10 years, the prevalence number at any given point in time equals the sum of all infections that occurred in the past ten years, regardless of whether the annual number of new infections was rising or falling. The number of infected individuals reaches a maximum when the

declining number of new infections equals the rising number of AIDS deaths. This point is typically reached a number of years after the peak in HIV incidence.

Figure 6 summarizes the peak years for incidence and prevalence rates for the world and for each major region. The left end of each bar represents the year of the incidence peak and the right end of each bar represents the peak year for prevalence. For example, world incidence peaked in 1995 and world prevalence in 2003, a difference of eight years. As noted, the first incidence peak occurred in North America and the last one in Eastern Europe. The prevalence peaks occur nearly a decade later except in Asia where the delay is shorter. The main finding from this analysis is that the peak years of HIV incidence are past for all regions and the peaks of prevalence are mostly in the past except in Eastern Europe where it is expected to occur in 2008.

The preceding discussion of epidemic trends relied on incidence and prevalence *rates* to quantify epidemic sizes. An alternative measure of epidemic size is simply the absolute number of newly or currently infected individuals. Analysis of absolute numbers has been avoided here because they are affected by population size, thus potentially giving misleading results. For example, in 2006 the number of infected adults in China is estimated to have been more than twice the number of infected individuals in Botswana. But the HIV prevalence rate in Botswana (24%) is more than a hundred times the rate in China (0.1%). It is clear that the societal impact of the epidemic is better measured with the prevalence rate than with the absolute numbers infected. Nevertheless, absolute numbers are sometimes useful, for example for the allocation of international resources to combat the epidemic. Figure 7 presents the absolute annual number of new infections by region. As expected, sub-Saharan Africa has the largest number of annual infections per year. Asia is second because a low incidence is applied to a much larger population. The annual number of new infections worldwide peaked in the mid 1990s at just over 3 million per year, then declines to 2010, followed by a plateau until about 2030 as population growth offsets the continuing decline in the incidence rate. Figure 8 presents absolute numbers of infected adults (15-49) by region. Africa and Asia are again the highest and second highest regions. The world total reached 29 million in 2006 and is expected to remain near this level for several decades. The peak in the number of infected individuals in sub-Saharan Africa is not expected until around 2030. As noted, rapid population growth and the extension of the lives of AIDS patients due to ART are largely responsible for this trend.

### **New prevention technologies and their potential impact**

The future spread of HIV can be reduced with more vigorous prevention efforts. Prevention options can be divided into those that are widely available today and encouraged by most ongoing intervention programs and those that depend on the development and introduction of new technologies. The former include abstinence, reduction in number of partners and condom use. Prevention programs, by broadcasting motivational messages, have encouraged abstinence, sexual fidelity and condom use in many settings, even though the question as to which of these prevention interventions should be given highest priority (in particular the role of abstinence vs. condom use) is controversial (Merson, 2006). Secondary transmission of HIV from an infected mother to her newborn can be prevented by the use of antiretroviral drugs, by encouraging safer infant feeding practices and by improving access to family planning to prevent unwanted pregnancies. Declining prevalence rates in several countries are consistent with the view that these prevention efforts have had an impact. However, despite these encouraging trends in a few populations, high risk behavior remains pervasive and HIV continues to spread in much of the world. In addition, many women who wish to protect themselves lack the cooperation of their partners.

To make prevention efforts more effective, a massive global effort is underway to develop, test and introduce new prevention technologies, such as male circumcision, microbicides and vaccines.

#### *Male circumcision.*

As noted, recent randomized controlled trials confirmed the substantial protection male circumcision provides against HIV infection. Shortly after the publication of these results a joint WHO/UNAIDS Technical Consultation recommended that male circumcision be implemented in areas with a high prevalence of HIV infection (WHO/UNAIDS 2007). Unfortunately, introduction of male circumcision programs is a complex process and further research is needed on a host of critical issues including the acceptability among uncircumcised males, the safety of the surgical procedure, the ability of overburdened health care systems to handle a large volume of procedures, the training of medical personnel, the need to use condoms due to partial effectiveness, the target age group (infants, adolescents or all adult males), and the need to prevent behavioral disinhibition (Sawires et al. 2006; WHO/UNAIDS 2007).

#### *Microbicides.*

A microbicide is a vaginal product (e.g., gel or cream) that blocks the transmission of HIV during sexual intercourse. Microbicides have two highly desirable characteristics: they are under the control of women and they do not require the consent or knowledge of male partners. First-generation microbicides typically form physical barriers against the virus or change the chemistry of a woman's vagina. Ongoing trials will conclude over the next few years and any products found to be effective should be approved, manufactured and distributed relatively quickly. Second-generation microbicides promise to be more effective because they fight viral transmission through multiple mechanisms (e.g. by combining a physical barrier with an anti-retroviral agent) and some of them are designed to increase adherence by making them less coital dependent and longer acting. Several such compounds are now under development (Nutall et al. 2007).

#### *Vaccines.*

A vaccine does not require daily action and is therefore the preferred prevention technology. Unfortunately, no effective vaccine candidates are available and all new experimental vaccines face big obstacles: HIV mutates rapidly to escape immune responses and a single vaccine is unlikely to protect against the many existing HIV variants. As a result, an effective and easily manufactured vaccine is at least a decade away (Johnson and Fauci 2007).

Microbicides and vaccines-- like male circumcision-- will likely be only partially effective, i.e., the user's risk of infection is reduced but not eliminated. Such technologies are nevertheless crucial because each averted infection benefits the individual user directly, but also reduces the risk of future infections in the general population. With consistent use, even moderately effective products can potentially avert millions of deaths.

The future impact of these new technologies on the epidemic depends on how soon they become available, on how acceptable they are to users, on how effective they are if used as recommended and on how diligent users are in following instructions. Variations in each of these factors will lead to widely different outcomes when these technologies are introduced in different populations. To provide an upper and lower boundary on the potential impact of new technology



two projections are compared. The upper boundary for impact is provided by a projection in which all infections stop after the year 2010. This very unlikely scenario could be brought about by discovery of a new 100% effective vaccine and its immediate and universal application. The lower boundary for impact is given by the medium variant projection which assumes no additional prevention technology.

Figure 9 compares the future trends in HIV prevalence rates in these two projections for sub-Saharan Africa. The upper and lower bound scenario diverge after 2010. Although no new infections occur in the upper bound scenario, there is a substantial number of infected persons in 2010 and some of these survive more than a decade. As a result, prevalence declines gradually, reaching half the 2010 level by 2016.

