The Origin of HIV AIDS

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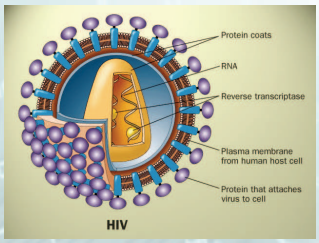
Human Immunodeficiency Virus



- HIV is a lentivirus, which is a class of viruses that attack the immune system.
- Lentiviruses are in turn part of a larger group of viruses known as retroviruses.
- The name 'lentivirus' literally means 'slow virus' because they take such a long time to produce any adverse effects in the body.
- These viruses have been found many different animals including cats, sheep, cattle and horses A retrovirus is a virus which has a genome consisting of two RNA molecules, which may or may not be identical.
- It relies on the enzyme reverse transcriptase to perform the reverse transcription of its genome from RNA into DNA, which can then be integrated in the host's genome with an integrase enzyme.

Simian Immunodeficiency Virus

- It is now generally accepted that HIV is a descendant of a Simian Immunodeficiency Virus because certain strains of SIVs bear a very close resemblance to HIV-1 and HIV-2, the two types of HIV.
- HIV-2 for example corresponds to SIVsm, a strain of the Simian Immunodeficiency Virus found in the sooty mangabey (also known as the green monkey), which is indigenous to western Africa.
- The more virulent strain of HIV, namely HIV-1, was until recently more difficult to place. Until 1999, the closest counterpart that had been identified was SIVcpz, the SIV found in chimpanzees. However, this virus still had certain significant differences from HIV.

Simian Immunodeficiency Virus: February 1999

- Researchers at the University of Alabama announced that they had found a type of SIVcpz that was almost identical to HIV-1.
- This particular strain was isolated from a sample taken from a sub-group of chimpanzees known as Pan troglodytes, which were once common in west-central Africa.

- Researchers concluded that wild chimps had been infected simultaneously with two different simian immunodeficiency viruses which had "viral sex" to form a third virus that could be passed on to other chimps and was capable of infecting humans with HIV AIDS.
- These two different viruses were traced back to a SIV that infected red-capped mangabeys and one found in greater spot-nosed monkeys.
- They believe that the hybridization took place inside chimps and that humans had become infected with both strains of SIV after they hunted and killed the two smaller species of monkey.

Zoonosis

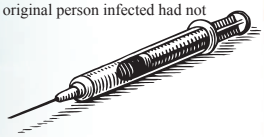
- is any infectious disease that may be transmitted from other animals, both wild and domestic, to humans.
- It has been known for a long time that certain viruses can pass between species. Because chimpanzees obtained SIV from two other species of apes shows just how easily this crossover can occur.
- As animals ourselves, we are just as susceptible.

The "Hunter" Theory

- The most commonly accepted theory
- SIVcpz was transferred to humans as a result of chimps being killed and eaten, or their blood getting into cuts or wounds on a hunter.
- Normally a hunter's body could have fought off the SIV, but in a few cases it adapted to its new human host and evolved into HIV.
- Early on, there were several different strains of HIV, each having a slightly different genetic makeup. The most common being HIV-1 group M
- Each time it was passed from a chimp to a human it adapted differently therefore creating a different genetic makeup and becoming a new strain.
- In a study of 1099 individuals in Cameroon it was discovered that 10 (1%) were infected with SFV (Simian Foamy Virus), which, like SIV, was previously thought only to infect chimps.
- These infections were all thought to have come from butchering and eating of monkey and ape meat.

The Contaminated Needle Theory

- This is an extension of the Hunter Theory
- In the 1950s the use of disposable, plastic needles became common around the world as a cheap and sterile way to administer medicine.
- However, in Africa the enormous amount of needles needed to give inoculations and other medication would have been very costly.
- It is likely that one syringe would have been used to give multiple people injections without sterilizing the needle.
- This would rapidly have transferred any viral particles from one person to another, creating huge potential for the virus to mutate and replicate in each new individual it entered, even if the SIV within the original person infected had not yet converted to HIV.



Oral Polio Theory

- HIV-1 evolved from accidental vaccine contaminations and subsequent transmissions to African villagers.
- In his book, *The River*, the journalist Edward Hooper suggested that HIV could be traced to the testing of an oral polio vaccine called Chat, given to about a million people in the Belgian Congo , Rwanda and Burundi in the late 1950s.
- Hooper's belief is that Chat was grown in kidney cells taken from local chimps infected with SIVcmz.
- *Polio Vaccine being grown* →

Oral Polio Theory

- **The vaccine was analyzed and in April 2001 it was announced that no trace had been found of either HIV or chimpanzee SIV.**
- **A second analysis confirmed that only macaque monkey kidney cells, which cannot be infected with SIV or HIV, were used to make Chat.**
- **Most have taken this evidence to mean that the OPV vaccine theory is not possible.**

The "Heart of Darkness" Theory

- This is a more recent theory based on the basic 'hunter' premise, but more thoroughly explains how this original infection could have lead to an epidemic.
- It was first proposed in 2000 by Jim Moore, an American specialist in primate behavior,.
- During the late 19th and early 20th century, much of Africa was ruled by colonial forces.
- During this time many Africans were forced into labor camps where sanitation was poor, food was scare and physical demands were extreme.

The "Heart of Darkness" Theory

- Moore proposed that these factors alone would have been sufficient to create poor health in anyone, so SIV could easily have infiltrated the labor force and taken advantage of their weakened immune systems to become HIV.
- The most likely possibility he says was a stray and perhaps sick chimpanzee with SIV that would have made a welcome extra source of food for the workers.
- Moore also believes that many of the laborers would have been inoculated with un-sterile needles against diseases such as smallpox and that many of the camps actively employed prostitutes to keep the workers happy, creating numerous possibilities for onward transmission.

What About HIV-2

- Until recently, the origins of the HIV-2 virus had remained relatively unexplored.
- HIV-2 is thought to come from the SIV in Sooty Mangabeys rather than chimpanzees, but the crossover to humans is believed to have happened in a similar way (i.e. through the butchering and consumption of monkey meat).
- It is far rarer, significantly less infectious and progresses more slowly to AIDS than HIV-1. As a result, it infects far fewer people, and is mainly confined to a few countries in West Africa.

