'*AIDS in the Twenty-First Century* explains the background to the epidemic, and explores why Africa has been hit so hard and why some countries have been hit harder than others. Reading this book will shock you into an understanding of the long – perhaps century-long – impact we are all going to feel from this disease.' – *New Agriculturist On-line*

'Aimed at a general audience, this is a valuable and well-written addition to the literature on the HIV/AIDS epidemic, commonly dominated by biomedical and epidemiological perspectives.' – John Bongaarts and Geoffrey McNicoll, *Population and Development Review*

'This book needs and deserves to be read. Unlike some writers on AIDS, who appeal emotively to compassion and a sense of injustice, Barnett and Whiteside explicitly address the reader's pragmatism, self-interest and capacity to reason. Somehow, this makes the book's underlying humanity and call to action all the more compelling. If it is read and acted upon, as the authors hope, AIDS would still change history, but perhaps for the better.' – Vicki Luker, *Papua New Guinea Medical Journal*

'Economists Tony Barnett and Alan Whiteside's *AIDS in the Twenty-First Century: Disease and Globalization* provides impressively comprehensive coverage of the social and economic roots and likely impacts of HIV/AIDS.' – Michael J. Selgelid, *Developing World Bioethics*

'Barnett and Whiteside have constructed a monumental book ... It wrestles with complex moral dilemmas such as how we define our responsibilities to others, as well as providing a wealth of informative graphs and tables that nail down the statistics and trends behind HIV/AIDS ... the book is a very valuable resource. Stylistically easy to read, it provides clear, sharp and incisive commentary on the unfolding epidemic. It can be used as a reference book for both novices in the HIV/AIDS field and those who need quick access to a range of studies and statistics.' – Kerry Cullinan, *Transformation: Critical Perspectives on Southern Africa*

# AIDS in the Twenty-First Century

## Disease and Globalization

Second Edition

Tony Barnett

and

Alan Whiteside

palgrave
macmillan



First edition published 2002
This edition published 2006 by
PALGRAVE MACMILLAN
Houndmills, Basingstoke, Hampshire RG21 6XS and
175 Fifth Avenue, New York, N.Y. 10010
Companies and representatives throughout the world

PALGRAVE MACMILLAN is the global academic imprint of the Palgrave Macmillan division of St Martin's Press LLC and of Palgrave Macmillan Ltd.
Macmillan® is a registered trademark in the United States, United Kingdom and other countries. Palgrave is a registered trademark in the European Union and other countries.

ISBN-13 978–1–4039–9768–5 paperback
ISBN-10 1–4039–9768–3 paperback

This book is printed on paper suitable for recycling and made from fully managed and sustained forest sources.

A catalogue record for this book is available from the British Library.

A catalog record for this book is available from the Library of Congress



10 9 8 7 6 5 4 3 2 1
15 14 13 12 11 10 09 08 07 06

Printed and bound in Great Britain by
Antony Rowe Ltd, Chippenham and Eastbourne

# Contents

1. the wave of HIV infection
2. the wave of tuberculosis (some of it multi-drug resistant) which, because it is the most common opportunistic infection, is usually the first visible wave of the epidemic
3. the wave of AIDS illness and death
4. the wave of impact – which includes household poverty, orphaning and many other effects which will be discussed in this book.

Taken together, this long-wave event extends over many decades and probably as long as a century. The global HIV/AIDS epidemic has deep roots in social and economic inequalities. It is a long-wave event: the effects of unusual levels of illness and death will profoundly affect the lives of many individuals and many societies for decades to come.

## Books, papers, websites and other useful sources for further reading

Tony Barnett et al., *HIV/AIDS in Eastern Europe and the Commonwealth of Independent States: Reversing the Epidemic – Facts and Policy Options* (Bratislava, New York and Moscow: United Nations Development Programme, February 2004), available at: <http://hdr.undp.org/reports/detail_reports.cfm?view=876>.

Tony Barnett and Justin Parkhurst, 'HIV/AIDS: Sex, Abstinence and Behaviour Change', *Lancet Infectious Diseases* Volume 5 (September 2005): 2–5.

*The Economist* (2005) 'Aids in China: Anatomy of an Epidemic' (28 July), <http://www.economist.com/World/asia/displayStory.cfm?story_id=4223578> (accessed 25 August 2005).

Eileen Stillwaggon, *AIDS and the Ecology of Poverty* (Oxford and New York: Oxford University Press, 2005).

Gerald Stine, *AIDS Updates* (published annually by Pearson Education as Benjamin Cummings, San Francisco).

*UNAIDS Global Epidemic Report* (published biannually by UNAIDS/WHO), available at <http://www.unaids.org/>.

# 2
# The Disease and its Epidemiology

HIV/AIDS is not the first global epidemic, and it certainly won't be the last: it is a disease that is changing human history. HIV/AIDS shows up global inequalities. Its presence and impacts are felt most profoundly in poor countries and communities. Here we look at its origins, how it is transmitted and the particular characteristics which make consideration of its social and economic roots and impacts necessary. Because of its scale and the international and local concern it evokes, we are confronted by quantities of information that may threaten to overwhelm us. Thus, in the last part of the chapter we look at data: what we know about AIDS and HIV, and how we know it, and how those data are used to construct particular accounts of the epidemic process.

Communicable diseases have been responsible for past epidemics and pandemics. They played an important role in human history and we had few defences against them. Bubonic plague, which spread from the Mediterranean ports of southern Europe in 1347, changed the course of European, and thus of world, history.

> Most historians now accept the plague's role in destroying feudal barriers to economic growth, and creating an instant demand for labour which had to be satisfied from a drastically reduced work force. In effect, the fourteenth century bubonic plague intensified the action of powerful structural forces which were turning Europe toward modernity. (McGrew, 1985, p. 40)

During the first outbreak of plague in Europe from 1347 to 1351, mortality varied at between one-eighth and two-thirds of the population. Overall, three out of ten Europeans may have died, some 24 million people (Watts, 1999 in Cook, 1999). Some historians have argued that consequent labour scarcity led to technical, social and religious innovation, and ultimately to capitalism.

While Europe was affected by epidemics, they devastated other regions of the world. From the middle of the last millennium, contact between Europe, the Americas, Australasia and parts of Africa proved disastrous for immunologically naive indigenous populations. Lacking defences against common European

**Box 2.1 Definitions**

An **epidemic** is a rate of disease that reaches unexpectedly high levels, affecting a large number of people in a relatively short time. Epidemic is a relative concept: a small absolute number of cases of a disease is considered an epidemic if the disease incidence is usually very low. In contrast, a disease (such as malaria) is considered **endemic** if it is continuously present in a population, but at low or moderate levels, while a **pandemic** describes epidemics of worldwide proportions, such as influenza in 1918 or HIV/AIDS today. (Barfield, 1997, p. 150)

diseases such as smallpox, typhus, measles and influenza, these populations fell ill faster and diseases were more virulent. Diseases spread easily and mortality rates were very high. The result was massive depopulation: whole peoples disappeared; others were so seriously depleted as to have been written out of history.

Documentation of this process begins with Columbus's landfall on the Caribbean island of Hispaniola. In 1492 at the time of his arrival, there were at least 1 million Taino people. A disease akin to smallpox appeared in 1519 and by 1550 the Taino were extinct (Watts, 1997, p. 88). This pattern of devastation was repeated throughout the Caribbean islands. The Aztec and Inca kingdoms of mainland South and Central America were next. The troops of the Spanish conquistador Hernán Cortès brought smallpox. It is estimated that the population of Mexico fell from 25.2 million in 1518 to 1.1 million in 1605. Similarly affected were the Inca to the south and Native American populations to the north. There, Spanish explorers had encountered a vibrant culture with towns and temples in the Mississippi valley. By the early 1700s this had vanished along with most of the people.

The role of disease in human history has been charted by a number of authors: initially by McNeil (1976) and most recently by Diamond (1999). McNeil began by posing the question, 'How did Cortès and his tiny band of less than 600 Spaniards conquer the mighty Aztec empire, whose subjects numbered many millions?' (McNeil, 1976, p. 1). Diamond's perspective is informed by a question posed by a Papua New Guinean: 'Why is it that you white people developed so much cargo and brought it to New Guinea, but we black people had little cargo of our own?' (Diamond, 1999, p. 14). For McNeil the disease was the key. Diamond, however, saw disease as part of a broader geographical determinism.

By the end of the nineteenth century the principles of disease transmission were generally known in Europe. The first well-known public health intervention was in 1854 when Dr John Snow tracked the source of an outbreak of cholera in London to a water pump in Broad Street. Closing the public pump brought the outbreak under control. However, it was not until 1883 that Robert Koch identified the cholera bacillus. The first identified 'germs' or disease-causing organisms were the bacilli of anthrax and tuberculosis discovered by Louis Pasteur



in the 1870s. In the latter part of the nineteenth century a flurry of activity (often associated with expansion of European empires) led to the identification of more 'germs' and linked them with specific diseases. Thus began scientifically based public health interventions.

Among these was the US-funded Yellow Fever Commission, which in 1900 identified the mosquito as the vector for disease transmission. In Havana anti-mosquito measures reduced the number of cases from 1,400 in 1900 to none in 1902. Public health interventions were being developed and seen to work.

Medical advances led to the development of vaccines initially for polio, and by the 1960s for most other major childhood illnesses. Global smallpox vaccination resulted in eradication of the virus; the last case was reported in Somalia in 1977. By the mid-twentieth century, drug and vaccine development suggested to many that the world might be entering a period when the battle against infectious disease could be won. The next challenge was viral disease.

Prior to the emergence of HIV/AIDS, the last global epidemic had been influenza in 1918–19, so long ago that there was little 'institutional memory' of global epidemics. In the wealthy world there was also little memory of any killer epidemics. Poliomyelitis ceased to be a major concern with the introduction of a vaccine in 1955. Between 1946 and 1955 in the US there were on average 32,890 cases per year and 1,742 deaths. After the introduction of vaccination the number of cases fell to 5,749 and deaths to 268 (Oldstone, 1998, p. 109). In the rich world preventable diseases are generally prevented. Most people have clean water, heat, decent housing, nutritious diets and access to health care. The diseases that kill the rich are diseases of affluence such as heart disease. Outside of the rich world there have been major successes in immunisation against childhood diseases, although large numbers of children are still not reached and they die.

Where epidemics do emerge, scientific and medical responses are mobilised and emergencies are contained. However all is not well. Public health systems are underfunded; politically they attract few votes, and in parts of the world they are close to collapse. For the moment, there is only a mere intimation of any system of *global* public health.

Neither public health nor clinical medicine pay sufficient attention to what does improve health – escaping from poverty, access to good food, clean water, sanitation, shelter, education and preventative care. Clinical medicine has only marginal effects on people's long-term health. In the US – which spends the largest proportion of GNP on medical care of any country – 'less than 4% of the total improvements in life expectancy can be credited to twentieth century advances in medical care' (Garrett, 2000, p. 10). Preventive medicine is often piecemeal. For example, measles immunisation may be undertaken in slums where diarrhoeal disease is rife. Social and economic conditions negate many gains made by any particular intervention. Health is not only about confronting

individual diseases. Well-being, of which health is a part, is a reflection of general social and economic conditions.

The 1990s has seen the recognition of many 'emergent' diseases – Ebola, Lassa fever, Marburg fever are well-known and hit the headlines. More serious is multi-drug resistant TB. Also of concern are the rise of antibiotic-resistant bacteria, new strains of salmonella and most recently bovine spongiform encephalopathy (BSE) and the related human form, new variant Creutzfeld-Jakob Disease (nvCJD).

HIV/AIDS has emerged into this setting. It is the first global epidemic for 60 years. Working from past experience, many hoped that the solutions lay in a quick technical fix – drugs or a vaccine. But there has been no medical-scientific solution. With the exception of its first manifestations in the US, this disease is linked to poverty and inequality and the ways that globalisation exacerbates these. Its consequences will be felt for decades to come, and its origins lie far back in time and deep within the structures of social, economic and cultural life. The epidemic is not just about medicine or even public health.