The potential effect of new technologies is substantial but the real world impact will likely be much less than the upper boundary of impact presented here. This is demonstrated by a simulation study of the potential impact of male circumcision by Williams et al. (2006). Assuming an effectiveness level demonstrated in the recent trials and full coverage by 2015, this study estimates for South Africa a reduction by just 19% in the number of infected individuals and by 13% in AIDS deaths below the no intervention scenario for the decade 2015-2025. This is obviously an important step in the right direction, but falls well short of stopping the epidemic.

## **Demographic impact**

The following analysis of the demographic impact of the AIDS epidemic compares two scenarios-- "AIDS" and "No-AIDS" --prepared by the United Nations as part of its 2006 assessment (United Nations 2007). The AIDS scenario is simply the medium projection variant, which includes the effect of the epidemic, while the No-AIDS scenario represents a projection in which the mortality of uninfected individuals is applied to the entire population<sup>5</sup>. It should be noted that the AIDS scenario does not incorporate the updates of HIV prevalence estimates made in 2007 in selected countries. The epidemic's demographic impact may therefore be slightly overestimated.

The epidemic directly affects standard demographic indicators of population mortality such as the crude death rate and life expectancy. Effects on fertility and migration also exists, but they are smaller and more complex (United Nations 2002). Changes in vital rates in turn affect population growth and the age structure. To simplify the presentation of these demographic effects the discussion will be limited to three key indicators:

### *Crude death rate*

Figures 10 A and 10 B plot the estimated past and projected future crude death rates with and without AIDS by world region for 1980-2050. As expected, the difference between these two scenarios is largest in sub-Saharan Africa. In this region, the death rate declines steadily over time in the No-AIDS scenario, while it pauses between 1985 and 2005 in the AIDS scenario before resuming a decline. The crude death rate in the AIDS scenario exceeds the rate in the No-AIDS scenario by 22% in 2000-2005 and this difference is projected to decline slightly by the end of the projection period. In the other regions the impact of the epidemic is an order of magnitude smaller (see Figure 10 B). The largest future differences between the regional AIDS and No-AIDS scenarios range from 2 to 3%.

### *Population age structure*

The rise in death rates due to AIDS varies widely by age with the largest effects among adults aged 20-49 and among young children who are infected by their mothers around the time of birth. As a result, the epidemic has an impact on the distribution of the population by age. This effect is very small in all regions except sub-Saharan Africa. Figure 11 compares the age distribution in 2015 of sub-Saharan Africa for the two scenarios. The largest effects occur in the age groups above age 30 and below age 15, reflecting the impact of AIDS deaths that occurred before 2015. The decline in the number of children is also caused by a projected reduction in the number of women of reproductive age due to AIDS mortality. In the most heavily affected countries the changes in the age structure are more pronounced. This is evident in Figure 12 which plots the AIDS and No-AIDS age distributions for South Africa in 2015. These results are broadly consistent with the in-depth analysis of the impact of the epidemic on the population age structure by Dorrington et al. (2006).

### *Population growth*

Excess deaths due to the HIV epidemic can have a substantial impact on population growth. Figure 13 compares regional population growth between 2005 and 2050 with and without the AIDS epidemic. In all regions the epidemic leads to lower population size in 2050, but outside sub-Saharan Africa the effect is a fraction of one percent and hence not visible in Figure 13. The largest impact is forecast for sub-Saharan Africa where population in 2050 is projected to reach 1.95 billion without the epidemic and 1.76 billion with the epidemic (see Figure 14). The difference between these projections is due to the direct effects of AIDS deaths and the absence of descendants from individuals who have died from AIDS. A key implication of this finding is that despite the huge mortality associated with the AIDS epidemic the population of sub-Saharan Africa is expected to grow by 1 billion between 2005 and 2050.

The impact on future population growth is of course larger in Southern Africa. For example, as a result of the AIDS epidemic, population size in 2050 is projected to be reduced below the No-AIDS scenario by 29% in South Africa and by 25 % in Botswana. But in both countries population growth remains positive over the next several decades. The explanation for this finding is that the birth rate in these countries is much higher than the death rate. As a result, even a doubling of the death rate leaves the birth rate higher than the death rate and population growth continues although at a lower rate. For example, in Botswana the birth and death rates without AIDS in 2005-10 would have been 2.6% and 0.6% per year respectively, leading to a natural growth rate of 2% per year. The epidemic more than doubled the death rate to 1.6% which –with the same birth rate- gives a natural growth rate of 1%. So this large epidemic reduced the growth rate from 2 to 1 % per year but it remains positive. No country in the world is expected to see a decline in population size between 2005 and 2050 due to higher mortality related to AIDS. Earlier predictions of possible population declines in Africa (e.g. Garnett and Anderson, 1993) therefore appear to have been too pessimistic.

### **Conclusion**

Over the past quarter century the HIV virus has spread very unevenly around the world with very large epidemics in parts of Eastern and Southern Africa and relatively small epidemics outside Africa. The explanation for this unexpected variation lies in several epidemiological factors. The largest epidemics occur in populations where multiple and concurrent partners are relatively common, where condom use and male circumcision are rare and where ulcerating STIs are

highly prevalent. In the absence of most of these factors the HIV virus has difficulty establishing and sustaining itself and the HIV prevalence rate is typically less than 1%.

The answer to the question “Has the epidemic peaked?” depends on which indicator is used to assess epidemic trends. Although statistics on the evolution of the epidemic often contain substantial measurement errors, it appears that the incidence rate has peaked in all world regions and is now declining. The same is true for the prevalence rate although the peak occurred later. Ongoing declines in incidence and prevalence rates are probably attributable to behavior change and the natural burn-out of epidemics which leads to the saturation of high risk groups.

A somewhat different picture emerges when epidemic size is measured in terms of absolute numbers of infections and infected individuals. A peak in the annual number of new infections occurred in the past decade in all regions; these peaks are expected to be followed by steady declines in all regions except sub-Saharan Africa. In this region a rebound is expected as a slowly declining incidence rate is offset by a rapidly rising population size. The world total annual number of new infections is projected to remain close to current levels for the next quarter century.

The HIV epidemic reached a major turning point over the past decade. Incidence and prevalence rates have peaked in all world regions between the early 1990s and 2008. But in part due to the life-prolonging effect of ART and population growth, the number of infected individuals is expected to keep growing slowly in sub-Saharan Africa and remain near current levels worldwide, thus posing a continuing challenge to public health programs. These findings indicate a continuing need to develop new prevention technologies and for prevention and treatment programs especially in the countries with substantial epidemics. They may also lead to discussion and possible revision of priorities assigned to the HIV/AIDS epidemic relative to other public health challenges in countries where the epidemic is declining.