The Conspiracy Theory

- Some people believe HIV is a “conspiracy” theory or that it is man-made.
- In a recent US survey showed that a significant number of African Americans believe that HIV was invented as part of a biological warfare program designed to wipe out large numbers of African Americans and Homosexuals.
- They believe this was done under the US Federal “special cancer virus program” (SCVP), possibly with the help of the CIA.
- Some even believe it was spread (either deliberately or inadvertently) to people all over the world through the smallpox inoculation program, or to gay men through Hepatitis B vaccine trials.
- Although none of these theories can be definitively disproved, they clearly ignore the link between SIV and HIV and the fact that the virus has been seen in humans as far back as 1959.
- There was also a lack of genetic-engineering technology available when AIDS first appeared in Humans.

Did HIV Come from Africa?

- Given the evidence of all the different studies, it is likely that Africa was indeed the continent where the transfer of HIV to humans first occurred
- Monkeys from Asia and South America have never been found to have SIVs that could have caused HIV in humans.
- However, who exactly spread the virus from Africa , to America and beyond remains a mystery. It is quite possible that separate strains of the virus could have been developing in a number of different countries years before the first cases were ever officially identified, making it virtually impossible to trace one single source.

Some of the Earliest Instances of HIV in Humans

- A plasma sample taken in 1959 from an adult male living in what is now the Democratic Republic of Congo
  - This suggests that HIV-1 was first introduced into humans around 1940s or the 1950s, which is much earlier than previously though
- HIV found in tissue samples of an American teenager who died in St. Louis in 1969
- HIV found in tissue samples from a Norwegian soldier who died around 1976
- In January 2000 a studied conducted by Bette Korber of the Los Alamos National Laboratory suggested that the first case of HIV-1 occurred around 1930 in West Africa.
- This finding was based on a computer model of HIV’s evolution.

How Did the Epidemic of AIDS Spread So Suddenly?

- **Blood Transfusions**:
  - As they became more common in medical practice the demand for blood became very high.
  - At one point in the US donors were paid for blood donations which attracted those most desperate for cash and blood donations were not scanned for HIV because doctors were unaware of how the virus was transmitted. Sometimes these donations were sent overseas as well.

- **Drug Use**:
  - In the 1970s Heroin use increased dramatically following the Vietnam War and Middle Eastern conflicts
  - The development of “shooting galleries”, where drugs and equipment were sold allowed easy access of IV drugs, again with people being unaware of how easily the disease was spread.

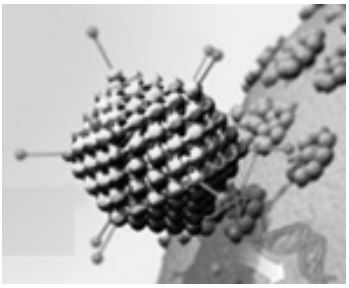
How Did the Epidemic of AIDS Spread So Suddenly?

- **Travel**:
  - Escalated in the late 1970s and early 1980s with many young men making the most of the homosexual revolution.
  - In Africa it possibly spread along truck routes through cities within the country itself.

Works Cited

- http://www.avert.org/origins.htm
- http://www.originofaids.com/
- http://www.thebody.com/whatis/origins.html

Figure 1. Drawing of a virus attached to a cell. The capsid with spikes is visible and the genetic material is located inside the capsid. <http://ghr.nlm.nih.gov/handbook/illustrations/therapyvector>



The outermost layer of all viruses (whether a capsid or an envelope) has viral attachment glycoproteins (sometimes called envelope proteins). These viral molecules are mainly protein with a small piece of carbohydrate attached. Viral glycoproteins match up with receptor proteins on the outside of host cells. In fact, it is this specific match between host cell receptors and viral attachment glycoproteins that determines which cells are hosts of a particular virus. Typically, viruses can only infect specific types of cells in one species of host, although some viruses, like influenza viruses, can infect more than one host species. Every kingdom of organisms has viruses that attack its member species.

Viruses must enter a living host cell in order to replicate because they do not have the necessary enzymes and structures to replicate on their own. Once in the host cell they control the cell's metabolism, energy, and resources, while using cellular structures to read viral genes. These viral genes code for viral nucleic acids and proteins that will be used to assemble new viral particles within the host cell. Once replication of new virus particles is complete, they leave the host cell. Some viruses leave by budding off the plasma membrane, while others burst from the cell, thereby destroying the host.

Questions for Investigation 1:

1. Compare the picture of the virus above to a picture of a cell in your textbook or on the web. Which structures are the same? Which are not found in the virus?
2. How does the virus make up for the parts it doesn't have?

Resources for this investigation:

If you are not familiar with DNA and the usual way that the genes of a cell are transcribed and translated, you should review the basics of molecular biology in a general biology text or at a reputable website, or you might view a video online, such as Transcription and Translation at <http://www.youtube.com/watch?v=41_Ne5mS2ls> or interact with a simulation such as the one at <http://learn.genetics.utah.edu/content/begin/dna/transcribe/>

Investigation 2: HIV biology

The HIV is a member of the retrovirus family. Retroviruses contain RNA, reverse transcriptase enzyme, and an envelope with glycoprotein spikes. In the illustration of HIV in Figure 2, the envelope is labeled as lipid membrane and the protein coat is labeled as capsid. Those labeled gp120 are the viral attachment glycoproteins of HIV. HIV contains two identical pieces of RNA within the capsid.

After a retrovirus enters the host cell, the virus's own reverse transcriptase uses the viral RNA as a template to make a double-stranded DNA molecule. The flow of genetic information goes from RNA to DNA, which is the opposite of what happens in a normal cell, and the name of the enzyme reflects this reversed flow.

The double-stranded viral DNA is transported into the cell's nucleus where it is

inserted, or integrated, into the cell's DNA. This viral DNA is now called a provirus. Often, it may remain in the host cell DNA for many years before becoming active. When the host cell synthesizes a new copy of its own DNA, the provirus is copied right along with it. This type of replication, where the viral DNA is dormant for an extended period within the host nucleus is a feature of only some kinds of viruses. Others do not interact at all with the host's DNA.

Reverse transcriptase does not have the "proofreading" capabilities of the enzymes used in normal cell replication. Thus a high rate of transcription errors occur during the process of copying viral RNA to DNA. These errors cause mutations in the new viral RNA that is produced during viral replication, which is why the HIV virus can mutate so quickly.