## The emergence of the new epidemic: the discovery of AIDS and HIV

The story of HIV/AIDS begins in 1979 and 1980 when doctors in the US observed clusters of previously extremely rare diseases. These included a type of pneumonia caused by the fungus *pneumocystis carinii* (now sometimes called *pnuemocystis jaroveci*) and a previously rare cancer called Kaposi's sarcoma. *Pneumocystis carinii* is commonly found in rats, guinea pigs, monkeys, dogs, sheep, humans, and other animals. Humans with adequately functioning immune systems carry *pneumocystis carinii* as a harmless, latent and lifelong infection, so something was terribly wrong when a clustered appearance of these cases was reported. The phenomenon was first recorded in the *Morbidity and Mortality Weekly Report* (*MMWR*) of 5 June 1981, published by the US Centers for Disease Control in Atlanta. The *MMWR* recorded five cases of *pneumocystis carinii*. A month later it reported a clustering of cases of Kaposi's sarcoma in New York. Subsequently, the number of cases of both diseases – which were mainly centred around New York and San Francisco – rose rapidly, and scientists realised that they were dealing with something new.

The first cases were among homosexual men. As a result the disease was called Gay-Related Immune Deficiency Syndrome (GRID). American epidemiologists began to see cases among other groups, initially mainly haemophiliacs and recipients of blood transfusions. Subsequently the syndrome was identified among injecting drug users, and infants born to mothers who used drugs. It was apparent that this was not a 'gay' disease. It was renamed 'Acquired Immunodeficiency Syndrome', shortened to the acronym AIDS:

- The 'A' stands for Acquired. This means that the virus is not spread through casual or inadvertent contact like flu or chickenpox. In order to be infected, a person has do something (or have something done to him or her) which exposes him or her to the virus.
- 'I' and 'D' stand for Immunodeficiency. The virus attacks a person's immune system and makes it less capable of fighting infections. Thus the immune system becomes deficient.
- 'S' is for Syndrome. AIDS is not one disease but rather presents itself as a number of diseases that come about as the immune system fails. Hence, it is regarded as a syndrome.



The illness was seen simultaneously in a number of locations outside the US. In Zambia, Dr Anne Bayley, Professor of Surgery at the University Teaching Hospital in Lusaka, reported a significant rise in the number of Kaposi's sarcoma cases (Bayley, 1984). In 1982, reports of a significant wave of deaths in the south of the country began to reach the Ugandan Ministry of Health. In 1983 the ministry sent a team to investigate this new disease in the Lake Victoria fishing village of Kasensero. They concluded that it was AIDS (Kaleeba et al., 2000; Hooper, 1990). Hooper (1999) documents similar recognition of the disease in Tanzania, Congo and Rwanda.

In October 1983 a team of American and European doctors travelled to Kigali and Zaire where they identified and described cases of AIDS. Of course many hundreds of African doctors were well aware that a new disease was killing their patients. However, these frontline health care workers do not write for learned journals such as the *Lancet* or *Science* and *Nature*, so the cases and the disease remained unreported.

Outside Africa, AIDS cases were identified in all Western countries and in Australia, New Zealand and some Latin American countries – most notably Brazil and Mexico. From 1981 there was global recognition of the syndrome; clinicians and others now knew what to look for and that it could be given a name. Immediately there was a question of where HIV/AIDS was seen, by whom it was seen and what it meant. What it meant and how it was represented in the press and the popular consciousness was of the greatest significance for people affected by a disease linking sex, sexuality, death, ethnicity and status. Inevitably it became a vehicle for stigma (Farmer, 1992).

Once the new syndrome had been identified, the pace of scientific and epidemiological activity to identify the cause of the disease increased. In 1983 a team led by French scientist Luc Montagnier identified the virus we now know as HIV-1 (the Human Immunodeficiency Virus). In 1985, a second Human Immunodeficiency Virus, HIV-2, was identified. This is more difficult to transmit and is slower acting and less virulent than HIV-1. Initially HIV-2 was found in west Africa with the greatest number of infections outside this area in Angola, Mozambique, France and Portugal. 'Overall, the most striking feature about the

global epidemiology of HIV-2 is its lack of epidemic spread internationally' (De Cock and Brun-Vézinet, 1996).

Viruses have been defined as 'a piece of nucleic acid surrounded by bad news' (Oldstone, 1998, p. 8). They are genetic material covered with a coat of protein molecules. They do not have cell walls, are parasitic, and can only replicate by entering host cells. The genetic material of viruses is commonly DNA (deoxyribonucleic acid), or less frequently RNA (ribonucleic acid). Viruses have few genes compared with other organisms: HIV has fewer than ten genes (as does Ebola and measles); smallpox has between 200 and 400 genes. The smallest bacterium has 5,000–10,000 genes (Oldstone, 1998, p. 9). The human genome is estimated to contain 30,000 genes (US Department of Energy, 2003).

HIV belongs in the family of viruses known as retroviruses, scientifically called *Retroviridae*. The first retroviruses were only identified in the 1970s. All members of this family have the ability to produce latent infections. HIV is in a virus group called the lentiviruses. These develop over a long period, producing diseases, many of which affect the immune system and brain (Schoub, 1999). The viruses have a unique enzyme, reverse transcriptase. Outside the cells they infect, they consist of two strands of RNA. Once they infect a cell they make DNA copies of their own RNA and are able to reproduce. It is this feature as well as the ability of the virus to mutate rapidly which makes it hard to develop pharmaceutical responses.

## How HIV works

For infection to occur, the virus has to enter the body and attach itself to host cells (see Figure 2.1). HIV attacks a particular set of cells in the human immune system known as CD4 cells. There are two main types of CD4 cells. The first type are CD4 positive T cells which organise the body's overall immune response to foreign bodies and infections. These T helper cells are the prime target of HIV. For a person to become infected, virus particles must enter the body and attach themselves to the CD4 cells. HIV also attacks immune cells called macrophages. These cells engulf foreign invaders and ensure that the body's immune system will recognise them in the future.

Once the virus has penetrated the wall of the CD4 cell it is safe from the immune system because it copies the cell's DNA, and therefore cannot be identified and destroyed by the body's defence mechanisms. Virus particles lurk in the cells until their replication is triggered. Once this happens they make new virus particles that bud from the surface of the host cell in vast numbers, destroying that cell as they do so. These viruses then go on to infect more CD4 cells.

When a person is infected a battle commences between the virus and the immune system. There is an initial burst of activity during which many cells are infected, but the immune system fights back, manufacturing immense numbers of antibodies. This period is marked by an unseen and unfelt war in a person's



body. The viral load is high, the immune system is taking a knock, and th
person's HIV status cannot be detected using standard tests. This is commonl
called 'the window period' and lasts from several weeks to several months. A
this stage a person is highly infectious as his or her viral load (the number o
viral particles they are carrying) is considerable. This fact is of epidemiologica
importance. The more people there are in the early stage of infection, the greate
the chance of effective transmission between people.

An infected person will usually experience an episode of illness at the end o
the window period – but this will often resemble flu and will not be seen as
marker for HIV.

The window period is followed by the long incubation stage. During this phase
the viruses and the cells they attack are reproducing rapidly and being destroye
as quickly by each other. Every day up to 5% of the body's CD4 cells (abou

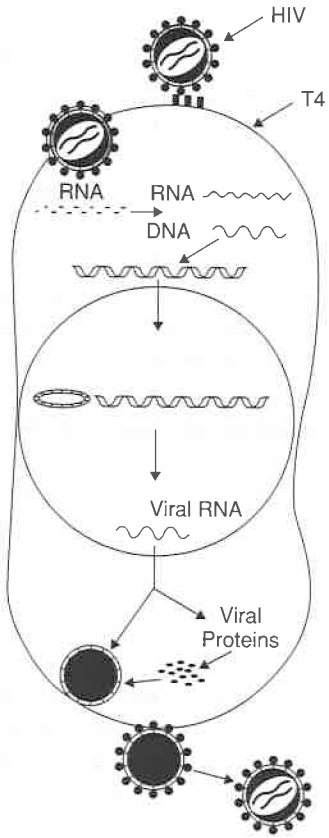



*Figure 2.1* The virus in action

*Source*: Whiteside and Sunter (2000, p. 7).

2,000 million cells) may be destroyed by the approximately 10 billion new virus particles produced daily (Schoub, 1999, p. 85). Eventually, the virus is able to destroy the immune cells more quickly than they can be replaced and slowly the number of CD4 cells falls. There is some medical disagreement as to the number of CD4 cells per microlitre of blood in healthy people. It is generally considered that a healthy CD4 cell count is in the range 500–1,600 cells. However, the US Department of Health and Human Services maintains that healthy adults usually have CD4 cell counts of 1,000 or more (US Department of Health and Human Services, 2005). As infection progresses, this number falls. When the CD4 cell count falls below 200 a person is said to have AIDS. Additionally, according to the US Centers for Disease Control (CDC, 2005), the definition of AIDS includes some 26 clinical conditions characteristically affecting people with advanced HIV. Some may experience early symptoms of HIV disease with CD4 counts above 200, while others show no symptoms with CD4 counts below 200. The generally acknowledged CD4 count for beginning drug therapy varies between 200 and 350. Some opportunistic infections occur at CD4 counts as low as 75. Infections will increase in frequency, severity and duration until the person dies. These opportunistic infections constitute the syndrome referred to as AIDS.

The period from HIV infection to illness and death is crucial. It was generally believed that, in the rich world, on average people lived for ten years before they began to fall ill. Without treatment, the normal period from the onset of AIDS to death was thought to be a further 12–24 months. With the development of effective anti-retroviral therapies, infected people can expect to live a reasonable life for a longer time. Indeed, it is hoped that AIDS can be turned into a manageable chronic disease like diabetes. In this event, people could expect to live longer though they would remain infected. However, recent evidence suggests that viral resistance to these drugs is growing; approximately 20% of new HIV diagnoses in the UK are of drug-resistant mutations.[1] If, as is feared, this phenomenon is generalised, then the threat from the epidemic is as great in the future as it is in the present.

Development and use of anti-retroviral therapies creates new problems:

- the virus mutates – there are over 120 sites in its structures which can mutate, and 'with hundreds of millions of virus particles being produced daily, it is not difficult to see how readily mutations occur which give rise to a wide range of biological variants even within the same individual' (Schoub, 1999, p. 87). This gives rise to drug resistance
- if people perceive AIDS as 'just' a chronic manageable condition they may be less inclined to take precautions against infection.

The incubation period in the developing world was thought to be shorter – between six and eight years. This was based on the assumption that people in



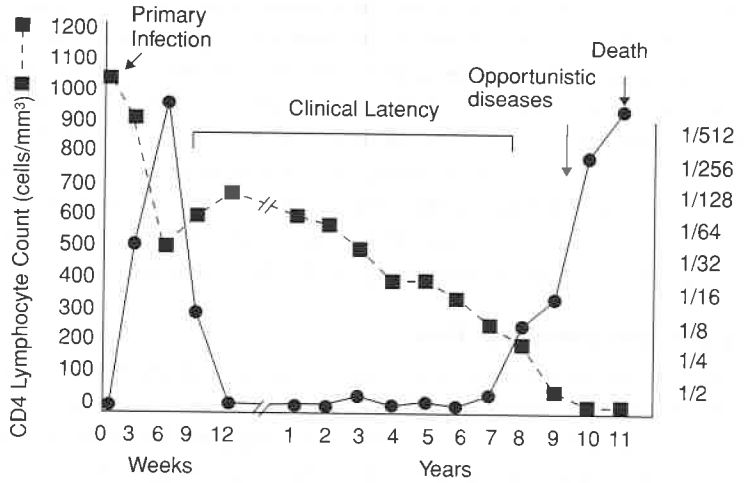



*Figure 2.2* Viral load and CD4 cell counts over time

*Source*: Whiteside and Sunter (2000, p. 9).

the poor world had more challenges to their immune systems, poorer nutrition and less access to health care. It seemed inevitable that they would progress to symptomatic AIDS faster. However, of six African studies reported in 1996, four suggested progression rates similar to those in the industrial world, and two found shorter periods. Data then were 'scanty and are limited to sub-Saharan Africa' (Mulder, 1996, p. 15). Schoub (1999) notes, 'little is known as yet about the rate of progression in African patients where the prognosis appears to be considerably worse (than among homosexual men in Western countries)' (Schoub, 1999, p. 42; parentheses added).

One study found that the time from HIV illness to death is shorter for untreated patients in Uganda than in the rich world, and the spectrum of HIV/AIDS-related disease is different. However the period from infection to illness did not seem to vary. This suggests that tropical diseases and infections such as TB or sexually transmitted infections do not hasten the progression of HIV to AIDS in Uganda (French et al., 1999, p. 509).