## End notes

1. Infectiousness varies with duration of infection with the most infectious periods immediately following infection and after the onset of AIDS (Wawer et al. 2005; Halperin and Epstein, 2007; Shiboshki and Padian, 1998).
2. There is no precise standard definition of the term “concurrency”, but it generally refers to an individual having sexual relationship with more than one partner within a brief interval of time, e.g. a few weeks. This behaviour facilitates the rapid spread of the epidemic because an infected individual is highly infectious for several weeks immediately following the time of infection (see endnote 1)
3. A regression analysis of data from 33 sub-Saharan countries found that male circumcision and duration of premarital sex together explain 64% of the variance in HIV prevalence (Bongaarts 2007).
4. ART has additional fairly complex and not well measured effects on the spread of the incidence of HIV, including a reduction in viral load in infected individuals (making them less infectious to others), possible behavioural disinhibition and the emergence of drug resistance with poor adherence to treatment.
5. The mortality of those who are not infected is not the same as the mortality that would have been experienced if the epidemic had never occurred. This distinction is of little importance during the early years of the epidemic or in countries with small epidemics, but is more important when a sizeable part of the population is infected and ultimately dies of AIDS. The difference is the result of the assumption that the epidemic is likely to affect the capacity of countries to provide health care services to its population, including uninfected people. Therefore, the assumed trends in No-AIDS mortality are somewhat less optimistic than if the epidemic would never have occurred. Nonetheless, the difference between the AIDS and No-AIDS scenarios provide a useful indicator of the epidemic’s impact.

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## **Appendix: Projecting the demographic impact of HIV/AIDS epidemic**

In the *2006 Revision* of the United Nations population projections, all countries with a HIV prevalence among persons aged 15 to 49 equal to or greater than one per cent are considered as seriously affected by the HIV/AIDS epidemic and their mortality is projected by modeling explicitly the course of the epidemic and projecting the yearly incidence of HIV infection. Also considered among the affected countries are those where HIV prevalence is lower than one per cent but whose populations are so large that the number of individuals infected is large, such as Brazil, China, India and the United States of America. In total, the course of the epidemic is explicitly modeled for 62 countries.

The model developed by the UNAIDS Reference Group on Estimates, Modelling and Projections (2002), and adopted by the Population Division for its own prerequisites, was used to fit past estimates of HIV prevalence provided by UNAIDS in 2006 for each of the affected countries. Prevalence levels for countries with large revisions in 2007 were adjusted so as to approximately reflect updates from UNAID (2007). The fitting exercise produces estimates of the model parameters that determining the past dynamics of the epidemic for each country. For most countries, the model parameters are held constant in the past, but beginning in 2005, the parameter reflecting the rate of recruitment of new individuals into the high-risk or susceptible group is projected to decline by half every twenty years, and the parameter representing the force of infection, is projected to decline by half every thirty years. These reductions reflect the assumption that changes in behavior among those subject to the risk of infection, along with increases in access to treatment for those infected, will reduce the chances of transmitting the virus. In addition, the increased survival time of those receiving treatment gradually increases the survival time of all infected persons.

Prevention of mother-to-child transmission is modeled using estimated country-specific coverage rates that average 13 per cent in 2005 among the 62 affected countries, but vary from 0 to 90 per cent among them. These coverage rates are projected to reach 60 per cent on average by 2015, varying between 40 per cent and 100 per cent among the affected countries (UNAIDS, UNICEF, WHO 2007). After 2015, the coverage rate is assumed to remain constant at the level reached in each of the affected countries until the end of the projection period. Among women receiving treatment, the probability of transmission from mother to child is assumed to be 1 per cent. These assumptions produce a reduction in the incidence of HIV infection among children born to HIV-positive women, but the size of the reductions varies from country to country depending on the level of coverage that treatment reaches in each country (Stover et al. 2006).

The survivorship of infected children takes account of varying access to pediatric treatment. HIV-infected children are divided into two groups: (i) for those infected in-utero, among whom the disease progresses rapidly, average survival is modeled to be 1.3 years, and (ii) for those infected after birth through breastfeeding, among whom the disease progresses slowly, average survival is 14 years without treatment (Stover et al. 2006; Boerma et al. 2006). Explicit inclusion of pediatric treatment is done via country-specific coverage rates which average 9 per cent in 2005 but vary between 0 and 99 per cent among the 62 affected countries. By 2015, the projected coverage is expected to reach 60 per cent on average varying from 40 per cent to 100 per cent among the affected countries (UNAIDS, UNICEF, WHO 2007). Coverage levels remain constant from 2015 to 2050 at the level reached in each country by 2015. The annual survival of children receiving treatment is 95 per cent, so that their mean survival time is 19.5 years and the median survival time is 13.5 years in the absence of other causes of death (Stover et al. 2006).

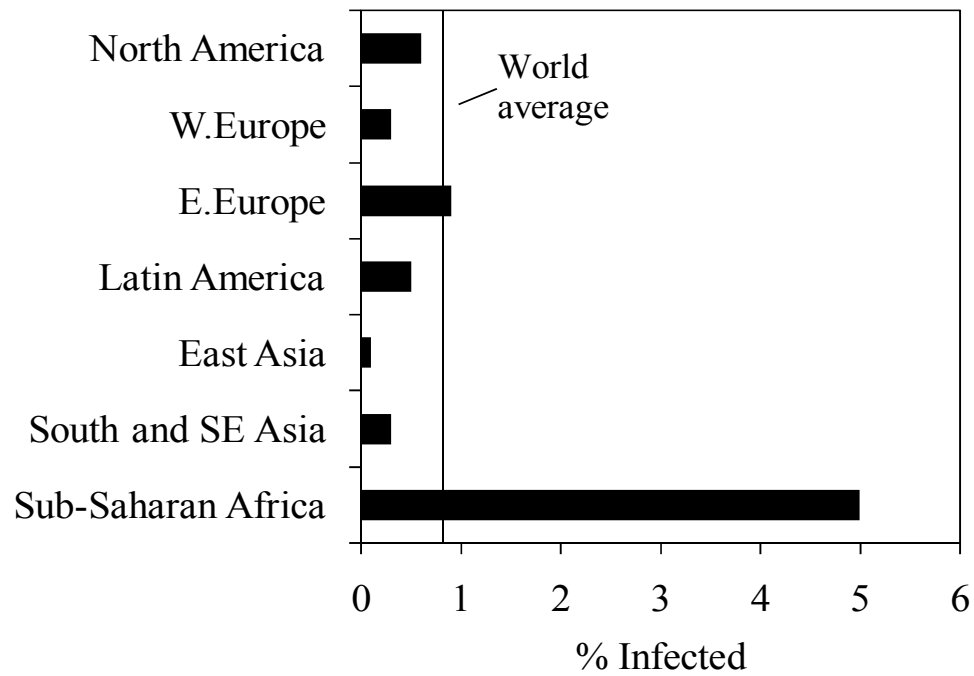
The *2006 Revision* incorporates a longer survival for persons receiving treatment with highly active antiretroviral therapy (ART). The proportion of the HIV-positive population receiving treatment in each country is consistent with estimates prepared by the World Health Organization, which average 25 per cent in 2005 but varied between 0 and 100 per cent among the 62 affected countries (WHO and UNAIDS, 2006). Coverage is projected to reach between 40 per cent and 100 per cent by 2015, averaging 60 per cent for the affected countries. Between 2015 and 2050, coverage levels remain constant at the level reached in each country by 2015. It is assumed that adults receiving treatment have, on average, a 90 per cent chance of surviving each year in the absence of other causes of death. Under this assumption, mean survival time after the initiation of therapy is 9.5 years and the median survival time is 6.6 years, in the absence of other causes of death. Therapy is assumed to start at the time full-blown AIDS develops. Without treatment, infected adults have a mean survival time of two years after the onset of full-blown AIDS.