The host cells of HIV are cells of the human immune system: macrophages and T helper cells. Macrophages are non-specific initiators of the immune response and may engulf HIV as it arrives. Helper T cells have a particular receptor protein called CD4, which also can bind HIV. Thereby, the gp120 glycoprotein on the HIV surface attaches to the CD4 receptor protein enabling the HIV to infect the cell.

A diagram of the HIV replication cycle is show in Fig. 2. View the animation of the HIV replication cycle and explore other resources for this investigation to understand the basic steps.

Figure 2. Structure of an HIV virus. <http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/Biology/Pages/structure.aspx>

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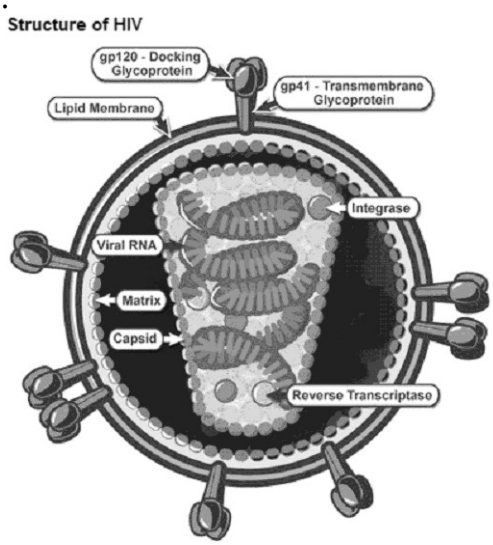
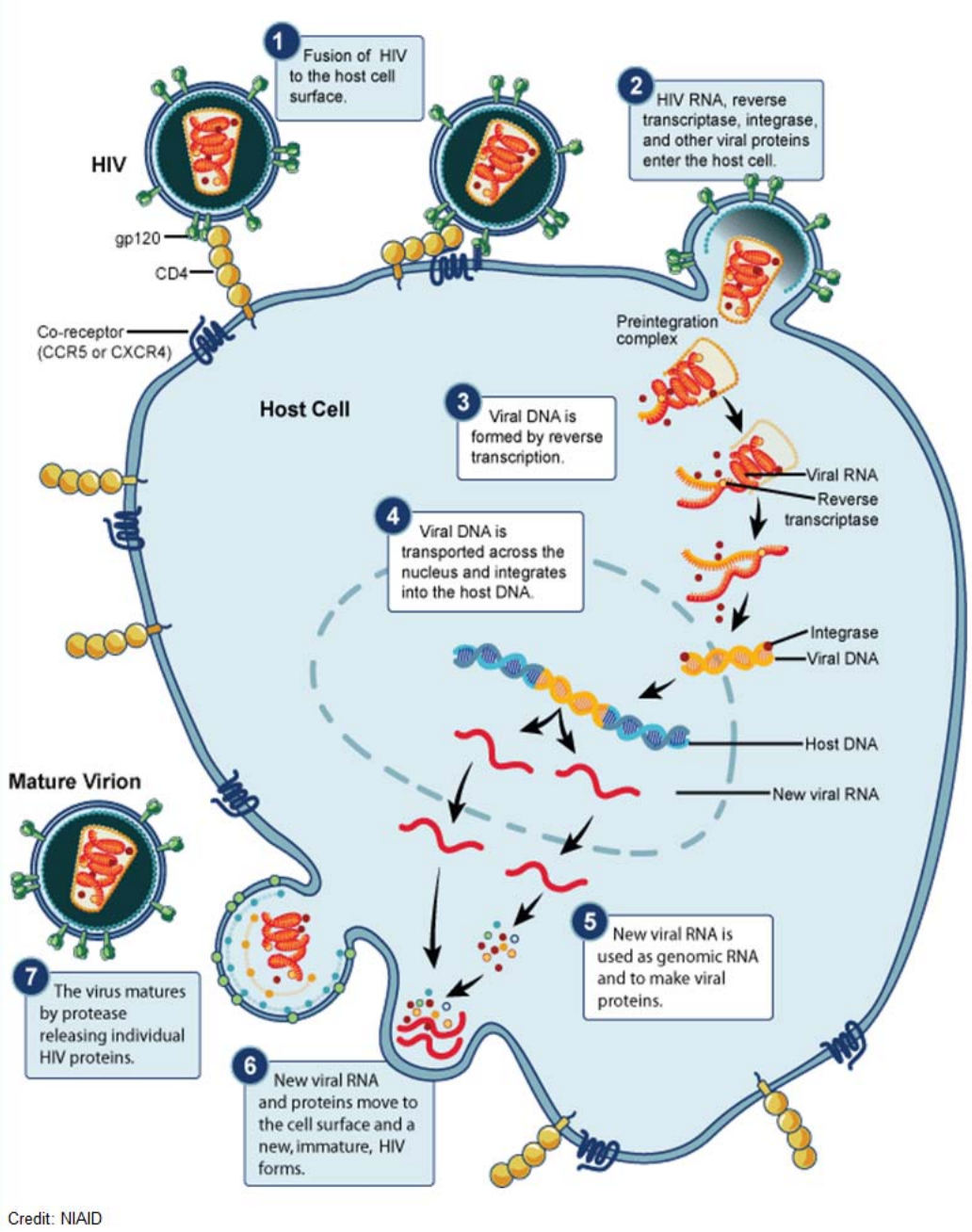


Figure 3. Diagram of HIV replication cycle
<http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/Biology/Pages/hivReplicationCycle.aspx>



Question for Investigation 2: The replication cycle of HIV

1. HIV works by hijacking the cell's normal ability to make new proteins. Look at the descriptions of major steps in the HIV

replication cycle and identify the organelles and cell structures that are involved in HIV replication. For each of the HIV replication below, identify the organelles in the host cell that are involved and match them with the replication processes.

a. **Binding and Fusion:** HIV begins its life cycle when the GP120 glycoprotein on the HIV envelope binds to a **CD4 receptor** and one of two **co-receptors, CCR5 or CXCR4,** on the surface of a $CD4^+$ **T- lymphocyte**. The virus then fuses with the host cell. After fusion, the virus releases RNA, its genetic material, into the host cell.

b. **Reverse Transcription:** An HIV enzyme called reverse transcriptase converts the single-stranded HIV RNA to double-stranded HIV DNA.