The issue of how long a period a person has between infection and illness is crucial for planning for the epidemic's economic and social impact. There is no one easy answer: time from infection to illness and from illness to death appears to be linked to disease environment, availability of health care and other factors. The period from onset of symptoms to death is shorter in poor countries. This has been borne out by a number of studies, for example French et al. (1999), who speculate that it is because patients do not receive early and appropriate treatment – an obvious issue in resource-constrained environments.

The differences between the poor and rich worlds also apply to the rich and the poor worldwide, and come down to the following: people who are able to eat enough nutritious food, who lead stress-free lives and who are not exposed to multiple infections will stay healthy and live longer. This is true generally and does not apply just to those who are HIV infected. However HIV infection throws inequality into even starker relief. 'Extreme poverty deprives people of almost all means of managing risk themselves' (World Bank, *World Development Report*, 2000/01, p. 146). For the poor, HIV is more likely to be a death sentence than for those who can care for themselves and afford treatment.

### Detecting HIV and describing AIDS

HIV was hard to locate because it is a retrovirus, hiding itself in the body's immune system. The first tests detected the *antibodies* to the virus rather than the virus itself. These might be compared to footprints on a sandy beach: they show a person has been there even though that person cannot be seen. Antibodies show that a person has been (and in the case of HIV, is) infected. Even today, most screening and diagnostic tests are based on discovery of antibodies rather than the virus. These tests have a high degree of sensitivity (which means that they do not miss positive results – if the person is infected then the tests will show this) and specificity (which means that they do not miss negative results – if the person is not infected the tests will not suggest that they are). The most advanced tests have reduced the window period to about three weeks. People are said to be HIV-positive when the HIV antibodies are detected in their blood.

It is more difficult to define AIDS. In areas where CD4 counts and viral loads can be measured, people are regarded as having AIDS when their CD4 count falls below 200. In most settings, however, the capacity to carry out such sophisticated tests does not exist. In such places AIDS is defined clinically by examining the patient and making an assessment of his or her condition. A number of opportunistic infections, some of which are common in HIV-infected people, take particular advantage of a depleted immune system. TB is one of these. Complicating matters further, new drug therapies make it possible for people to move from a state of AIDS, when they are very sick, to one of being HIV-positive and leading a fairly normal life.

### The origin of HIV

HIV derives from a virus that crossed the species barrier into humans. It is closely related to a number of Simian (monkey) Immunodeficiency Viruses (SIVs) found in Africa. The evolution of the virus over time is traced through a 'family tree', as shown in Figure 2.3. This differs from the more familiar family tree because to read it you must start near the middle. In this case, the proximity of the different types of virus is an indication of how closely they are related. For example, HIV-1 is clearly related to chimpanzee SIV and HIV-2 to macaque SIV.



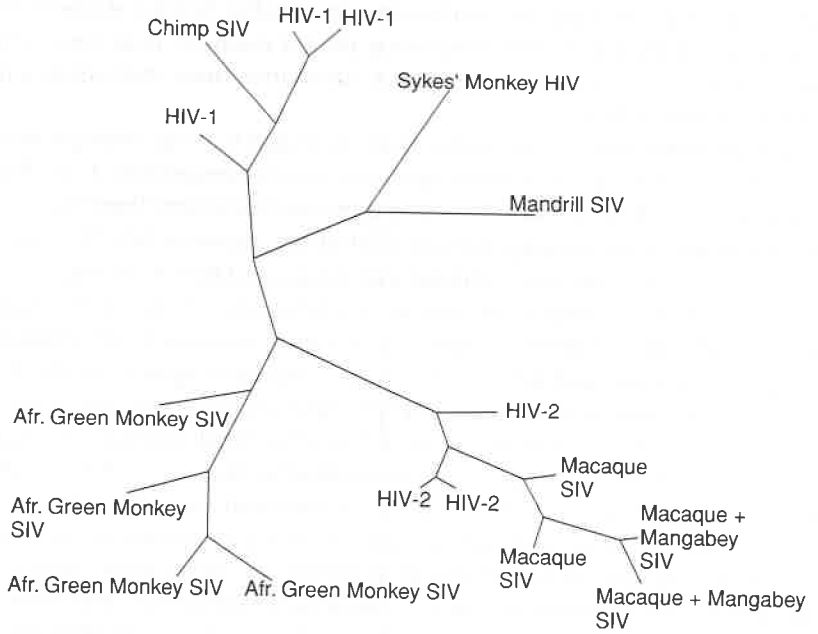



*Figure 2.3* The HIV family tree

*Source*: Wills (1996).

How did HIV enter the human population? Here we need to make a brief diversion to look at some other diseases. An important starting point is that the spread of diseases from animals to humans is not unique to HIV. Indeed, we know that human diseases also spread to animals – but animals do not have access to science and the media, thus this goes unrealised and unremarked by most people. The influenza virus evolves in birds – waterfowl to be exact.[2] Virologists describe these birds as 'reservoirs' of infection. They carry nearly all known types of influenza, frequently with no ill-effects, and spread them to the rest of the animal kingdom through their faeces. Hence, many kinds of animals can get flu – horses, ferrets, seals, pigs – as well as human beings.

However, viruses can only infect and take over a cell if it has a proper 'receptor'. Human cells do not have a receptor enabling them to contract avian flu directly. For human infection to occur another species must usually act as an intermediary; it can play this role by having a receptor for avian flu and humans in turn having a receptor for its flu. Pigs are one such species. The process can be as simple as a flu-contaminated duck dropping faeces into the dirt in which a pig then rolls. The pig is then infected and passes the virus on to a farmer. It can also be more complex. It is possible for a pig to be infected with one kind of flu, say human flu, only to contract another avian flu. The pig then has two

types of flu simultaneously. When the pig re-infects the human, it passes on a pig-bird-human influenza. The Hong Kong flu, for example, held seven genes from a human virus and one gene from a duck virus: these met inside a pig, producing a new hybrid.

There are three types of influenza virus, A, B and C. Type A originates in aquatic birds and is capable of infecting a wide variety of mammals. Types B and C are mostly found in humans. Wild and domestic birds carry these flu viruses as harmless and latent passengers in their intestines. However, in other species, particularly humans, the virus is highly contagious and may be lethal.

The Hong Kong flu virus is one among a wide variety of subtype flu viruses. The 1997 outbreak type H5N1 changed the history of avian flu by infecting both animals and humans (see Table 2.1). Two cases of human infections by the H7N7 strain had previously been reported, in 1995 (inflammation of the eye) and 1999 (respiratory infection). The H5N1 virus, on the other hand, infected 18 humans in China and the Hong Kong Special Administrative Region in 1997; six died. Poultry farms and markets were the foci of infection and a slaughter of domestic birds was ordered. During the latest outbreak in Asia, considered the largest in terms of geographical spread and viral prevalence, more than 120 million birds died or were destroyed within three months between December 2003 and March 2004. During the same period there were 35 human cases in Thailand and Vietnam. Cambodia, China, Indonesia, Japan, Lao People's Democratic Republic, Malaysia, Korea, Thailand and Vietnam experienced the H5N1 Avian flu outbreak in 2004 (WHO, 2005a). By 2006 this had spread to western Europe.

*Table 2.1* Major H5N1 influenza outbreaks

| *Year* | *Strain of influenza* | *Animal species implicated* | *Country/region* | *Target* |
|---|---|---|---|---|
| 1959 | H5N1 | Avian | Scotland | Animal |
| 1991 | H5N1 | Avian | England | Animal |
| 1997 | H5N1 | Wild geese, poultry | Hong Kong | Animal/human |
| 2003 | H5N1 | Poultry | Asia | Animal/human |
| 2004 | H5N1 | Poultry | Asia | Animal/human |

*Source*: Ebrahim (2004), WHO (2005a).

Humans typically experience more harmless symptoms like fever, cough, sore throat and muscle aches, but also serious diseases like eye infections, pneumonia and respiratory diseases. Symptoms of bird flu depend on which virus caused the infection. Viruses inhabit bird saliva, nasal secretions and faeces. Birds become infected when in contact with contaminated excretions or surfaces. Humans become infected through contact with infected poultry or contaminated surfaces. Typical influenza prescription medicines would prevent bird flu infection in humans, but flu viruses can become resistant to these medicines (CDC, 2005).



Such is the case with current concerns about the possible global spread of avia flu, and in this context the implications for people whose immune systems ar impaired by HIV are serious.

*Table 2.2* Cumulative number of confirmed human cases of avian influenza A/(H5N1), 28 January 2004 to May 2005

| Country/Territory | Total cases[a] | Deaths |
|---|---|---|
| Cambodia | 4 | 4 |
| Thailand | 17 | 12 |
| Vietnam | 68 | 36 |
| **Total** | **89** | **52** |

[a] Total number of cases includes number of deaths. WHO reports only laboratory-confirmed cases. Report date: 4 May 2005.

*Source*: WHO (2005a).

It is not just different viruses that can combine to create new and possibly more deadly diseases in the host. Viruses, and indeed all diseases, also replicate themselves within the host. This gives rise to variants of the virus within one person. These may in turn recombine to create new variants, some of which may be more virulent or drug resistant.

The speed with which HIV-1 replicates makes it a formidable enemy. There are two major strains of HIV-1. Group M causes over 99% of the world's HIV/AIDS infections. Groups O and the newly discovered N cause the remainder (Stine, 2001). Group M is divided into 11 subtypes or clades (A to K). The ability of the virus to mutate rapidly has significance in the quest for both a cure and a vaccine.

The question of when and how HIV entered human populations has been a source of great debate. We know that at some point the virus entered the blood of humans and then spread through sexual contact from person to person. In west Africa the less virulent HIV-2 spread from macaque monkeys. HIV-1 spread from chimpanzees into humans in central Africa. Four lines of evidence have been used to substantiate the zoonotic (transmission of a disease from one species to another) origin of AIDS:

1. similarities in organisation of the viral genome
2. phylogenic[3] relatedness of a particular HIV strain to that of SIV in the natural host
3. geographical coincidence between the SIV and particular HIV strains
4. plausible routes of transmission (Van Rensburg, 2000).

How did HIV cross the species barrier? We know that it is not an easily transmissible disease. It is carried in body fluids, with the highest concentration in blood, semen and vaginal secretions. For transmission to occur it had to enter the human body and reach the susceptible cells. It thus had to breach the skin or mucosal barriers. There are a number of hypotheses as to how this might have happened:

- *Bush meat*. It is not hard to imagine a hunter killing, or someone butchering, an infected monkey and in the process contaminating a cut on his or her hand with the monkey's blood.
- *Contaminated vaccine*. This is most elegantly (and lengthily) argued by Hooper (1999). He suggests that experimental polio vaccination campaigns in central Africa in the 1950s, using vaccine cultured on chimpanzee kidneys, may have provided the opportunity for the virus to cross the species barrier.
- *Contaminated needles*. The arguments above may explain how the virus crossed into humankind but they do not explain the rapid spread. It has been suggested that vaccine campaigns and poorly equipped clinics in rural Africa may have contributed to this through the use of unsterilised needles on one patient after another.
- *Ritual behaviour*. Finally, it has been suggested that use of monkey blood in certain rituals might have caused transmission. This hypothesis reflects a high degree of ethnographic ignorance and no little prejudice, as no one has described these rituals or given any examples as to where they take place.

The second and third hypotheses place the beginnings of the epidemic in the twentieth century. Hooper suggests that the polio campaigns of the late 1950s in Congo and Rwanda were the spark that ignited the fire. The cut hunter view has been used to suggest that the epidemic originated in infection across the species barrier in the 1930s.[4] Interestingly, in this case the transfer of the virus from an animal into a human may have happened on a number of previous occasions. However, because on those occasions each infected person did not in turn infect more than one other person, the potential epidemic petered out. There could have been a pool (or pools) of infection among isolated peoples in some parts of Africa for many years. What was different about the crossing of the species barriers in the 1930s (and the subsequent pattern of the epidemic) was the environment into which the virus was introduced. The upheavals of the colonial and post-colonial periods and development of modern transport infrastructure allowed HIV to spread quickly into the global community.

When all is said and done, the debate about the exact manner of zoonotic transmission is largely irrelevant. What matters today and in the future is that the virus has infected humans and is spreading fast.