The above procedures apply to the 62 AIDS countries included in the *2006 Revision*. For the present study the following additional assumptions were made to obtain comprehensive regional estimates of various indicators.

1) According to estimates of the number of infected individuals for all countries in the world prepared by UNAIDS (2006), the 62 AIDS countries in the *2006 Revision* cover 92.8% of infected adults in the world. To adjust for this difference all other “non-AIDS” countries have been given an HIV prevalence rate of 0.17% and an incidence rate 0.018% in 2005. In each region the time pattern of prevalence and incidence in the non-AIDS countries is assumed the same as in the AIDS countries, except for Europe where the non-AIDS countries are in Western Europe and the AIDS countries are in Eastern Europe. The Western European time pattern is assumed the same as in the US (except at a lower level)

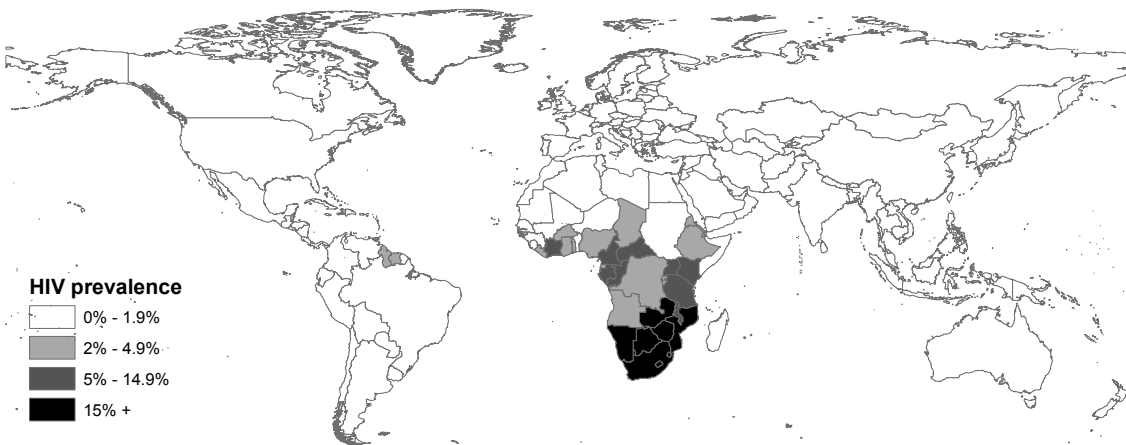
2) According to estimates of AIDS deaths for all countries in the world prepared by UNAIDS (2006), the 62 AIDS countries in the *2006 Revision* cover 96% of AIDS deaths in the world. AIDS deaths in every region have been inflated by a factor  $1/0.96$  to obtain regional totals.