c. **Integration:** The newly formed HIV DNA enters the host cell's nucleus, where an HIV enzyme called integrase "hides" the HIV DNA within the host cell's own DNA. The integrated HIV DNA is called a provirus. The provirus may remain inactive for several years, producing few or no new copies of HIV.

d. **Transcription:** When the host cell receives a signal to become active, the provirus uses a host enzyme called RNA polymerase to create copies of the HIV genomic material, as well as shorter strands of RNA called messenger RNA (mRNA). The mRNA is used as a blueprint to make long chains of HIV proteins.

e. **Assembly:** An HIV enzyme called protease cuts the long chains of HIV proteins into smaller individual proteins. As the smaller HIV proteins come together with copies of HIV's RNA genetic material, a new virus particle is assembled.

f. **Budding:** The newly assembled virus pushes out ("buds") from the host cell. During budding, the new virus is encapsulated in part of the cell's outer envelope. This envelope, which acts as a covering, is studded with protein/sugar combinations called HIV glycoproteins. These HIV glycoproteins allow the virus to bind CD4 and co-receptors. The new copies of HIV can now move on to infect other cells.

Resources for this investigation:

3D Animation of HIV replication. Accessed May 2011, from: <http://www.youtube.com/watch?v=RO8MP3wMvqg>

Description of the HIV replication cycle. Accessed May 2011, from: <http://www. avert.org/virus.htm>

Investigation 3: HIV Infection and the Body's Response

HIV's main host cells are macrophages and the helper T cells (T_H) of the human immune system. T_H cells are also known as T4 cells, or as CD4 cells, because of the CD4 receptors on their membranes. T_H cells secrete chemical messengers that control the action of other classes of immune system cells. The amount of T_H cells is measured by the "CD4 count," one indicator of the health of the immune system and progress of the disease. A normal CD4 count is between 800 and 1000 cells /mm^3.

During the initial infection the HIV replicates quickly and releases millions of new viral particles in the first few weeks (Figure 4). The population of viral particles in the blood may reach 10,000,000/ml. At this time infected persons may not know

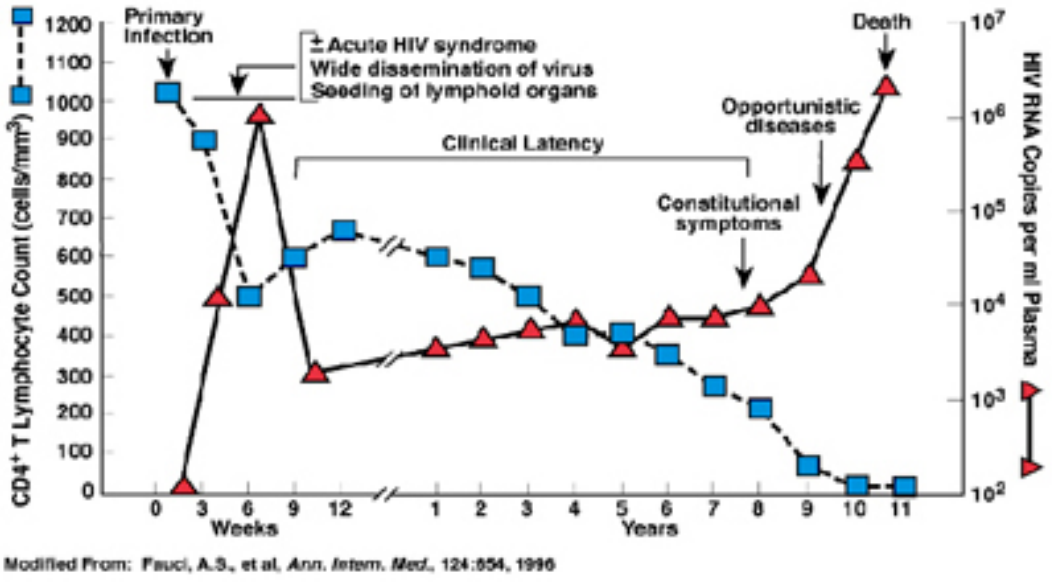
they are infected, but now is when they are most likely to spread the disease to others because of the high concentration of HIV in the blood. The population of T_H cells declines rapidly as they are infected and destroyed.

After the initial T_H cell decline, the body starts making antibodies against HIV, and these reduce the number of HIV particles in the blood. The T_H population recovers and continues to produce an immune response that keeps viral levels low and steady. By this time, some infected T_H cells have viral DNA integrated in their genome. These HIV genes remain latent until activated at a later time and produce virus.

Over time the host's immune response fails due to infection of the helper T cells and viral levels rise again. The patient begins to experience opportunistic infections such as *Candida albicans* yeast infections of the mouth or vagina, persistent diarrhea, fever, weight loss and reactivation of previous infections such as shingles and tuberculosis. Without treatment, as many as 100 billion HIV particles are produced each day in the lymph tissue and attacked by macrophages and antibodies. As many as 2 billion T cells are produced each day in response to the growing viral load. As HIV out competes the T cells, the host can no longer produce enough T cells to keep the virus under control. In the U.S., clinical AIDS is diagnosed when a person's CD4 count falls below 200/mm^3.

The time for progression from the initial infection with HIV to clinical AIDS varies widely, from months to years. Treatment with antiviral drugs (discussed in Investigation 4), can dramatically change the course of the infection by reducing the ability of the virus to replicate and thereby allowing the health of the immune system in HIV positive people to recover.

Figure 4. Typical course of HIV infection that shows the relationship between the levels of HIV (viral load) and CD4+ T cell counts over the average course of untreated HIV infection.
<http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/Biology/pages/clinicalcourse.aspx>



Chapter One

AIDS in Kenya: A status report

Key messages

A GENERALIZED EPIDEMIC

With HIV having spread throughout the Kenyan public at large, relatively low levels of risk behaviour may carry considerable risks of HIV transmission.

REDUCED HIV PREVALENCE

An estimated 6.2% of adults between ages 15 and 49 were living with HIV as of December 2011. The percentage of Kenyans living with HIV has fallen by roughly 40% since 1995–1996, although the number of people living with HIV is increasing due to population increases and a decline in AIDS deaths. Altogether, 1.6 million Kenyans were living with HIV in 2011, with women representing 59% of all people living with HIV.

DECLINING, YET STILL SUBSTANTIAL, HIV INCIDENCE

The annual number of new HIV infections is roughly one-third the number in 1993, when Kenya's epidemic peaked. However, the number of new infections remains unacceptably high, with an estimated 104,137 Kenyans becoming infected in 2011.