## Modes of infection

Fortunately for humankind, HIV is not a robust virus and it is hard to transmit Unlike many diseases, it can only be transmitted through contaminated body fluids. For a person to be infected, the virus has to enter the body in sufficien quantities. It must pass through an entry point in the skin and/or mucou membranes into the bloodstream. The main modes of transmission, in orde of importance, are:

- unsafe sex
- transmission from infected mother to child
- use of infected blood or blood products
- intravenous drug use with contaminated needles
- other modes of transmission involving blood; for example, bleeding wounds.

### Sexual transmission

The vast majority of HIV infections are the result of sexual transmission. Initially most cases were discovered among homosexual men. This was because HIV was first identified in this group in the West. Moreover, the chances of infection are higher during anal intercourse than vaginal sex. Box 2.2 indicates the limits to our knowledge of the risks of sexual transmission and Figure 2.4 shows fluctuations in risk over the course of the infection. The relative probability of HIV infection per type of exposure is shown in Table 2.3. There is a small chance that HIV can be transmitted through oral sex, especially if a person has abrasions in the mouth or gum disease.

*Table 2.3* Routes of exposure and HIV

| *Infection route* | *Risk of infection* |
|---|---|
| *Sexual transmission* | |
| Female-to-male transmission | 1:700 to 1:3,000 |
| Male-to-female transmission | 1:200 to 1:2,000 |
| Male-to-male transmission | 1:10 to 1:1,600 |
| Fellatio | 0 to 6% |
| *Nosocomial transmission* | |
| Transfusion of infected blood | 95:100 |
| Needle sharing | 1:150 |
| Needle stick | 1:200 |
| Needle stick/AZT[a] PEP (Post Exposure Prophylaxis) | 1:10,000 |
| *Transmission from mother to infant* | |
| Without AZT treatment | 1:4 |
| With AZT treatment | Less than 1:10 |

[a] AZT is an anti-retroviral drug also known as Zidovudine.

*Source*: Adapted from Cohen (2004).

**Box 2.2 Routes of exposure and HIV: limitations of estimations of sexual transmission**

Complex sexual behaviours with potential concomitant exposure of several different mucosal sites; anal intercourse in heterosexual couples may be fairly common.

Sexual history provided by study subjects limited by their memory. All studies depend on the reports of study subjects about the quantity and quality of sex, signs and symptoms of sexually transmitted diseases (STDs), use of medications, and so on. Sexual diaries have proven cumbersome and sometimes inaccurate.

Lack of knowledge of the HIV status of the sexual partners (except in the case of discordant couples).

Unrecognised or undetected factors that can amplify transmission, especially STDs. Many STDs are asymptomatic yet might still increase the risk of HIV acquisition.

Long periods of follow-up between visits of people at risk, confounding accurate interpretation of risk. HIV-uninfected subjects probably suffer only very brief periods of high risk, but if they are studied infrequently a large number of low-risk sexual encounters are included for consideration, reducing the calculated probability of HIV transmission.

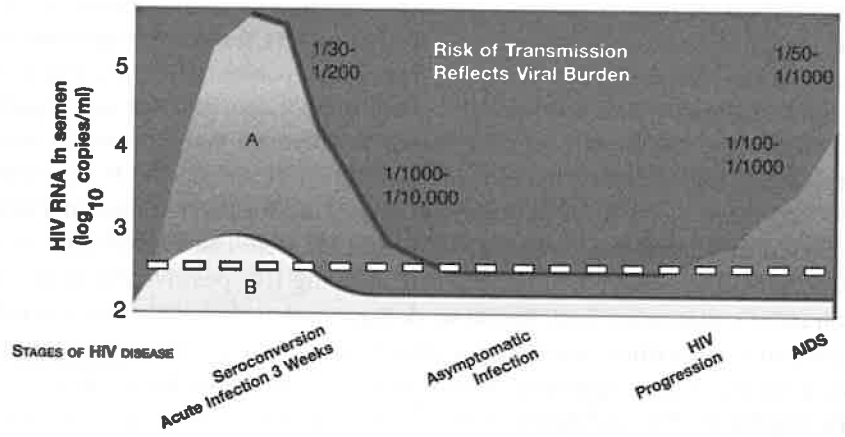
*Source*: Cohen (2004).

The presence of STDs, particularly those involving ulcers or discharges, will greatly increase the odds of HIV infection. An STD means that there is more chance of broken skin or membranes allowing the virus to enter the body. Furthermore, the very same cells that the virus is seeking to infect will be concentrated at the site of the STD because these cells are fighting the infection.

### Mother-to-child transmission

After sexual transmission, the next most important cause of HIV infection is mother-to-child transmission (MTCT). It is known that the child can be infected with HIV prenatally, at the time of delivery, or postnatally through breastfeeding. Infection at delivery is the most common mode of transmission. A number of factors influence the risk of infection, particularly the viral load of the mother at birth – the higher the load, the higher the risk. A low CD4 count is also associated with increased risk. Anti-retroviral drugs may decrease the viral load and inhibit viral reproduction in the infant, thus decreasing the risk of MTCT. A number of studies of the use of anti-retroviral drugs to combat MTCT have shown that the chance of this transmission can be greatly reduced at a relatively low cost and using fairly simple treatment regimes.






A Indicates HIV RNA copies per ml semen
B Indicates the reduced viral burden expected from host defences evoked by a vaccine or early use of anti-viral therapy

The dashed line offers a theoretical viral burden threshold below which HIV transmission will not occur. Numbers provided at the different stages of disease represent the probability of HIV transmission/episode of heterosexual intercourse.

*Figure 2.4* Sexual transmission of HIV: the relative risk of transmission over the course of the disease, as a function of viral load in semen

*Source*: Cohen (2004).

One cost-effectiveness study (Creese et al., 2002) found that there were large variations in the different strategies to reduce mother-to-child transmission. Single-dose Nevirapine to prevent MTCT costs from US$20 to US$341. Breastfeeding and formula-feeding interventions, on the other hand, cost from around US$4,000 to more than US$20,000 per infection prevented. Another study (Marseille et al., 2002) found that single-dose Nevirapine given to the mother during labour and to the child within 72 hours of birth costs as little as US$4 per mother–child pair. The efficacy rate for this intervention is about 50%. The advantages associated with this approach include its low cost and that more women have access to treatment as it is given during the later stages of labour. The costly part of the intervention remains the human resource intensive voluntary counselling and testing (VCT).

Mother-to-child transmission prevention programmes have not yet been implemented on a wide scale in regions and countries with scarce resources (WHO, 2004b). Service delivery remains complicated, due to fragile health systems (WHO, 2004a).

An important issue requiring clarification is the role of breastfeeding. On the one hand, formula feeding reduces the risk of MTCT; on the other hand, it increases the risk of children dying of other causes, particularly when they live in poverty. Breastfeeding has been promoted in poor countries for many years as part of child health and survival strategies. There are many problems with formula feeding, including the cost and availability of the product in the short and long term, access to clean water, the means and fuel to boil the water and prepare the feed, and knowledge of how to mix the feed. The formula approach also means that women can be 'labelled' as being HIV-positive, by virtue of their using replacement feed. Recent work suggests that the key to reducing risk is consistency in either breastfeeding or formula feeding an infant. Mixing the two is the most risky approach. 'A baby who is fed both the breast-milk of an HIV-positive mother and poorly made-up formula feeds is "getting the worst of both worlds"' (Chinnock, 1996, p. 15).

### Infection through blood and blood products

Use of contaminated blood or blood products is the most effective way of transmitting the virus as it introduces the virus directly into the bloodstream. This is one of the reasons why so many haemophiliacs were infected during the early years of the epidemic: they received unscreened blood products. It also accounts for early infections among recipients of blood transfusions. Fortunately, in most countries, the risks of transmission through this route are now minimal. Blood banks seek to discourage those who might be infected from donating blood, and the technology is available to test all donations. However, because of the window period when people are infected but the antibodies are not detectable, the risk of infection cannot be entirely eliminated. The problem is greatest where blood is sold by donors and this gave the initial impetus to the epidemic in a number of Asian countries.

### Intravenous drug use

Drug users who share needles are at risk of infection. If the equipment or drugs are contaminated, then the virus will be introduced directly into the body. This has driven the epidemic in Eastern Europe, the former Soviet Union and parts of Asia.

### Other modes of transmission

There is a possibility that HIV may be transmitted in other ways. Medical or other instruments that are contaminated can transmit the virus. Examples include dental equipment, syringes and tattoo needles. Sterilisation procedures should ensure that this does not happen. Accidents through needlestick injury or during surgery are a concern for medical staff. Standard precautions, use of gloves and sterilising equipment, will protect doctors and nurses against HIV transmission from patients, and vice versa.



## Responding to the disease

First prize with any disease is to prevent it. If prevention programmes had been successful, there would be no story to tell around HIV and AIDS. Unfortunately prevention programmes have not been successful in many parts of the world, and, where the epidemic has been controlled, no one is quite sure what actually worked (see Chapter 13).

### Prevention

The principle of successful prevention is ensuring that people are not exposed to the disease or, if they are, that they are not susceptible to infection. Vaccines provide the latter form of protection but are not yet available for HIV. Preventing infections through blood transfusion depends on screening all donations and discouraging potentially infected donors from donating their blood. Occupational exposure can be reduced through adopting universally accepted precautions regarding safety and sterility. In the event that a health care worker is exposed, immediate treatment with anti-retroviral therapy can greatly reduce the risk of infection. In the case of injecting drug users, simple procedures such as the use of sterilised needles and needle exchange programmes have been very successful in some countries.

### Preventing sexual transmission

As sex is the main mode of transmission, prevention strategies are most important here. One of the first responses to the epidemic was to call for the isolation of HIV-infected people. This was seen by many as impracticable, oppressive and discriminatory. The one exception is Cuba. In the 1980s the authorities tested the entire population, isolating those found to be HIV-positive in 'sanatoria'. This has contributed to the low level of HIV infection seen to date in that country. At the end of 2003 it was estimated that there were only 3,300 infected Cubans (UNAIDS, *Global Report*, 2004a). However, for this approach to work, a high degree of governmental control is necessary, people entering the country who might be infected and/or spread the disease have to be tested, and there has to be good border control. In addition, there needs to be a programme of regular repeat testing. This was never an option for most countries and certainly not for poorer countries. Apart from the expense and difficulty of implementing such a programme, some argue that it is a violation of human rights.

To prevent sexual transmission there is a limited but potentially effective range of interventions. The first set of interventions is 'biomedical'; these aim to reduce sexual transmission. Good sexual health is paramount. This means

that STDs should be treated immediately, and the availability of STD treatment in the rich world has probably played a major role in controlling HIV. Sexual practices that increase risk can be discouraged or made safer: a southern African example is 'dry sex' where a woman may use a drying agent in her vagina to increase friction during intercourse. This practice increases the risk of tears and abrasions, and can therefore facilitate the entry of the virus.

The most available biomedical intervention is the use of condoms. These provide a barrier to the virus and, if properly used, are effective. Both male and female condoms are available, but female condoms are more expensive and more difficult to use.

The second set of interventions seeks to prevent exposure to HIV by altering sexual behaviour; these are the Knowledge, Attitude, Practices and Behaviour (KAPB) interventions. First, people need to have *knowledge*, then they need to change their *attitudes* and finally alter their *practices* and *behaviour*. People are encouraged to stick to one partner, to delay first sexual intercourse, and to use condoms if they have more than one partner. This is the classic ABC message: A – abstain; B – be faithful; C – condom if necessary. The problem is that even if people have the knowledge, they may not have the incentive or the power to change their behaviour. If prevention is to move beyond knowledge to action, we must look at the socio-economic causes of the epidemic and intervene there too. (This is discussed in Chapters 3, 4 and 5.)

### Treatment[5]

Enormous resources have gone into the search for a cure and a vaccine. Neither has yet been developed. However, there have been major advances in clinical treatment.

Developments in treatment have resulted in declining mortality rates from HIV among the rich. There are three stages in the treatment of HIV-positive people. The first is when they are infected, but CD4 cell counts are high. At this point, the emphasis is on 'positive living' – staying healthy, eating the correct food, and so on. The second stage is when the CD4 cell count begins to drop. At this stage, prophylactic treatment to prevent TB and other common infections commences. The third stage is the use of anti-retroviral drugs to fight HIV directly.