Figure 1: HIV prevalence rate, adults 15-49, by region, 2005



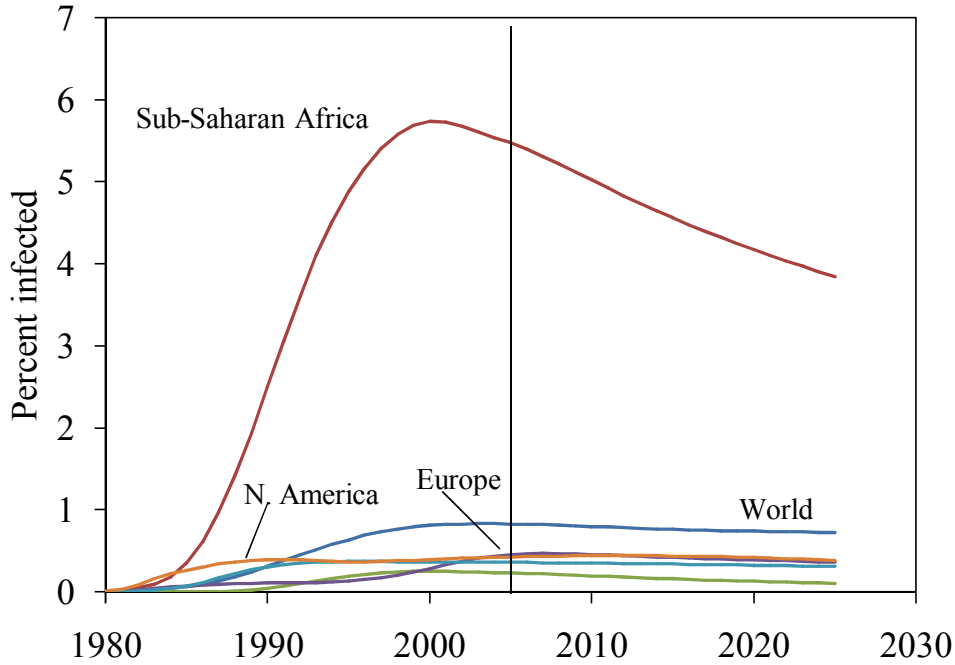
Source: UNAIDS 2007

Figure 2 Geographic distribution of the HIV epidemic



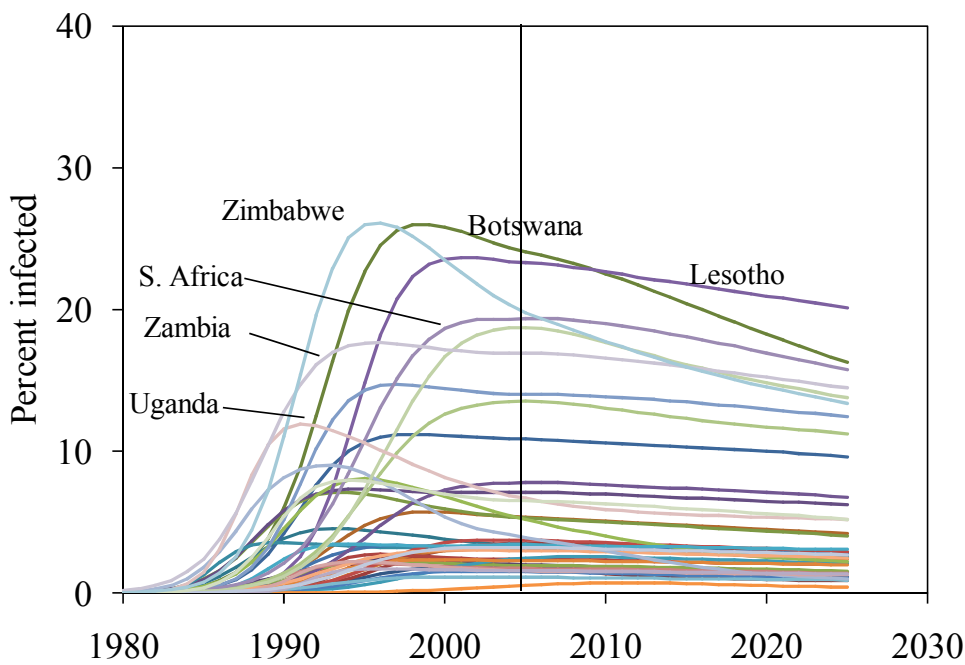
Source: United Nations 2007

Figure 3: HIV prevalence rate by region, estimates and projections



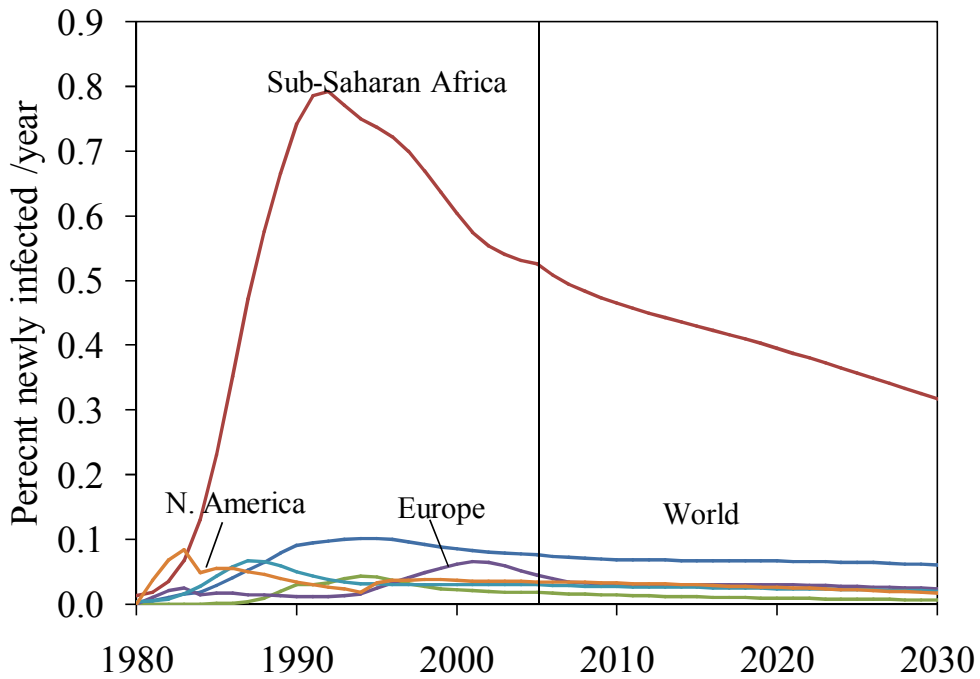
Source: UN 2007

Figure 4: HIV prevalence rate in 40 countries in sub-Saharan Africa, estimates and projections