A SEXUALLY DRIVEN EPIDEMIC

Sexual transmission accounts for an estimated 93% of new HIV infections in Kenya, with heterosexual intercourse representing 77% of incident infections. Adults in stable, seemingly low-risk heterosexual relationships make up the largest share of new HIV infections.

KEY POPULATIONS

Several key populations – namely, sex workers and their clients, men who have sex with men, and people who inject drugs – account for roughly one in three new HIV infections, a far larger share than previously understood.

SUB-NATIONAL VARIATION

The epidemic varies widely between and within provinces, with a 15-fold difference in HIV prevalence between the most heavily affected province (Nyanza) and the least affected (North Eastern).

YOUNG PEOPLE AND HIV

With 43% of the national population under the age of 15, the future of Kenya's epidemic will be determined in large measure by the success of efforts to slow the spread of HIV among young people. Prevention programmes should aim to build on statistically significant declines in HIV prevalence that have occurred over the last decade among both young men and women.

AN OVERRIDING NATIONAL PRIORITY

AIDS remains one of the central impediments to national health, development and well-being. AIDS has lowered life expectancy, deepened poverty in Kenya, reduced economic growth, exacerbated hunger, and worsened basic health indicators. In 2011, at least 1.1 million children in Kenya had lost one or both parents to AIDS.

AN EVOLVING EPIDEMIC

Kenya's epidemic continues to evolve, underscoring the need for continued vigilance and evidence-based action to respond to new challenges and opportunities.

STRENGTHENING AND ACCELERATING THE NATIONAL FIGHT AGAINST AIDS

Kenya has what is known as a "generalized" epidemic, with the virus having spread beyond discrete groups to affect the whole of society.

With a significant proportion of the national population already infected, the risks of encountering HIV during any single episode of risk behaviour are considerable, meaning that relatively low levels of risk behaviour may nevertheless carry a substantial likelihood of transmission. Among adult participants in the 2003 Kenya Demographic and Health Survey who said they had "no risk" for HIV, nearly 1 in 20 (4.6%) were in reality HIV-infected (Montana et al., 2007).

Since HIV was first recognized in Kenya in 1984, the universe of knowledge about the epidemic has continually expanded, providing national decision-makers with an ever-growing foundation for evidence-informed strategies. Although major progress has been achieved in Kenya's response to HIV, the epidemic remains one of the country's greatest health and development challenges. Moreover, the epidemic continues to evolve, presenting both new challenges and new opportunities as Kenya looks to the future.

Kenya:

The socio-economic context

A young, rapidly growing population

With a population of 38.6 million people (Kenya National Bureau of Statistics, 2010), Kenya has a rate of population growth (2.8% annually in 1990–2008) that exceeds the average for low-income countries overall (2.2%) and for sub-Saharan Africa as a whole (2.6%) (World Bank, 2010). Forty-three per cent of Kenya's population is below age 15 (World Bank, 2010). The large majority (78%) of Kenyans currently live in rural areas (World Bank, 2010), with 60% of Kenyan households engaged in farm work (Kenya Institute for Public Policy Research and Analysis, 2009).

Economic conditions

After experiencing modest economic growth in the 1990s (2.2%) annually, Kenya improved its economic performance during the last decade, with an average annual increase in the gross domestic product of 4.5% (World Bank, 2010; see Kenya Institute for Public Policy Research, 2009). The social and political crisis that followed the December 2007 election interrupted a five-year period of growth, although the country's economic performance has subsequently rebounded (Kenya Institute for Public Policy Research, 2009). Achieving the goals set forth in Vision 2030 will require annual increases of gross domestic product of 10% (Kenya Institute for Public Policy Research and Analysis, 2009) – a pace far in excess of Kenya's current and historic rates of growth.

A substantial portion of Kenya's population struggles to obtain the basic necessities of life. Nearly half (46.6%) of all Kenyans were living below the national poverty line in 2005–2006, and 40% of the population subsists on less than US$ 2 a day (World Bank, 2010). The country ranks 177th in per capita gross national income (World Bank, 2010).

Kenya is also among the world's most economically inequitable societies. Between 1995 and 2008, the poorest quintile (20%) accounted for only 4.7% of national wealth (World Bank, 2010). By contrast, the richest quintile claimed 53% of national income (World Bank, 2010).

The myriad ways in which Kenyans live and relate to each other are intrinsically linked with the epidemic's past, present and future.

Education and health

Kenya's literacy rates – 90% for males and 83% for females – are considerably higher than for sub-Saharan Africa generally (76% and 63%, respectively) (World Bank, 2010). Primary school completion rates are high (80%), but significantly fewer young people (49% in 2008) attend secondary school (World Bank, 2010). While Kenya outranks most African countries on basic education indicators, experts say that the country's rapidly growing labour force is generally lacking in the skills that will be required for the country to become globally competitive (Kenya Institute for Public Policy Research and Analysis, 2009).

Kenya's health profile is mixed. Largely because of the heavy impact of HIV, life expectancy in Kenya has fallen sharply (Gelmond et al., 2009), although it has begun to rebound in recent years, as HIV-related mortality has declined. The population-based mortality rate for children under age five was higher in 2008 (128 per 1,000 live births) than it was in 1990 (105 per 1,000) (Kenya National Bureau of Statistics, 2010), but the under-five mortality rate has fallen over the last 10 years (Kenya National Bureau of Statistics, 2010). By contrast, maternal mortality in Kenya (560 per 100,000 live births in 2008) is considerably lower than the average for low-income countries (790 per 100,000) or for sub-Saharan Africa as a whole (900 per 100,000) (World Bank, 2010). Pregnant women in Kenya are significantly more likely to receive antenatal care than sub-Saharan African women as a whole (World Bank, 2010), with 92% of Kenyan women receiving an antenatal care from a medical professional (Kenya National Bureau of Statistics, 2010). Kenya also outperforms sub-Saharan Africa overall with respect to access to sanitation, child immunization, and tuberculosis treatment success (World Bank, 2010).