Since the first anti-retroviral drugs were developed, many new generations of drugs have become available. At the moment anti-retroviral drugs may be used in single therapies (just one drug), double therapies (a combination of two drugs) or triple therapies (three drugs). The way the drugs act is shown in Figure 2.5. Single drug therapy is no longer used much because it causes fairly swift mutation of the virus into drug resistant strains. Dual therapy is cheaper than triple therapy, but the anti-viral effect is less immediate as the viral load falls slowly and the viral control may be of a limited duration. Highly Active Anti-Retroviral Therapy



(HAART) is any anti-retroviral regimen capable of suppressing HIV for many years in a significant number of individuals. Such is the case with triple therapy. It usually involves the use of two reverse transcriptase inhibitors and one protease inhibitor. Although not a cure, such treatments are effective in rapidly reducing the viral load to undetectable levels, thereby prolonging survival.

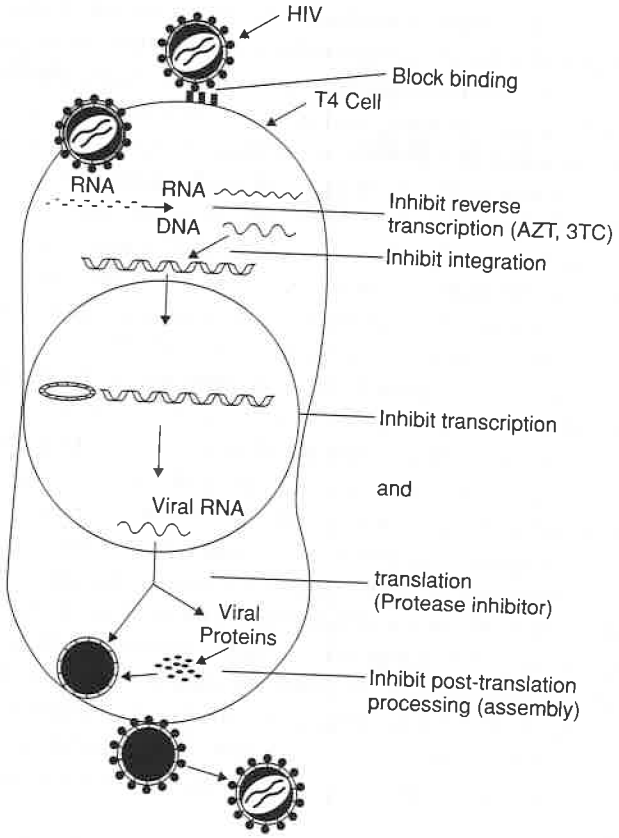



*Figure 2.5* Where the drugs act

*Source*: Whiteside and Sunter (2000, p. 23).

When to introduce a HAART regimen is of importance. Early treatment prevents damage to the body caused by high and prolonged viral loads – but it does use up the big guns sooner, which can decrease subsequent options if resistance builds up. That is why some clinicians prefer to step up the treatment gradually starting with single drug therapy. Cost is also a factor. The cost of anti-retroviral AIDS treatment in the rich world ranges between US$10,000 and US$20,000

per patient per year, although it can go much higher. Effective treatment of HIV/AIDS involves more than merely prescribing drugs: patients need regular consultations, testing for viral load and CD4 cell counts and, if treatment fails, testing for drug resistance. All this adds to costs.

Drug prices in poor countries have fallen drastically since 2000, due to the competition from generic producers in Brazil and India as well as political and activist pressure. The tendency has been clear: the lowering of drug prices over the five-year period 2000–05 demonstrates a combination of the development of a market; the effectiveness of political pressure from poor countries and some of their leaders; skilful advocacy; and in a few cases recognition by some rich country governments and influential individuals of moral responsibility to help those affected. Thus annual costs of patented medications have gone down from around US$10,000 to below US$600, while the prices of generic competitors have dropped from just below US$2,800 to below US$200. Figure 2.6 shows a sample of ARV triple-combination therapies, including Stavudine (d4T), Lamivudine (3TC), and Nevirapine (NVP), indicating the lowest world prices per patient per year. Brazil offered a price per patient per year of US$2,767 in August 2000, followed by the Indian company Cipla six months later in February 2001 with a price tag of US$350 per patient per year. The Aurobindo company had the price at US$209 by November 2002, while the Hetero company took the lead in this growing competition with a price of US$201 in April 2003 and reducing further to US$168 by January 2005. In turn, the originator pharmaceutical companies have responded to this generic competition and lowered their prices (MSF, 2005).

The new Global Fund for AIDS, TB and Malaria has become a major actor in these developments. It is an important response to a global problem and involves partnerships between the public sector, NGOs, development partners and the private sector of each country. In 2004, the Global Fund contributed 20% of all international HIV/AIDS funding and it managed to reach ten times more people compared to previous years, for example 130,000 people on anti-retroviral therapy and more than 1 million people reached with voluntary counselling and testing.

Despite price reductions, affordability and access remain an issue for the vast majority of people in poor countries. One study (Voelker, 2000) determined that for treatments to be affordable, HAART would need to be available at a monthly cost per person of US$10 for Zambia, US$20 for Botswana and US$45 for Mozambique. These figures assumed that it would be reasonable to spend 15% of the total health budget to treat 25% of the HIV-positive population. Another study from Uganda (Whyte et al., 2004) showed that access to ARV treatment remains highly uneven and produces new dilemmas of unequal access. The cost of triple combination therapy at US$28 per month is a very high proportion of combined public and private spending on health care in Uganda, which is estimated at US$38 per capita per year. The conclusion is clear: most Ugandans



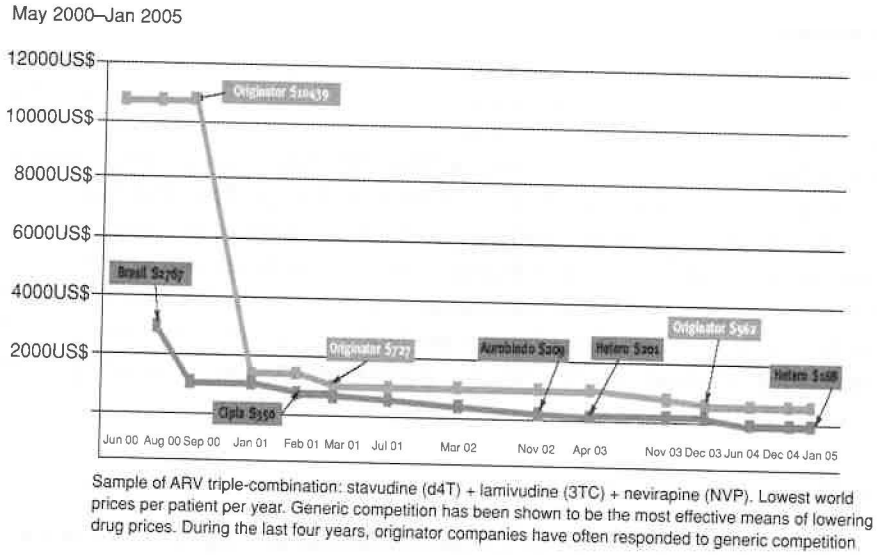



*Figure 2.6* The effects of generic competition on anti-retroviral prices: May 2000 to January 2005

*Source*: MSF (2005, p. 9).

cannot afford to pay US$28 per month to stay alive. Moreover, this amount only covers the cost of drugs. It does not include essentials like clinical consultations and monitoring, tests and drugs for opportunistic infections.

Only wealthier people can now afford these medicines, and even they have to make difficult decisions about whether and when to spend their household resources on drugs. Interviews by one of the authors in Uganda in 2004 showed that in poor rural communities, the absence of 35 cents to cover a sick woman's fare to a treatment centre was an insuperable obstacle to initial access. Such poverty is bound to affect adherence to treatment even when drug prices have fallen. When adherence falls below 95% of the total dosage in a month, the natural history of the virus population in the body means effective evolutionary selection for viral forms that are insensitive to particular drugs and indeed to whole classes of drugs. Viral resistance may become a serious issue accompanying the roll-out of ARVs in poor settings. There is evidence that effective ARV programmes are possible in these situations; however we do not know what will happen as availability of these medications becomes more widespread. Anti-retroviral therapies are used when patient CD4 counts fall and their immune systems fail. Before this happens most HIV-infected people will experience infection from other treatable diseases. These include candidiasis, meningitis and TB. In most of the poor world, drugs to treat these infections are not available or are too expensive.

### Vaccines

Intensive research is being carried out to develop a vaccine, so far with limited success. More than 15 years have passed since the first efforts, but as yet a vaccine remains elusive. Unfortunately the amount of money spent on researching AIDS vaccines is small (US$300–600 million a year) and is focused on strains found mainly in the US and Western Europe. The World Bank and the European Union, among others, have been involved in the search for new mechanisms and incentives to increase research and development of vaccines for developing countries. The International AIDS Vaccine Initiative (IAVI), based in New York, plays an increasingly important role in mustering resources and facilitating development. (The vaccine question is discussed in greater detail in Chapter 13.)

## HIV and other diseases

As their immune systems are progressively suppressed, other diseases will affect HIV-positive people. Most of these are not a threat to uninfected people. But people with HIV are very much more likely to develop active TB.[6] In the absence of HIV, the chance of developing TB is low. In the event that a person is co-infected with HIV, the chance rises greatly. It is estimated that 40–50% of people with TB in South Africa are co-infected with HIV, and one-third of people with HIV are expected to contract TB. This has to be seen against a general background of high TB infection in South Africa. The annual incidence there in 1998 was 254 per 100,000 people – in Europe it is 19; in China, 113, and in India, 187.

TB can be treated. For instance, the DOTS regime (Directly Observed Treatment, short course) has dramatically raised cure rates. But this is for all patients. Prophylactic treatment for HIV-positive people is far more costly and problematic. Not for nothing are HIV and TB variously referred to as 'the terrible twins' and 'Bonnie and Clyde'.

Some evidence suggests that there are links between HIV and malaria. It is possible that people with HIV contract malaria more easily and they certainly have a poorer prognosis.

So far we have described disease and processes in the individual body as a result of this particular virus. Disease is of social and economic significance. It causes groups of people to become infected, fall ill and die. HIV/AIDS is unique. The disease is sexually transmitted, therefore it affects prime-age adults; it is fatal and it is widespread. It is unusual for this group (prime-age adults) to be the target of any disease. This is why it has profound social and economic consequences. To understand the aggregate nature of disease, as a precursor to looking at these consequences, we need to understand something about HIV/AIDS epidemiology and epidemiology in general.



## Epidemiology

Epidemiology has been defined as 'the study of the distribution and determinants of health-related conditions and events in populations, and the application of this study to the control of health problems' (Katzenellenbogen et al., 1997, p. 5).

Epidemiology examines patterns of disease in aggregate. It describes the social and geographical distribution and dynamics of disease. However, as we shall see, this is not at all straightforward, especially with regard to HIV/AIDS, because:

- data can be confusing; often people do not distinguish between HIV and AIDS
- data quality is variable
- data are *constructed* according to a variety of implicit or explicit assumptions
- data may be *interpreted* according to biases which people bring depending on their discipline, politics or paymaster.

Data are important. We need to know where the epidemic is located and where it might spread if we are to design effective prevention interventions. If we want to consider the potential social and economic impact of an HIV/AIDS epidemic, we need to have some idea of the numbers of people who are infected with the virus, and who and where they are. We need to be able to predict how many people will fall ill and die and when this will happen. For example, an education department needs to know how pupil numbers will change and what effects the epidemic will have on teacher availability and training needs. In this section we are concerned with how we know about the epidemic, how we obtain data on the disease, how we understand it and interpret it and the policy implications of this understanding.

Epidemiology provides only some of the required information. In later chapters we shall add to what epidemiology has to tell us by reviewing another set of questions about *why* epidemics take different forms in different societies. Our argument is that there are social and economic characteristics which make an epidemic grow more or less rapidly. They determine whether the epidemic is concentrated in a few 'high risk' or 'core' groups or whether it becomes generalised to the wider population. These determinants, which make a society more or less *susceptible* to epidemic spread, are closely tied to the characteristics which make that society more likely to suffer adverse consequences resulting from increased illness and death. We use the term *vulnerability* to talk about this greater or lesser likelihood of adverse impact. (Chapters 4 and 5 describe and discuss susceptibility, while Chapters 6–11 discuss vulnerability and impact.)