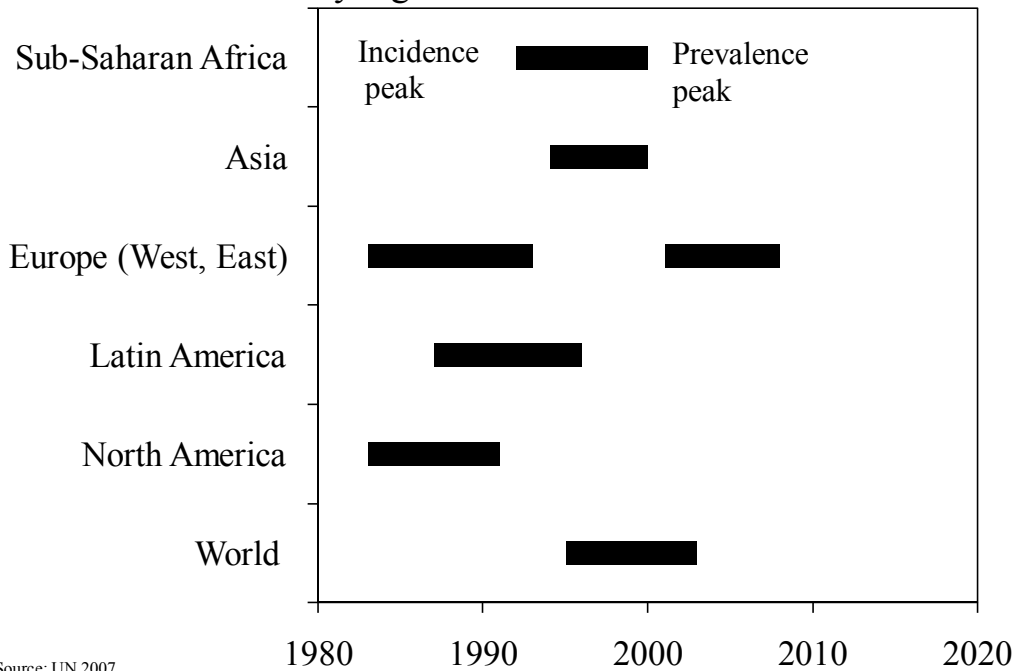
Source: UN 2007

Figure 5: HIV incidence rates by region, estimates and projections



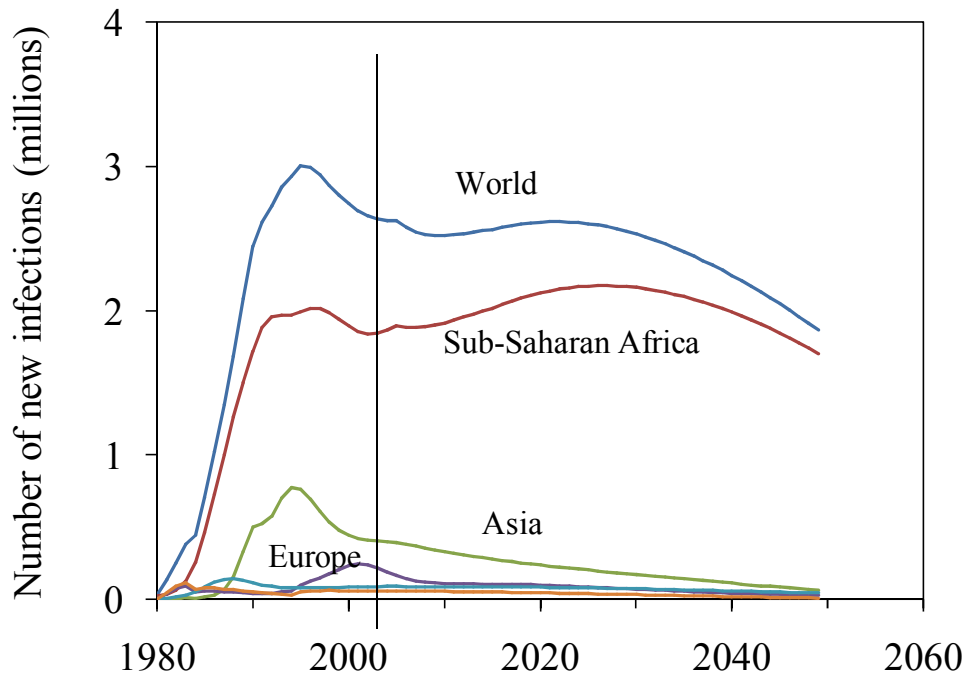
Source: UN 2007

Figure 6: Peak years of past incidence and prevalence rates by region



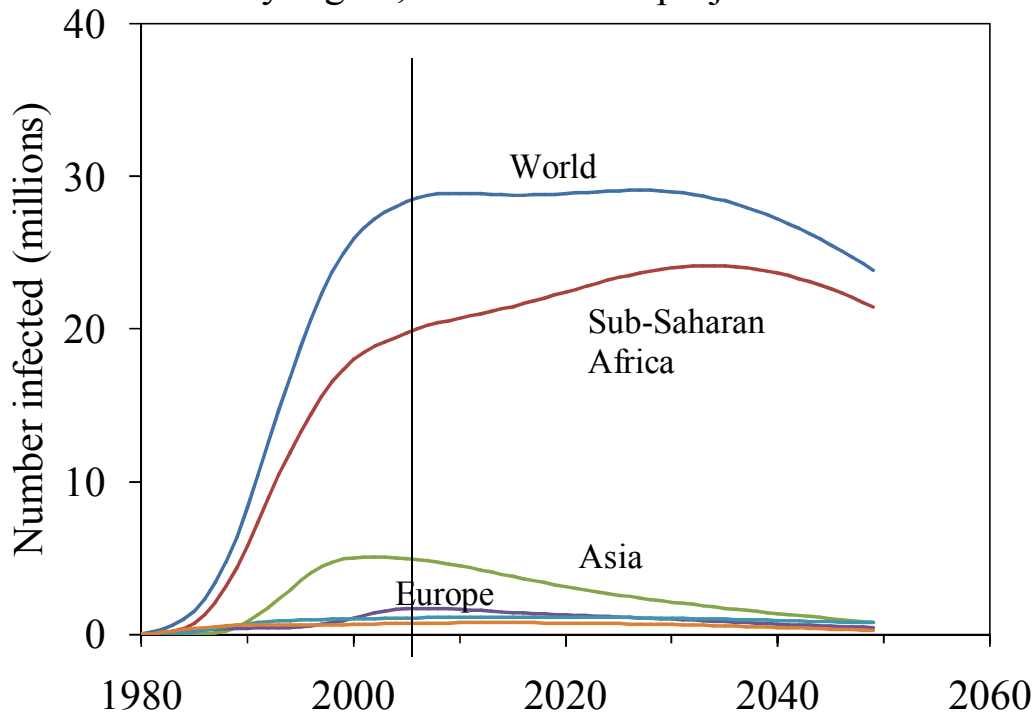
Source: UN 2007

Figure 7: Annual number of new HIV infections among adults by region, estimates and projections



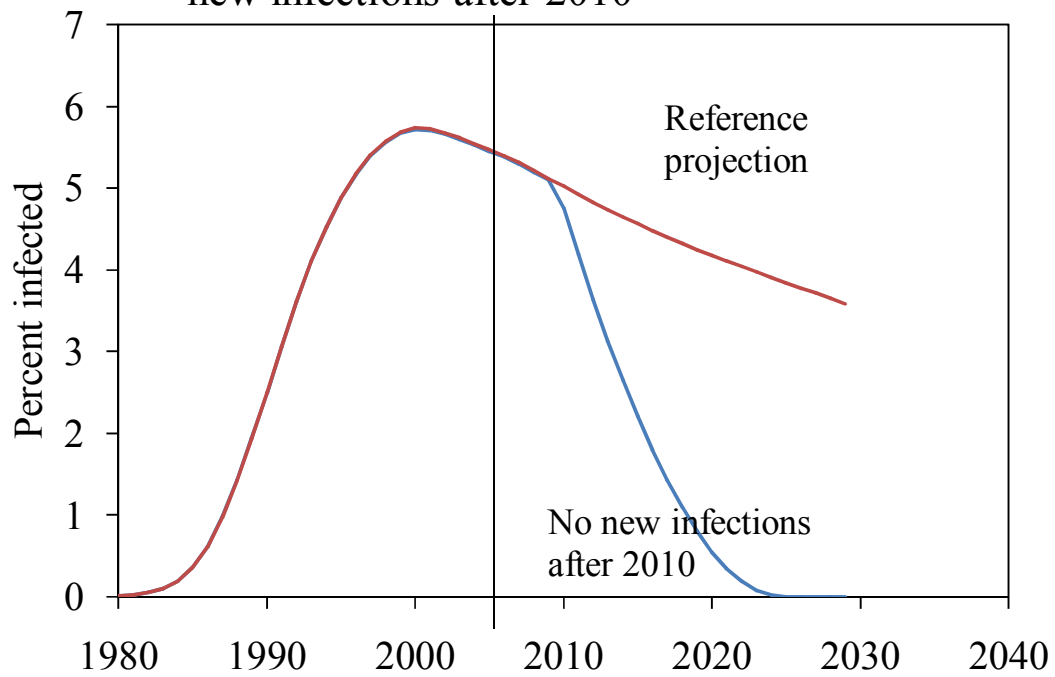
Source: UN 2007

Figure 8: Number of HIV infected adults (15-49) by region, estimates and projections