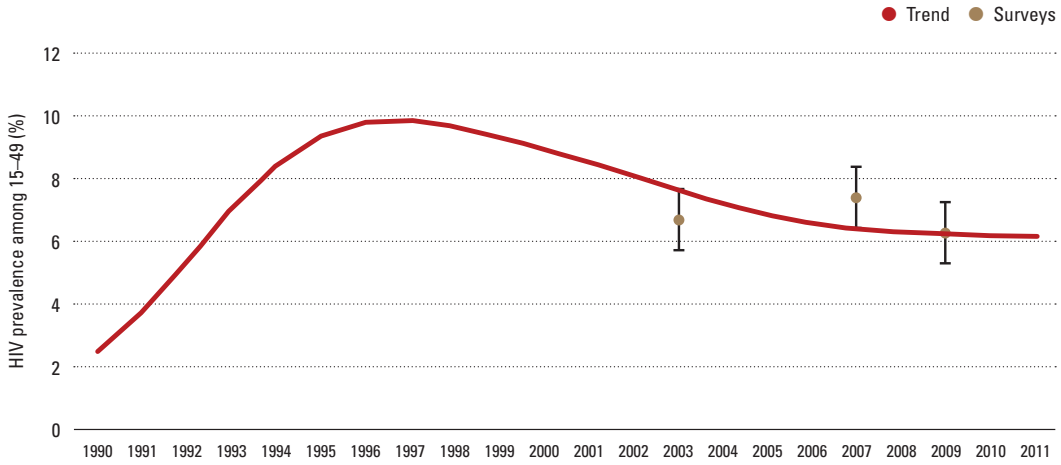
HIV prevalence in Kenya

In 2011, Kenya estimates that approximately 6.2% of the adult population is HIV-infected.[1] HIV prevalence in Kenya is believed to have peaked in 1995–1996, at 10.5%, subsequently falling by approximately 40% and remaining relatively stable for the last several years.

Historically a key marker for national progress in the AIDS response, HIV prevalence becomes more difficult to interpret as antiretroviral treatment is scaled up. Because treatment extends life and reduces rates of AIDS deaths, increases in HIV prevalence are likely even with incremental declines in the rates of new infections. Accordingly, performance indicators for Kenya's most recent national AIDS strategy project a relatively modest decline in HIV prevalence between 2007 and 2013, with an actual uptick on overall HIV prevalence anticipated over time due to the health benefits of improved treatment access.

Figure

Trends in national HIV prevalence in Kenya



An estimated 1.6 million Kenyans were living with HIV in 2011. This equals the peak number of HIV-infected people that had previously been maintained annually between 1996 and 2002, and it represents a nearly four-fold increase over the 400,000 people estimated to be living with HIV in Kenya in 1990. Kenya has the third largest population of people living with HIV in sub-Saharan Africa and the highest national HIV prevalence of any country outside Southern Africa (UNAIDS, 2008). As people living with HIV are living longer as a result of improved access to HIV treatment, it is anticipated that the total number of HIV-infected individuals in Kenya will continue to increase, approaching 1.8 million by 2015.

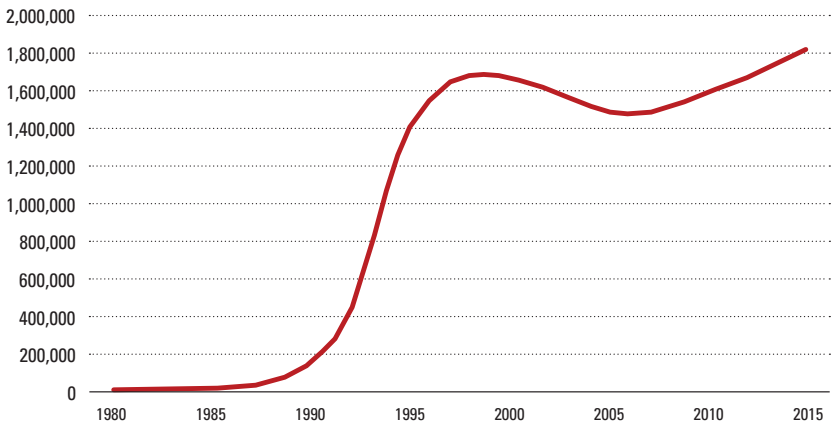
There is considerable geographic variability in the burden of HIV in Kenya. Provincial HIV prevalence ranges from a high of 13.9% in Nyanza Province to a low of 0.9% in North Eastern Province – a more than 15-fold variation (Kenya National Bureau of Statistics, 2010). Nyanza Province alone accounts for one in four HIV-infected people in Kenya.

Kenya's epidemic disproportionately affects women, who account for 59.1% of adults living with HIV. Among people between 15 and 49 years, HIV prevalence among women (8.0%) is nearly twice that among men (4.3%) (Kenya National Bureau of Statistics, 2010).

The odds of being infected increase as individuals transition from adolescence to adulthood. Although HIV is most likely to affect young adults, a considerable number of older people are living with HIV. In 2008–2009, roughly one out of 11 (9.1%) Kenyan men ages 50–54 were HIV-positive (Kenya National Bureau of Statistics, 2010).

For Kenyans as a whole, urban residents have historically more likely to be HIV-infected than rural dwellers (Kenya National Bureau of Statistics, 2010). However, there is a notable distinction between men and women in this

1 National surveys over the last several years have yielded somewhat different estimates of adult HIV prevalence in Kenya: 6.7% in 2003 (Central Bureau of Statistics, 2004), 7.4% in 2007 (NASCOP et al., 2009), and 6.3% in 2008-2009 (Kenya National Bureau of Statistics, 2010). Kenya's latest estimate for 2010 is derived from modelling based on the most recent available epidemiological evidence.



Figure

Number of children and adults living with HIV in Kenya

regard, with men in rural areas more likely to be HIV-infected than their urban counterparts (4.5% vs. 3.7%) (Kenya National Bureau of Statistics, 2010). Over time, HIV prevalence in urban and rural settings have converged, with HIV prevalence in urban areas only modestly higher than prevalence in rural settings.