### Epidemic curves

A key concept is the epidemic curve. HIV – indeed, any disease – will move through a susceptible population, infecting some and missing others. Epidemics follow an 'S' curve, as shown in Figure 2.7. They start slowly and gradually. At a certain stage, a critical mass of infected people is reached and the growth of new infections accelerates thereafter. The epidemic then spreads through the population until many of those who are susceptible to infection have been infected. Some are lucky because even though they are susceptible, they never come into contact with an infectious person. With modern transport networks there are few instances of isolated communities. Hence, epidemics can rapidly go global. The large and rising global population also means that many more people will be infected.

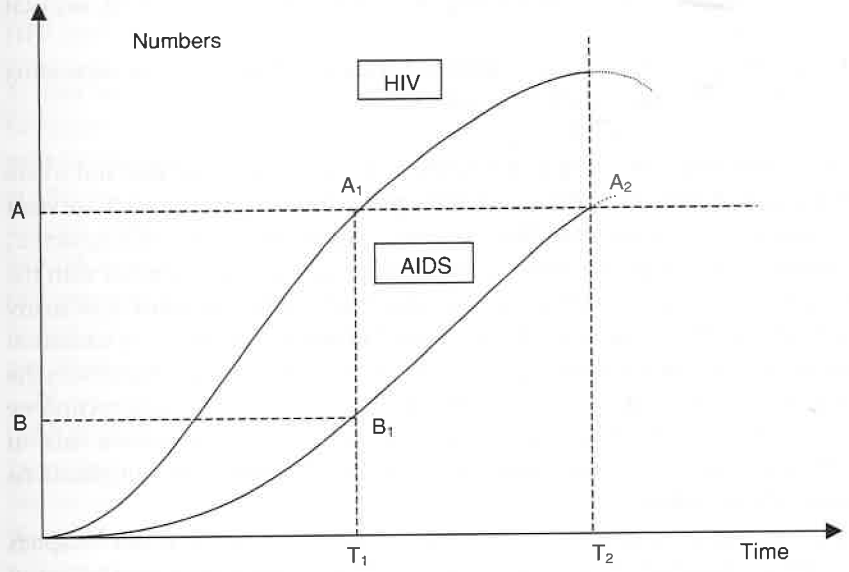



*Figure 2.7* The two epidemic curves

In the final phase of an epidemic – where the 'S' flattens off at the top and turns down – people are either getting better or deaths outnumber new cases so that the total number alive and infected passes its peak and begins to decline. With most diseases the curve will decline rapidly. HIV and AIDS are different.

What sets HIV and AIDS apart from other epidemics is that there are two curves, as shown in Figure 2.7. With most other diseases, infection is followed by illness within a few days or, at most, weeks. In the case of HIV the infection curve precedes the AIDS curve by between five and eight years. This reflects the long incubation period between infection and the onset of illness. This is why HIV/AIDS is in some ways such a lethal epidemic compared to, say, Ebola fever. In the latter case, victims of the disease quickly and visibly fall ill, putting the general population and public health professionals on their guard. The community takes precautions to halt spread and the infected person is rapidly immobilised, reducing his or her infective potential.



HIV infection moves through a population giving little sign of its presence. It is only later, when substantial numbers are infected, that AIDS deaths begin to rise. People do not leave the infected pool by getting better because there is no cure. They leave by dying (of AIDS or other causes). The effect of life-prolonging ARTs is, ironically, to increase the pool of infected people.

Figure 2.7 illustrates this point clearly. The vertical axis represents numbers of infections or cases of illness and the horizontal axis represents time. At time $T_1$, when the level of HIV is at $A_1$, the number of AIDS cases will be very much lower, at $B_1$. AIDS cases will only reach $A_2$ (that is, the same level as $A_1$) at time $T_2$. By then years will have passed and the numbers of people who are infected with HIV will have risen even higher.

Figure 2.7 also shows that while prevention efforts may aim to lower the number of new infections, the reality is that without affordable and effective treatment, AIDS case numbers and deaths will continue to increase after the HIV tide has been turned.

Beyond the point $T_2$, the lines are dotted. This is because we do not know how either the HIV or the AIDS curves will proceed. In a few poor countries, notably Uganda and Thailand, does national HIV prevalence (and incidence – see below) appear to have peaked and turned down.

Figure 2.7 shows an epidemic curve. But a national epidemic is made up of many sub-epidemics, with different gradients and peaks. These sub-epidemics vary geographically and in terms of their distribution among social or economic groups. In many countries in the poor world, HIV spread first among drug users and commercial sex workers (CSWs). From there it moved into other groups: mobile populations, men who visited sex workers, and eventually into the broader population. One common feature in both the rich and poor worlds is that HIV spreads among people at the margins of society, the poor and dispossessed. (Examples of national and sub-national epidemics are discussed further in the case studies in Chapter 4.)

### Incidence and prevalence

*Incidence* is the number of new infections which occur over a time period. The *incidence rate* is the number of infections per specified unit of population in a given time period. Rates can be per 1,000, per 10,000 or per million for rare diseases. The time may be per annum, but in the case of more rapidly moving infections it may be days or weeks. *Prevalence* is the absolute number of infected people in a population at a given time – it is a still photograph of current infections. The

*prevalence rate* is the percentage of the population which exhibits the disease at a particular time (or averaged over a period of time). A numerical example and an illustration appear in Table 2.4 and Figure 2.8, respectively.

Data on incidence and prevalence are key statistics for tracking the course of the HIV epidemic. With HIV, prevalence rates are given as a percentage of a specific segment of the population. Commonly used groups are antenatal clinic attenders, adults aged between 15 and 65, blood donors, men with STDs, or the 'at risk' population – usually taken to mean 15–to 49-year-olds who are sexually active.

*Table 2.4* Incidence and prevalence

| *Year* | *Population* | *Incidence (actual)* | *Incidence rate per 1,000* | *Prevalence* | *Prevalence rate (%)* |
|---|---|---|---|---|---|
| 1 | 9,750 | 0 | 0.0 | 0 | 0.0 |
| 2 | 10,000 | 50 | 5.0 | 50 | 0.5 |
| 3 | 10,500 | 50 | 4.7 | 100 | 1.0 |
| 4 | 11,000 | 150 | 13.6 | 250 | 2.3 |
| 5 | 12,000 | 750 | 62.5 | 1,000 | 8.3 |

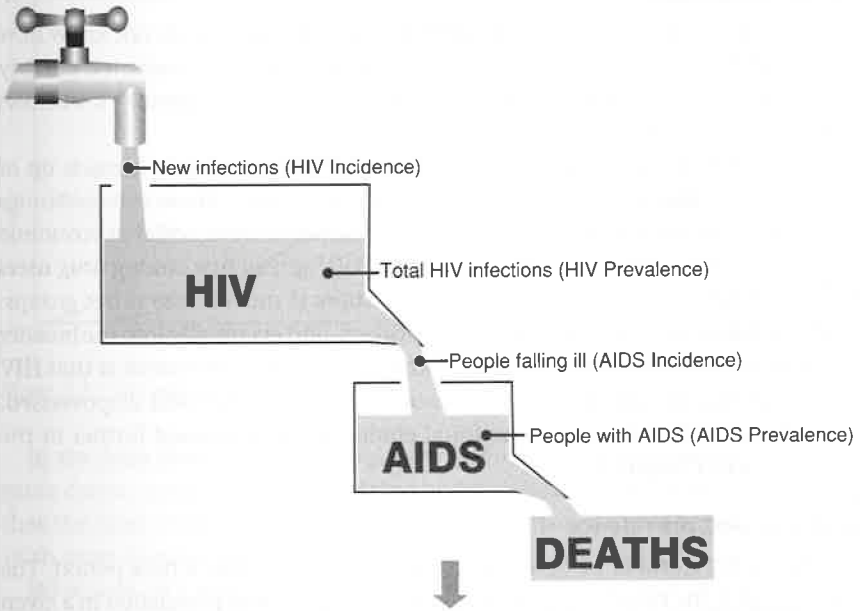



*Figure 2.8* HIV/AIDS incidence and prevalence

*Source*: Whiteside and Sunter (2000).



Uniquely, HIV prevalence is given as a percentage rather than as a rate, as is the case for other diseases. Why this is the case is not clear; it may be because of the need to communicate figures simply, or because advocates find percentages most compelling.

Annual incidence is calculated by subtracting the previous year's prevalence from that of the current year. Because we don't know when people were actually infected – we only know the date on which their serostatus is ascertained – the data (incidence), which would be most helpful in measuring the impact of prevention efforts, are simply not available. Moreover, high incidence may occur even when prevalence has levelled off, because those dying are being replaced by new infections.

Currently we have to use prevalence data to track how the epidemic is moving through a population, comparing one year with another. The aim of control and prevention measures is to reduce both prevalence and incidence. To achieve this the number of new infections produced by each existing infection must be reduced.

### The reproductive rate

The gradient, final height and rate of decline of an epidemic curve are determined by the average number of secondary cases generated by one primary case in a susceptible population and the period over which this takes place. This is also known as 'the basic reproductive number' and is represented by the symbol $R_0$ (Anderson and May, 1992; Anderson, 1999). In order for an epidemic to be maintained, $R_0$ has to equal 1; in other words, each person who gets better or dies has to infect one other person. At this point the disease is endemic but stable. When $R_0 > 1$, each person infects more than one other person, the number of cases will rise. When $R_0 < 1$ then the epidemic will be disappearing. In South Africa in 2000, the $R_0$ for HIV was estimated at 5, while that of malaria was 100 (Whiteside and Sunter, 2000, p. 10).

The percentage or number infected in a population depends on the degree of susceptibility of individuals in that population. This term is usually used in the narrow biomedical sense of transmission efficiency. 'Transmission efficiency is expressed as the probability that a contact will occur between infected and susceptible individuals multiplied by the likelihood that a contact will result in transmission' (Anderson, 1996, p. 73). In this book we argue that susceptibility is far more than the result of biomedical events in the body; understanding and acting on this insight is fundamental both to reducing the rate of spread of the HIV/AIDS epidemic and to dealing with its long-term economic and social consequences.

Most epidemics are of relatively short duration. This is determined by the time from initial infection to the end (recovery or death) of the infectious period. Cholera epidemics may last only a few months in any one location. A measles

epidemic with its typical two-week period from infection to illness will last between six months and one year. In the case of a disease where the gestation period is several years, the epidemic will last for decades. This is the case with HIV and it may be similar with nvCJD.

The HIV curve tells us where the epidemic has been. Projections tell us where it might go. HIV is not on its own important for understanding the social and economic impact of the epidemic. What is important is the AIDS curve (see Figure 2.7). If we are to consider impact we need to have an idea of the size of the potential AIDS epidemic which will hit a particular society.

How bad is the epidemic? How many people are infected and will die? How serious and global a crisis is it? These are all questions which are seldom posed in a precise way. Those who believe AIDS is a 'crisis' believe it is *the* major challenge facing most of the world. Thus 'Acquired immunodeficiency syndrome (AIDS) has become a major development crisis. It kills millions of adults in their prime' (General Assembly on HIV/AIDS, 2001). A memorandum issued on 2 June 1999 to World Bank staff and supporters announcing the new AIDS in Africa initiative (World Bank, *World Development Report*, 1998), stated: 'This fire is spreading. AIDS already accounts for 9% of adult deaths from infectious disease in the developing world. By 2020, that share will *quadruple* to more than 37%. The global death toll will soon surpass the worst epidemics of recorded history.'

Those who deny that there is an acute problem come in various shades: some say that there is no evidence of increased illness; others say that this can be explained by poverty, urbanisation or drug use. Even where the seriousness of the issue is recognised there is often debate over the exact figures. Effectively, people say: 'If you can't tell us exactly what is going on, why should we believe you at all?' This is a facet of denial processes, which appears throughout the history of the epidemic.

### Data sources

This section looks at how data are derived. We begin with AIDS case data and then go on to look at HIV. In Chapter 4 we establish the ways in which the epidemic trajectory differs from country to country, and how social, economic and cultural situations determine this. Here we provide a background to some of the difficulties in obtaining and interpreting such data.

Key data sources include governments, non-governmental organisations, academic establishments, and in some instances the private sector. Data are of variable quality but – *and this is important to note* – all data produced by all agencies originate from the countries themselves. Thus data reflect what is available in countries and what they choose to report. Epidemiologists and statisticians may make assumptions and extrapolate, but they are dependent on the information they are given.