Source: UN 2007

Figure 9: HIV prevalence rate for sub-Saharan Africa  
reference projection and projection with no  
new infections after 2010



Source: UN 2007

Figure 10 A: Crude death rate with and without AIDS in sub-Saharan Africa

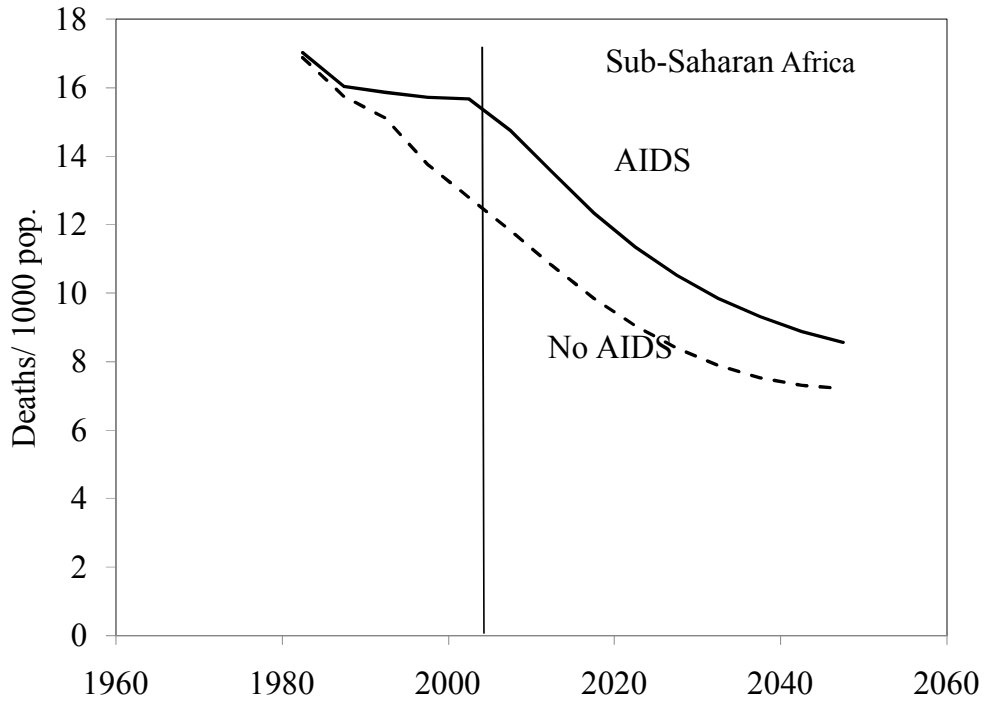


Figure 10 B: Crude death rate with and without AIDS in Europe, N.America, Asia and L.America

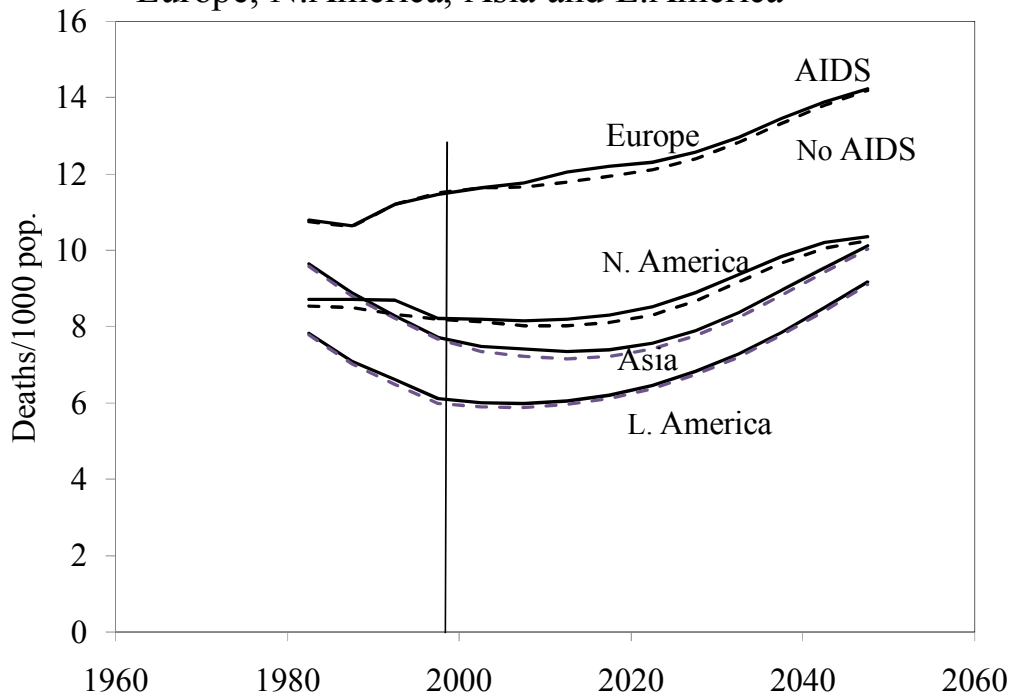




Figure 11. Population by sex and age group, "with AIDS" and "without AIDS", Sub-Saharan Africa, 2015