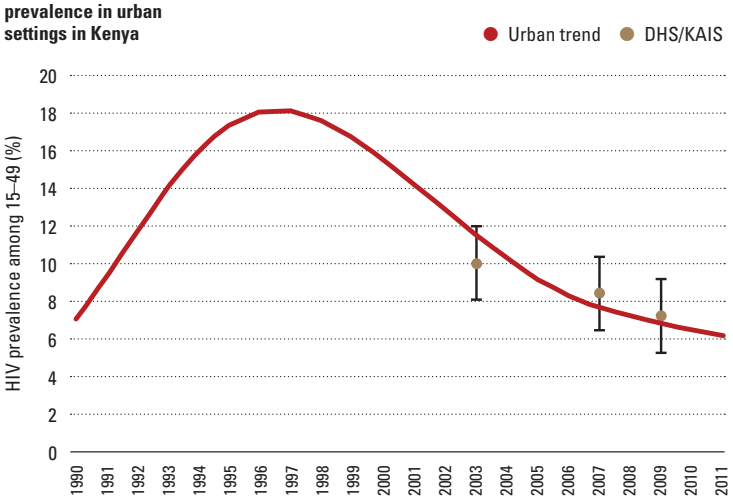
HIV affects Kenyans from all socioeconomic strata. Highest HIV prevalence (7.2%) is among the top wealth quintile, with the second highest HIV prevalence among the second lowest (6.8%). The poorest Kenyans (lowest wealth quintile) are least likely to be living with HIV, with a prevalence of 4.6%.

For sub-Saharan Africa generally, educational attainment is inversely correlated with HIV risk for women, at least according to surveys conducted over the last 10–15 years (Hargreaves et al., 2008). In Kenya, this pattern is not so clearly established. Although women with secondary education or higher have lower HIV prevalence (6.9%) than women who completed only primary education (8.9%), lowest HIV prevalence is reported among women with no education (5.8%) (Kenya National Bureau of Statistics, 2010).

Muslim Kenyans have HIV prevalence roughly half the national average (3.3%), compared with 5.9% of Roman Catholics and 6.6% of people of Protestant or another Christian denomination (Kenya National Bureau of Statistics, 2010). Among Kenyan tribes, the Luo are notably more likely to be living with HIV than other ethnicities, with more than one in five Luo (20.2%) testing HIV-positive in the 2008–2009 national

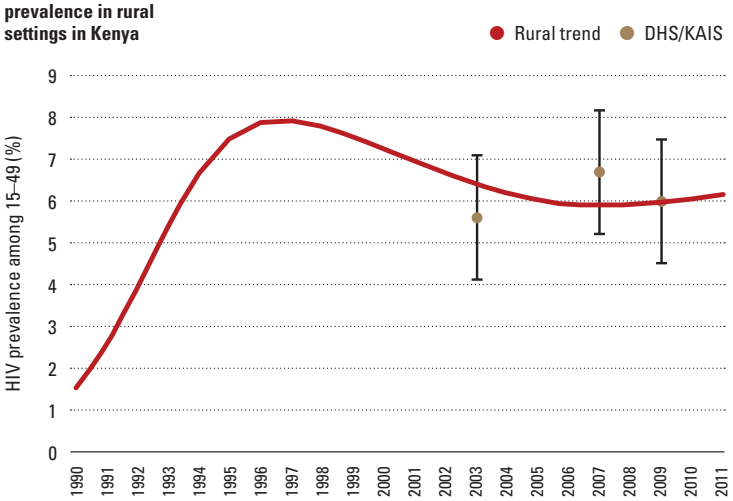
Figure

Estimated HIV prevalence in urban settings in Kenya



Figure

Estimated HIV prevalence in rural settings in Kenya



household survey (Kenya National Bureau of Statistics, 2010). Somalis have the lowest HIV prevalence of any ethnicity (0.8%).

New HIV infections in Kenya

Each year, roughly 0.5% of the Kenyan adult population (or 1 out of every 200) are newly infected. In 2011, more than 91,000 Kenyan adults became infected. The number of new infections in 2011 was less than one-third the annual number of new infections at the epidemic's peak in 1993, when more than 350,000 adults were newly infected. Although the pace of new HIV infections has slowed in Kenya, the number of new infections remains high. Based on current trends, it is projected that the number of new HIV infections will continue its slow, steady decline, with 81,972 new infections among people over age 15 anticipated in 2013.

In addition to the approximately 91,000 new infections among adults, it is estimated that 12,894 children under age 15 became newly infected with HIV in 2011, with the overwhelming majority contracting the virus during pregnancy or delivery or as a result of breastfeeding. With continuing success in expanding access to services to prevent new infections in children, it is estimated that the number of children newly infected in 2011 was 30% lower than in 2010. The number of people 50 years and older who were newly infected in 2011 is unclear, although comparison of the most recent estimates with the modes-of-transmission analysis for the 15–49 age cohort suggests that the annual number of newly infected older adults could range from 5,000 to 15,000.

In comparison to earlier stages of the epidemic, fewer young people in Kenya today are entering adulthood with HIV infection. Kenya is one of 10 high-burden countries in which HIV prevalence among young women (ages 15–24) has declined by significantly more than 25% (UNAIDS, 2010). Studies over time suggest that declines in new infections may be greater among young women than among young men. In such a young and comparatively sexually inexperienced segment of the population, HIV prevalence is regarded as a useful surrogate for HIV incidence. It is not altogether clear whether the pace of decline in the level of HIV infections among young people is sufficient to achieve the country's 2013 HIV prevalence targets for young women (3%) and young men (1%).

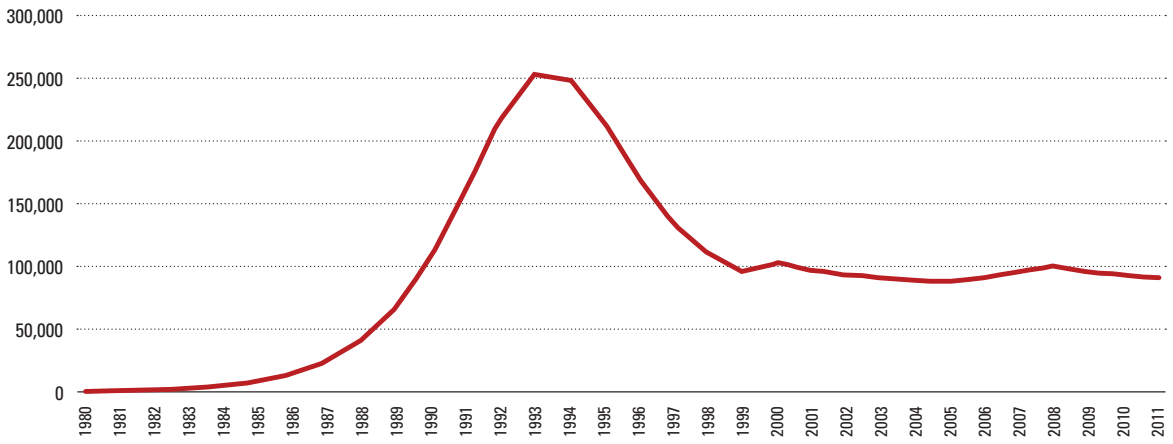
Understanding the rate and distribution of new HIV infections is critical to effective HIV prevention planning. According to Kenya's first-ever study to estimate new infections by modes of transmission, new infections derive from the following sources (Gelmon et al., 2009):

- Heterosexual sex within a union or regular partnership (44.1%)
- Casual heterosexual sex (20.3%)
- Sex workers and clients (14.1%)
- Men who have sex with men and prisons (15.2%)
- Injecting drug use (3.8%)
- Health facility related (2.5%).