Two main bodies collect and compile international data. The first is UNAIDS, which produces estimates of AIDS cases, HIV prevalence in various groups, numbers of deaths and orphans. These data are collected and published annually in the *Report on the Global HIV/AIDS Epidemic*. Thus we are told that in 1999, 5.4 million people were newly infected, 2.8 million died and 34.3 million were living with HIV/AIDS. These data often presented an unjustified impression of certainty and precision. In response to criticism, UNAIDS introduced a more transparent system of estimation in 2003 indicating ranges around the mean for its observations. Thus in 2004 we are told for the first time in the UNAIDS report that 4.8 million people were infected with a range between 4.2 million and 6.3 million people infected; 2.9 million people died with a range between 2.6 and 3.3 million, and that 37.8 million were living with AIDS – the range estimated as between 34.6 and 42.3 million.

As the epidemic has progressed, so UNAIDS and WHO have improved their surveillance and modelling methods and the demands of advocacy for simple, high and shocking figures have been trimmed in response to criticism. It is important to realise that estimates are based on *available* data, including surveys of pregnant women and household surveys from each country. Thus current estimates reflect current methodology in use, and data therefore do not compare to previous or subsequent ones. UNAIDS/WHO uses a six-step method to obtain national estimates of HIV prevalence. In countries with a generalised epidemic, national estimates are based on surveillance of pregnant women attending antenatal clinics. On the basis of these results over a number of years an epidemic curve can be drawn. This describes the evolution of adult HIV prevalence over time. The number of people infected, new infections and AIDS deaths are then calculated on the basis of the adult prevalence curve, population estimates and epidemiological assumptions. It is important to note that this approach assumes that the HIV prevalence among pregnant women is a good indicator of prevalence among the adult population, defined as those between 15 and 49 years of age (Grassly et al., 2004; UNAIDS/WHO, 2002, 2003a, 2003b; WHO/UNAIDS, 2002, 2003). This is not necessarily a sound assumption in all situations.

In countries with low level or concentrated epidemics, estimates of national HIV prevalence are based on surveillance data collected from 'high risk' groups (commercial sex workers, homosexuals, injecting drug users), along with estimates of the size of populations at high or low risk. The 'best fit curve' that describes the evolution of adult HIV prevalence over time is then produced. Similarly, the prevalence curve, national population estimates, and epidemiological assumptions form the basis for calculating the number of infected people, new infections and AIDS deaths (WHO/UNAIDS, 2002).

The second source of data is the United States Bureau of the Census. This is a primary source of data for UNAIDS. The Bureau deploys its considerable resources to collate official data and also data from many other published and

'grey literature' sources. The US Census Bureau runs an HIV/AIDS Surveillance website with tables, maps and a software program to download and use on PCs. The Bureau also offers an International Data Base, which is a computerised source of demographic and socio-economic statistics.[7] Staff of the Bureau can be seen at all conferences of note photographing posters, collecting papers and checking findings with people on site.

*AIDS case data*

In the early years of the epidemic, AIDS case data were the main source of information. Each year or month the 'body count' rose. This was most vividly demonstrated in Randy Shilts's documentation of the first few years of the epidemic, largely in the US (Shilts, 1987). As the 1980s unfolded, AIDS cases were reported from more and more countries across the world. Graphs were produced showing exponential increases in the numbers of cases and deaths. Unfortunately there was public confusion between HIV and AIDS, aided and abetted by press reports which failed to distinguish between infection and disease.

In the poor world, reporting required that someone actually took the time and trouble to notify public authorities that they were seeing AIDS patients. The question was and still is: 'Do we have a clear picture of the number of AIDS cases or deaths?' The answer is 'No', and indeed we never did.

In most countries AIDS is not a notifiable disease, which means that medical staff are not legally required to report cases. Even if they do, there are serious constraints to this process:

- reporting may be very slow. It takes time for data to flow into a central point and be collated
- data may be inaccurate because of unwillingness to report cases. This may be due to stigma associated with AIDS; to potential discrimination by medical insurance companies, not paying for treatment of AIDS-related conditions; and to the life insurance industry excluding claims where the cause of death is given as AIDS
- the condition from which a person dies may not be recognised as being AIDS related. Instead the patient may be recorded as having, for example, TB or meningitis
- doctors may feel that it is pointless to report cases as there is no incentive, they are too busy or they get no feedback.

Many people in poor countries are not seen by the formal medical services. Figure 2.9 shows the numbers of 'filters' a report has to go through before it becomes an official 'case'; in other words, before it is counted. The right-hand column shows the factors which can prevent this. Consider that somebody is dying in a small house, in a small village, several miles on foot from the nearest



| Person falls ill with AIDS | | **AIDS case not recorded because:** |
|---|---|---|
| ↓ | | |
| Is seen by formal medical service | → | Person visits traditional healer/does not seek care |
| ↓ | | |
| Is correctly diagnosed | → | Is not correctly diagnosed or diagnosed with an opportunistic infection |
| ↓ | | |
| Case recorded | → | Case not recorded |
| ↓ | | |
| Record sent to data collection point | → | Report not forwarded/lost in post, etc. |
| ↓ | | |
| Data collected and published | → | Report lost/not published |

*Figure 2.9* The problems of AIDS case reporting

motorable road and many miles from the nearest all-weather road. There is a small clinic ten miles away but the medical orderly has not been paid for several months and has little in the way of drugs or equipment. The person's family has exhausted its resources and strength in caring for her. How is this person to become a 'case' recorded in the capital city some 300 or more kilometres away?

The fact is that no poor country has counted its AIDS cases. Indeed even in hospitals, many of which lack test kits, we cannot know how many AIDS cases there really are. What then is the value of AIDS case data? First, if they are collected consistently and in sufficient quantities, trends will be apparent. Second, they can give an indication of the scale of the problem. Finally, they can show where the epidemic is located by age, gender, mode of transmission and geographical area. Figure 2.10 illustrates the situation in Malawi in 1995. The first cluster of cases is those resulting from mother-to-child transmission. The next is for young women, peaking in the 20–24 age group, and finally there is a cluster of male deaths in the 30–34 age group.

Most social and economic statistics have political ramifications. AIDS case data have always been 'political'. In the early years of the epidemic, countries were reluctant to admit to the existence of the disease because of what they felt its presence might suggest or imply about the morals and behaviour of their citizens, or what it might do to the tourist industry. This was the initial reaction in Kenya and Thailand.

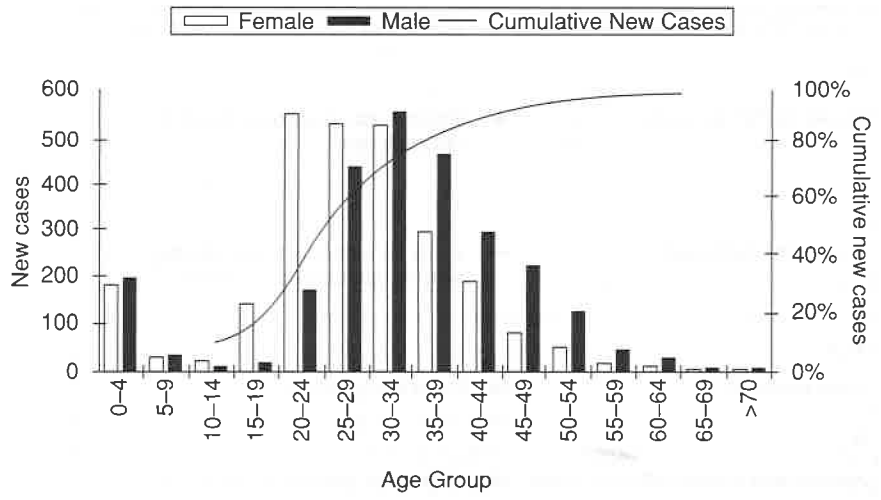



*Figure 2.10* Malawi – age and gender profile of new AIDS cases, 1995

*Source*: Loewenson and Whiteside (1997).

Perhaps the most telling example of the politicisation of data was in Zimbabwe. The first report to the Global Programme on AIDS (GPA) in Geneva was of several hundred cases in 1987. A few weeks later South Africa (then still under the apartheid regime) reported 120 cases. Within days the Zimbabwean government reduced its reported cases to 119 (*AIDS Analysis Africa*, 1990, p. 6).

The next potential data source is AIDS deaths. However, with few exceptions, there is no vital registration in poorer countries, and even where there is, information will not be collected on the cause of death by disease. Where death data are recorded information can be extracted (discussed in Chapter 6). But we must always remember that even if AIDS cases are accurately recorded at any given time, they reflect the HIV infections of five or more years earlier.

*HIV data*

HIV data tell us how many people are infected in a population, and are most frequently presented as prevalence. Ideally data would show exactly who in a population is infected and when they were infected. This would allow plans to be made for care and support of infected people and their families and human resource management. Deaths make families less able to provide for their members, the workforce less able to work, and increase demand for services such as health and welfare. Such data should also enable the epidemic to be tracked and the success (or failure) of interventions to be measured.

The ideal survey would cover an entire population. Every individual would give a blood or saliva sample for testing. Such a survey would furnish a point prevalence



(the prevalence at that point in time). To track the epidemic, subsequent survey would have to be carried out. This would be a logistical nightmare, would b costly and would raise ethical issues: do you compel people to take part? I people are identified then what do you do with them? As mentioned earlie this type of survey has only been done in Cuba – an island with a populatio of 11.1 million.

Second best would be a population-based random survey which samples me and women across age groups to provide a representation of the situation in th whole population within certain calculable bounds of error. Such surveys hav been done in a few places. They are expensive, require a lot of organisation raise ethical issues and need to be repeated if they are to have value. In the pas one of the major obstacles to population-based surveys was that the HIV tes required blood. Taking blood is an invasive procedure to which many people wil not consent. The development of saliva tests over the past few years has mad population surveys much more viable.[8]

Presently available data are drawn mainly from samples of specific populatio sub-groups. These are then extrapolated to larger populations. UNAIDS note that different types of epidemic require different types of surveillance:

> In largely heterosexually driven epidemics where there is evidence that men an women in the general population have become infected with HIV in significan numbers, HIV surveillance is based ... on pregnant women attending antenata clinics that have been selected as sentinel surveillance sites ... the more regula the studies, the clearer the picture of current prevalence. Where data are no available for the current year, all available data points are plotted on a curve and an estimate for the current year is made according to what is known abou the course of epidemics with predominantly heterosexual transmission. To account for differences in the spread of HIV, this is generally done separately for urban and for rural areas. (UNAIDS, 2000f, pp. 116–18)

Many sub-Saharan African countries and a few in Asia and the Caribbean have conducted regular antenatal clinic HIV prevalence studies since the end o the 1980s. Antenatal clinic attenders provide a good sample because they are sexually active and adult. A major advantage is that blood is routinely taken from women attending these clinics for a number of standard tests, and surveys can be repeated.

Population-based surveys were rare. However, where they have been done they show that in heterosexually driven epidemics the differences between these data and those from pregnant women are not great (Figures 2.11 and 2.12). Thus antenatal clinic data may be used cautiously as a proxy for the general population.

**Box 2.3 Antenatal surveys**

Antenatal HIV surveys are based on a sample of women attending antenatal clinics. A portion of the blood drawn for routine testing will be marked with the woman's age, the clinic's location and possibly some other social, economic or marital status data, and then sent for testing. This testing is called anonymous and unlinked. In other words, individual women cannot be identified as the source of a particular sample.

Such surveys should be done on a regular basis, either every year or every two years. In India they were initially done every six months to give rapid, consecutive results.

There are biases: younger women will be overrepresented as they are more sexually active and likely to fall pregnant; HIV-positive women will be underrepresented as HIV infection reduces fertility.

An obvious drawback is that the survey is confined solely to women attending state antenatal clinics. It does not cover those women who either do not have access to state health care or who can afford to see private practitioners.