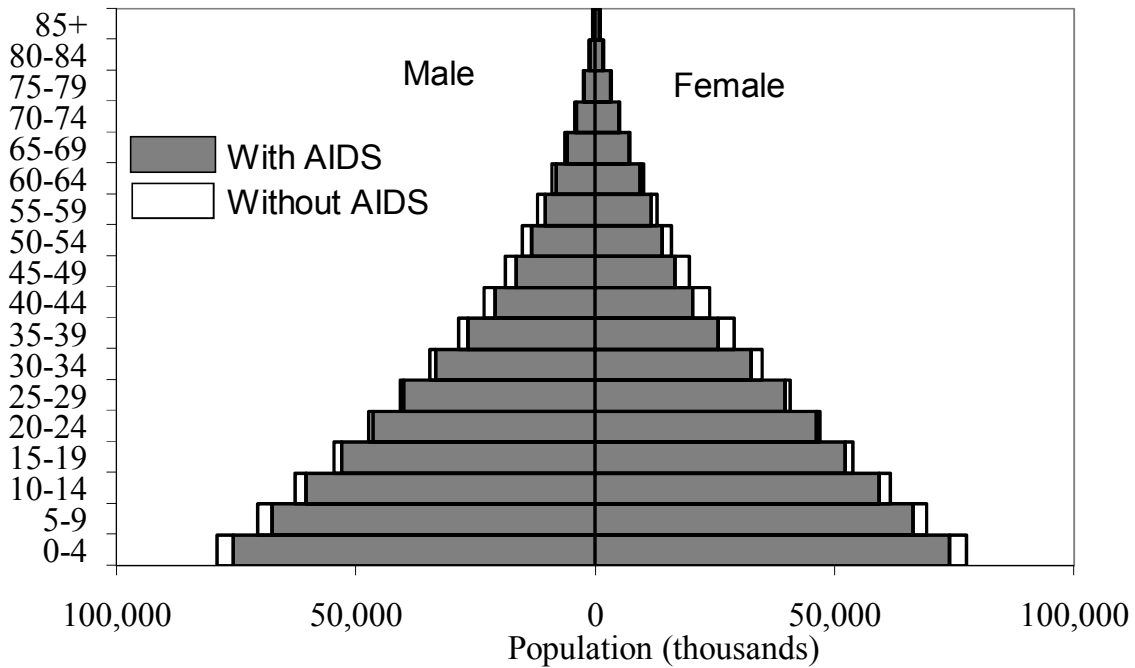


Figure 12. Population by sex and age group, "with AIDS" and "without AIDS", South Africa, 2015

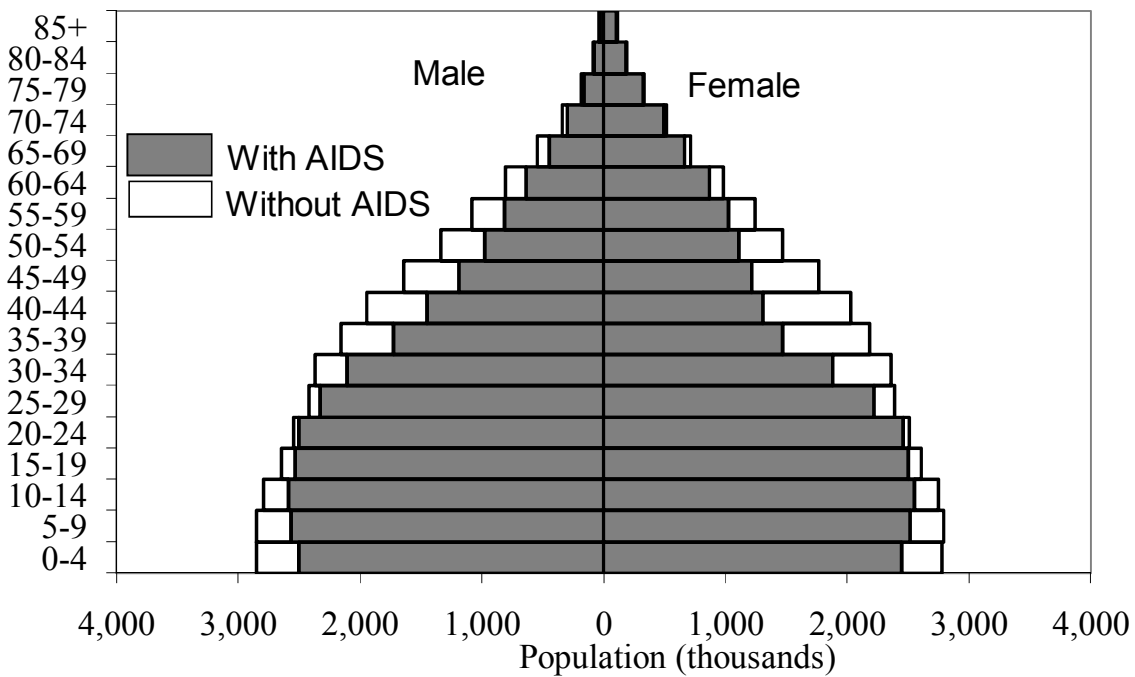


Figure 13: Percent change in population size between 2005 and 2050 with and without AIDS epidemic, by region

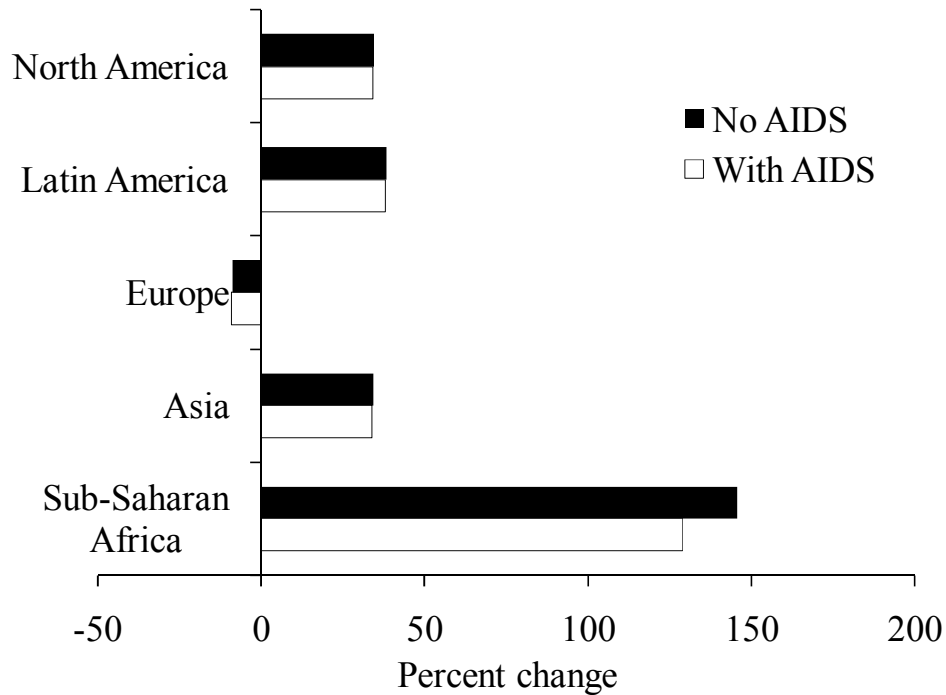


Figure 14: Populations size of sub-Saharan Africa, with and without the AIDS epidemic, estimates and projections