Nyanza Province contributes roughly one-third of all new HIV infections in Kenya. The other two provincial centres for new HIV

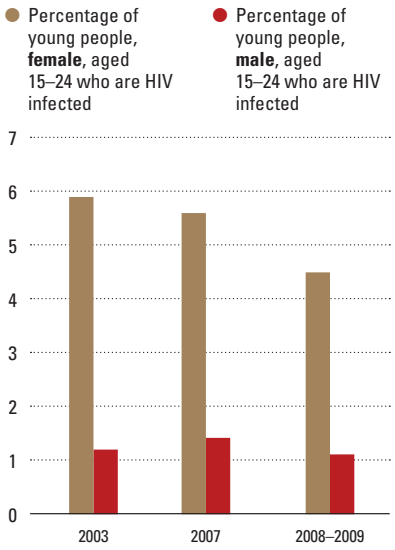
Figure

New HIV infections among adults in Kenya



Figure

HIV prevalence among young people by sex, 2003–2009



infections are Nairobi (10,155 new infections in 2006) and Coast Province (6,656 new infections in 2006) (Gelmon et al., 2009).

Although heterosexual intercourse remains the driving force in Kenya's epidemic, accounting for more than 77% of all new infections, other transmission routes contribute a much larger share of new HIV infections than previously estimated (see Guows et al., 2006). Sex work, sex between men, and injecting drug use together account for nearly one-third of all new infections (Gelmon et al., 2009).

The epidemic continues to exert a disproportionate effect on adolescents and young adults. Young people between ages 15–35 represent 38% of the national population but are believed to make up more than 60% of new HIV infections (NACC, 2009).

AIDS mortality in Kenya

Since the epidemic began, HIV has claimed the lives of at least 1.7 million people in Kenya. In 2011, an estimated 49,126 people in Kenya died of AIDS-related causes.

The AIDS death toll in 2010 represents a nearly two-thirds drop from the peak in AIDS deaths in 2002–2004, when an estimated 130,000 people died each year. Peak mortality followed peak HIV incidence in Kenya by roughly a decade, which one would expect given the roughly 10-year life expectancy of a newly infected individual in the pre-ART era.

Were current trends to continue, Kenya would achieve its 2013 target for reducing the annual number of AIDS deaths to 61,000 or lower. Indeed, current projections indicate that 26,720 Kenyans are likely to die of AIDS-related causes in 2013.

The impact of HIV in Kenya

The epidemic continues to have far-reaching social, economic, health and population effects. In addition to the harms directly inflicted on HIV-infected individuals and the households in which they live, AIDS has had indirect effects that are nevertheless real and substantial on communities and the whole of society.

In particular, HIV infection results in severe economic consequences for affected households (Bates et al., 2004). One out of nine households in Kenya has been affected by AIDS, with the head of household having HIV in more than three out of four AIDS-affected households (NASCOP, 2009).

The epidemic has resulted in a sharp deterioration of basic health indicators. Between 1998 and 2003 – or roughly between the epidemic's peak in Kenya and the early introduction of antiretroviral therapy – the adult mortality rate (ages 15–49) rose by 40% for women and by 30% among men (Gelmond et al., 2009, citing findings from consecutive Demographic and Health Surveys). With a large number of newborns newly infected each year, the epidemic has also increased mortality among children under five (Gelmond et al., 2009).

The concentration of the epidemic's burden among young adults has visited particular hardships on Kenya's children, regardless of whether children themselves become HIV-positive (K'Oyugi, Muita, 2002). In 2011, an estimated 1.1 million children in Kenya had lost one or both parents to AIDS. Kenyan children with one or more HIV-infected

parents are significantly less likely than other children to be in school, more likely to be underweight, and less likely to receive basic medical care (Mishra et al., 2005).

While children have experienced among the harshest effects of the epidemic, AIDS has burdened Kenyans from all age strata and all walks of life. Nearly one in five (18%) Nairobi residents over age 50 report having been personally affected by AIDS, such as becoming infected, caring for an AIDS patients or orphaned child, or losing a loved one (Kyobutungi et al., 2009).

AIDS appears to have affected fertility patterns. On average, HIV-infected women have 40% fewer children than the norm (Akinyi Magadi, Agwanda, 2010). HIV-infected women are notably less likely to express a desire for a child within the next two years than women who had tested HIV-negative or who had not received HIV test results; women living with HIV are also significantly more likely than other women to report not desiring to have a child at any point in the future (NASCOP, 2009).

An ever-evolving epidemic

Change has been a constant feature of Kenya's experience with AIDS, as the epidemic's trajectory has repeatedly defied predictions. The epidemic continues to evolve, and it is certain that AIDS has additional surprises in store for the future.

When the epidemic was first recognized in Kenya in the 1980s, evidence indicated that HIV transmission was primarily concentrated among female sex workers and their clients, especially mobile workers such as long-distance truck drivers. Although these populations remain highly vulnerable to HIV, their relative roles in the spread of HIV have declined over time. The constant evolution of national and sub-national epidemics underscores the importance of continued vigilance, even following extended public health successes.

■ ■ ■ *Available data leave little room for doubt. While HIV prevalence and the rate of new HIV infections are lower now than at earlier points in the epidemic, AIDS will remain a preeminent national priority for decades to come. The generations-long challenge posed by AIDS not only highlights the need for urgent action to address the epidemic, but also for a long-term perspective that emphasizes sustainability, national resolve, and policy and programmatic responses that address the root causes of HIV risk and vulnerability.* ■ ■ ■