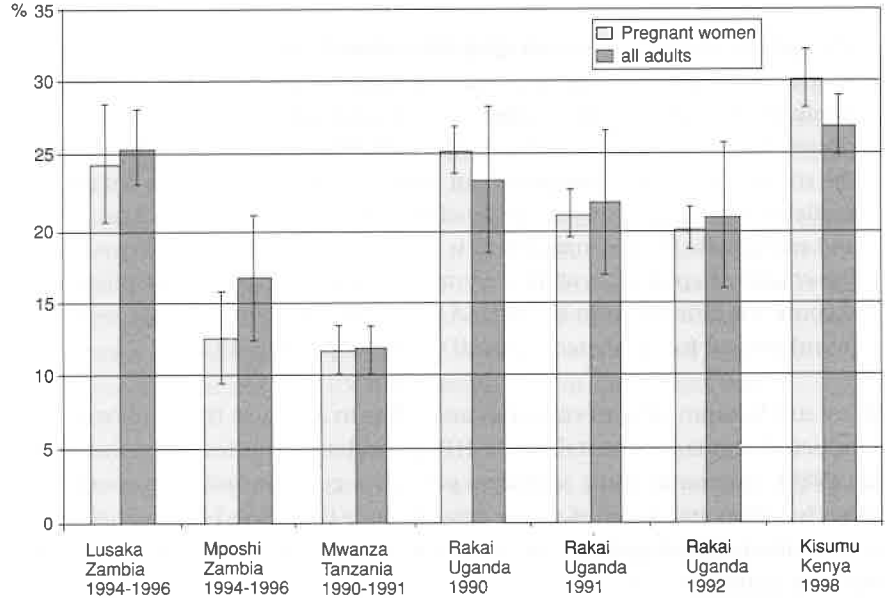
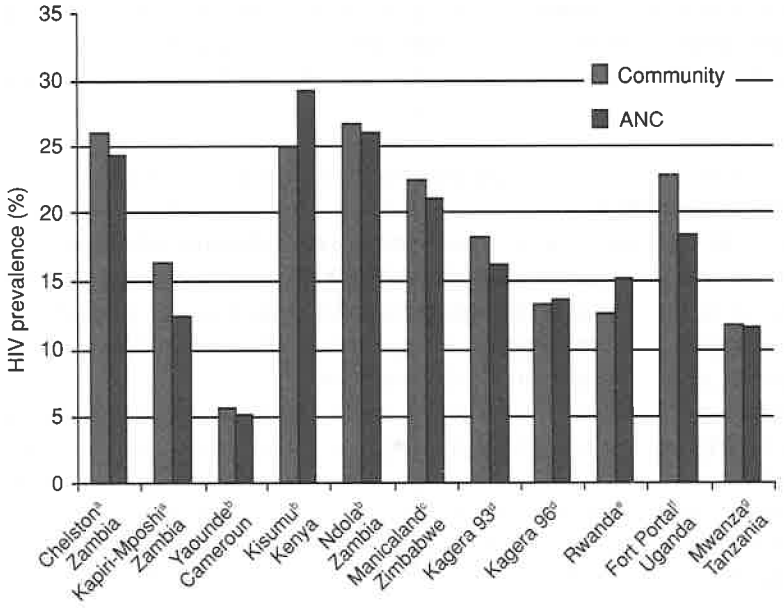



*Figure 2.11* HIV prevalence rates among pregnant women and among all adults aged 15–49

*Source*: UNAIDS (2000f).






[a] Comparisons on 15–39, adjusted.
[b] Marks comparisons on 15–50, age standardised.
[c] Comparisons on 15–44.
[d] Marks comparisons 15+, age standardised.
[e] Comparisons on 15–44, non-adjusted.
[f] Comparison of crude data.
[g] Crude data, community 15–54, antenatal clinic data 15+.

*Figure 2.12* HIV prevalence in adults (community studies) and pregnant women (antenatal clinics (ANC))

*Source*: WHO/UNAIDS (2003).

In most countries in Asia, South and Central America and Eastern Europe, the first manifestations of the disease, AIDS cases, were found in particular groups. These became subject to epidemiological surveillance. It was assumed that they represented high-risk behaviours. They included intravenous drug users; men who have sex with men, and sex workers and their clients. Here the methods for estimating HIV prevalence are different. What is needed is information on HIV prevalence in each group with high-risk behaviour, together with estimates of the size of each of these populations and the prospect of the epidemic bridging to the broader population. 'Since these behaviours are often socially unacceptable and sometimes illegal, information on both HIV prevalence levels and the size of the population affected can be much harder to come by. Consequently, uncertainties around these estimates may well be greater for countries where the epidemic is

concentrated in specific groups' (UNAIDS, 2000f, pp. 116–18). A good example of the difficulties is countries where drug possession is a capital offence. In these circumstances it is particularly hard to track the epidemic in intravenous drug users.

UNAIDS presents data in its annual reports, but it is hedged with caveats. Estimates of new HIV infections and HIV-related deaths are developed through 'simple back-calculation' procedures, which are based on the 'well-known natural course of HIV infection which determines the relationship between HIV incidence, prevalence and mortality' (UNAIDS, 2000f, pp. 116–18). Estimates for mother-to-child transmission (including breastfeeding) and HIV mortality in children are calculated from countries' age-specific fertility rates and documented region-specific rates of mother-to-child transmission.

Private sector companies and organisations are beginning to collect data for their own purposes. We know that in southern Africa insurance companies are gathering such information because they routinely test people before offering cover. These data are biased to those applying for policies and are often commercially sensitive, and so they tend not to be publicly available. For companies wishing to estimate how the epidemic is going to affect their workforce, the advent of saliva and urine tests mean surveys can be carried out more easily. These tests are non-invasive and relatively cheap. (In Chapter 10 we describe how, in consultation with the workers, this information was collected by one major employer in Botswana and how, if it is correctly and sensitively used, it can be valuable in designing company responses to the impact of the epidemic.)

Finally it is a routine procedure to test blood donations and these data can provide a picture of what is going on in what should be a low-risk group. Blood donors may be considered a low-risk group because organisations collecting blood try to exclude HIV-positive people.

HIV data are also collected and constructed according to political, social and other biases. The mere act of looking for HIV in one particular group has political and social significance. A national epidemic is the construct of a particular reporting system embedded in a specific polity which filters information into data; it is the signal which is modulated out of the background noise. The polity is a part of the modulating process. It defines and enables the reporting system, and may itself be an aspect of the relative susceptibility of societies. For example, a political system that insists on classifying HIV infections by 'race' would present one perspective. A system which refused to recognise the existence of male homosexuality or widespread intravenous drug use would present another filter; and a political system which could not afford to report accurately because there was no money for test kits would produce yet another slant.

Arguments about numbers may also be politically charged. This was apparent in the correspondence pages of the *South African Medical Journal* in 2000. Four



independent researchers – Dorrington, Bradshaw, Bourne and Abdool Karim (Dorrington et al., 2000) – argued that the officially stated decline in HIV prevalence from 1998 to 1999 (from 22.8% to 22.4%) was incorrect. An examination of the 1999 results showed that prevalence fell only in Mpumalanga, a province with 7% of South Africa's population. Otherwise rates of infection showed little or no change in three provinces and rose in the remaining five. Dorrington et al. therefore concluded, using population weighted methods, that national prevalence should not have fallen; rather, a small increase was to be expected. Government officials and a respondent from the South African Medical Research Council (2000) argued that the data were accurate, and castigated Dorrington et al. for their pessimism, for their failure to approach the Department of Health before writing with 'whatever suggestions they might have', and for not 'joining in an active partnership against HIV/AIDS'.

Readers may think that this debate smacks of rearranging the deckchairs on the *Titanic*. The magnitude of the crisis is not debated, just the detail. However, such a discussion points to the danger of debating figures rather than focusing on what they tell us about dealing with prevention and impact mitigation. It also shows the defensiveness of some governments (Dorrington et al., 2000).

### *The use of data*

Data have three key functions: advocacy, prevention and prediction.

Advocacy requires people to see and understand the potential for the epidemic to develop and the impact it may have. The problem, clear from the preceding discussion, is that AIDS data are inadequate and outdated and HIV data do not show a visible epidemic.

Prevention remains the goal of all in the field of HIV/AIDS. HIV data give a picture of where the epidemic is located, the scale of the problem and who should be targeted for prevention interventions. They help in assessing whether prevention activities are working. A decline in prevalence among younger women is seen as the first sign of hope. However, this needs to be treated with caution, as it is not clear whether the infections are averted or simply deferred. Women may be uninfected in their late teens because they do not have sex or because they use condoms. They become infected later, when they become sexually active or decide to have children.

Lack of incidence data also means that if the prevalence plateaus, we cannot be sure whether this is because people who die are being replaced with new infections. The turnover can be considerable. An apparently stable epidemic hides many deaths and new infections. The new generation of tests – both blood and saliva – may assist in providing incidence data, which will help to show whether interventions are having any effect. But few give any consideration to the question of the impact of the disease. HIV infections become AIDS cases and

AIDS deaths. AIDS cases need care, and AIDS deaths cut to the core of households and societies, leaving orphans and impoverishment.

Prediction tells us about the future course of the epidemic and its possible impacts. This is done through modelling. Here we use HIV data because AIDS case data have limited value. While it is useful to know the scale of the problem facing us in the present, what is most important in planning for impact is to know what will happen in the future. How many people will fall ill? How many orphans will there be? In order to look into the future the epidemic has to be projected through a process of modelling.

Mathematical models (which are translated into computer programs) may be used to create projections of the course of the epidemic and its impacts, and more specifically estimate their magnitude.

HIV/AIDS projection models may be used for several different purposes, such as:

- projecting HIV prevalence and numbers
- projecting future AIDS cases, AIDS-related deaths and orphans
- examining the demographic impact of AIDS and addressing questions regarding the impact of AIDS on population growth rates, the population age structure, numbers of orphans,[9] and life expectancy
- simulating different intervention strategies and comparing their strengths and weaknesses
- assessing the impact of the AIDS epidemic; for example, in terms of increased health expenditure and interactions with other diseases such as tuberculosis[10]
- creating different scenarios which illustrate the effect of different assumptions on the projected outcome.

All models depend on data, and the amount and type of data required will depend on the model used, the questions to be answered and the data to hand. This in turn will depend critically on whether a country is able to collect information about its epidemic. Does it have the technical, financial and political resources to do so? It is important to keep in mind that models are simply tools, which may be used to guide decision making. Models are by definition a *representation* of an *aspect* of reality and they cannot possibly replicate the complexity of any real situation.

## Conclusion

This chapter has described the basic science of HIV and the AIDS. It has explored the epidemiological instruments through which we 'know' or construct our knowledge about the aggregate effects of the disease.



At every point we see that data about the epidemic – including what it is, how it is defined, and how it is measured – are not neutral. Data are the outcomes of social, economic and cultural processes. Data are political: it may be that the governments do not want to admit that the epidemic exists; perhaps because they don't believe that their citizens 'behave in *these* ways', or that there are potential economic consequences. Admitting that there is an uncontrolled epidemic may also mean acknowledging that government policies have failed.

The form in which data appear depends on who is looking at information and how: doctors look among their patients, actuaries among those who form their client pool, anthropologists and sociologists in the particular group they are studying. Bias also arises from who is paying for the data – if you have an AIDS project it is not in your interest to show there is no AIDS!

Data are used to model the development and impact of the epidemic, but this too, is not a neutral activity: models have assumptions and biases built into them according to the disciplines and beliefs of those who develop them and those who pay for them. Results will be interpreted differently according to the biases (explicit or implicit) of those who use them.

We have indicated that the disease has implications far beyond the individual bodies that it destroys. It has social and economic causes and consequences. In Chapter 3 we consider why epidemics should differ so dramatically between societies.

## Books, papers, websites and other useful sources for further reading

Michael Adler, *The ABC of AIDS* (London: BMJ Publications, 2001).
Roy M. Anderson and Robert M. May, *Infectious Diseases of Humans: Dynamics and Control* (Oxford: Oxford University Press, 1992).
Laurie Garrett, *The Coming Plague: Newly Emerging Diseases in a World Out of Balance* (Harmondsworth: Penguin Books, 1995).
Jaap Goudsmit, *Viral Sex* (Oxford: Oxford University Press, 1998).
Jaap Goudsmit, *Viral Fitness: The SARS and West Nile in the Making* (Oxford: Oxford University Press, 2004).
Salim Karim and Quarraisha Karim (eds), *HIV/AIDS in South Africa* (Cambridge: Cambridge University Press, 2005).
William H. McNeill, *Plagues and Peoples* (Oxford: Blackwell, 1977).
UNAIDS (Joint United Nations Programme on HIV/AIDS), at <http://www.unaids.org>.
United States Bureau of the Census, at <http://www.census.gov>.
Avert, at <http://www.avert.org>.
Centers for Disease Control and Prevention, at <www.cdc.gov>.